

## Original Article

# Neural adaptations after short-term wingate-based high-intensity interval training

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**Abstract**

**Objectives:** This study examined the neural adaptations associated with a low-volume Wingate-based High Intensity Interval Training (HIIT). **Methods:** Fourteen recreationally trained males were divided into an experimental (HIIT) and a control group to determine whether a short-term (4 weeks) Wingate-based HIIT program could alter the Hoffmann (H-) reflex, volitional (V-) wave and maximum voluntary contraction (MVC) of the plantar-flexor muscles, and the peak power achieved during a Wingate test. **Results:** Absolute and relative peak power increased in the HIIT group ( $ABS\_P_{peak}$ : +14.7%,  $P=0.001$ ; and  $REL\_P_{peak}$ : +15.0%,  $P=0.001$ ), but not in the control group ( $ABS\_P_{peak}$ :  $P=0.466$ ; and  $REL\_P_{peak}$ :  $P=0.493$ ). However, no significant changes were found in the MVC ( $P>0.05$  for both groups). There was a significant increase in H-reflex size after HIIT (+24.5%,  $P=0.004$ ), while it remained unchanged in the control group ( $P=0.134$ ). No significant changes were observed either in the V-wave or in the  $V_{wave}/M_{wave}$  ratio ( $P>0.05$  for both groups). **Conclusion:** The Wingate-based training led to an increased peak power together with a higher spinal excitability. However, no changes were found either in the volitional wave or in the MVC, indicating a lack of adaptation in the central motor drive.

**Keywords:** H-reflex, V-wave, HIIT, Wingate**Introduction**

The human nervous system is highly adaptable in response to different types of training<sup>1</sup>. These neural adaptations take place at both spinal and supraspinal levels<sup>2</sup> and they are task and training-dependent<sup>3,4</sup>. In this regard, it is well known that endurance and resistance training promote almost opposite adaptations. While endurance training has been shown to decrease motor units' firing rates, increased firing rates are observed after a period of resistance training<sup>3</sup>.

Neural adaptations to different training stimuli (i.e., endurance and resistance training) have been also investigated by measuring electrically evoked reflex responses<sup>5-9</sup>, such as the Hoffmann reflex (H-reflex) and the volitional wave (V-wave). The H-reflex is a good marker

of motoneuronal excitability and presynaptic inhibition of primary muscle spindle afferents<sup>10</sup> whereas the V-wave is very sensitive to changes in descending neural drive from the alpha-motor neuron pool<sup>11</sup> to the muscle. Thus, the combination of both measures can help to understand the neural adaptations elicited by different training protocols<sup>9</sup>. In this regard, several cross-sectional studies have reported higher amplitudes of the H-reflex in endurance-trained athletes than in strength and power-trained athletes<sup>6,8</sup>. Some longitudinal studies have also revealed a significant increase in the H-reflex amplitude after a traditional endurance-training regime<sup>9</sup>. This increased H-reflex size after endurance training has been attributed to an increased proportion of type I fibers<sup>6,8,12</sup>. Motor neurons associated with Type-I fibers, which form slow-twitch motor units, have a lower depolarization threshold (i.e., they are more excitable)<sup>12</sup>. This could explain the changes observed in the H-reflex amplitude, since the ascending limb of the H-reflex recruitment curve is mainly dependent on the excitation of small-diameter motor units<sup>13</sup>. In contrast, although resistance training does not seem to produce changes in the H-reflex<sup>7,14-16</sup>, some studies have reported higher V-wave amplitudes after a period of strength training, which probably reflects improvement of the neural drive in descending corticospinal pathways<sup>5,7</sup>.

In the last few years, high intensity interval training (HIIT)

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has become popular because it produces similar or even greater improvements in physiological and performance parameters than traditional endurance training<sup>17,18</sup>, despite a markedly lower total training time and volume<sup>18-21</sup>. Wingate-based training is one of the most studied types of HIIT. It is characterized by relatively short bursts of supramaximal cycling (i.e., 30s), interspersed by periods of passive rest or low-intensity exercise for recovery<sup>22</sup>. Several studies have observed improvements in maximum oxygen uptake<sup>21</sup>, muscle oxidative capacity<sup>18,19</sup>, mechanical mean and peak power<sup>23</sup> and body composition<sup>24</sup> after just a few sessions of Wingate-based training. However, little is known about the neural adaptations elicited by Wingate-based HIIT training.

It has been recently shown that Wingate-based training produces very high levels of fatigue associated with central and peripheral components<sup>25</sup>. The central fatigue generated by this type of training may lead to chronic adaptations at a neuromuscular level. Therefore, the purpose of this study was to analyze the neural adaptations associated with a low-volume Wingate-based HIIT. We hypothesized that this training could produce adaptations at both supra-spinal (V-wave) and spinal (H-reflex) levels. This is based on the fact that Wingate-based HIIT involves high levels of muscle activation, while at the same time a high O<sub>2</sub> consumption is demanded when performed repeatedly using incomplete recovery<sup>19</sup>.

## Methods

### Participants

Fourteen recreationally trained healthy males (age: 23.1±2.7 years; height: 174.2±5.4 cm; body mass: 72.1±9.0 kg) with no history of lower limb or neuromuscular disorder were recruited for this study. All of them 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 was engaged in regular training for a particular sporting event or competition. Once the pre-test measures were completed, all the subjects were randomly assigned into a control (control group, n=7) or a high intensity interval training (HIIT, n=7) group. All subjects gave written informed consent before being included in the study, which was approved by the University's Institutional Review Board and conducted in accordance with the latest version of the Declaration of Helsinki.

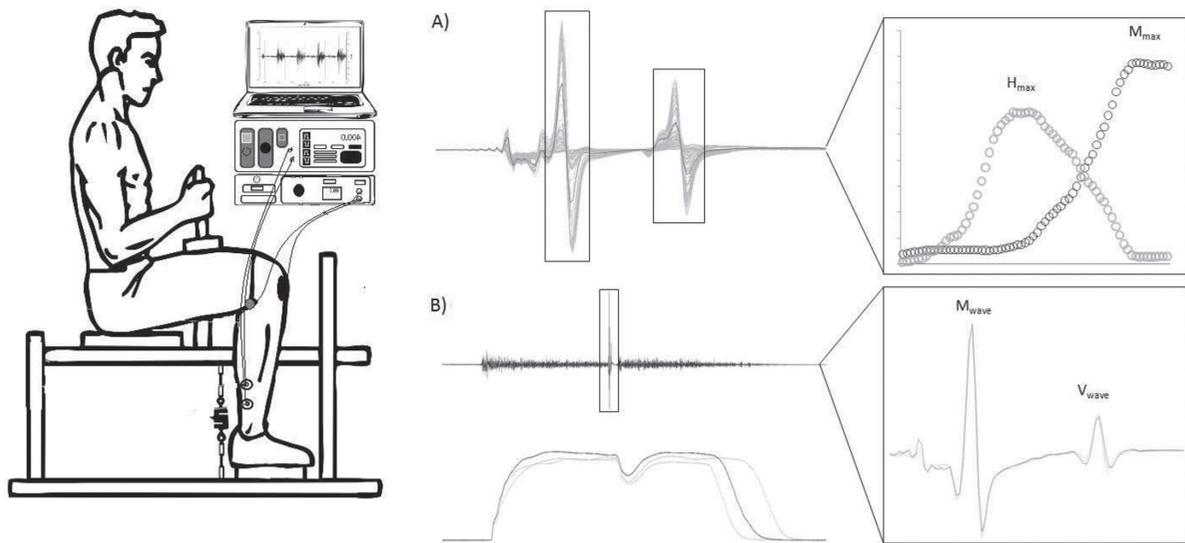
### General experimental procedure

This study used a quasi-experimental pre- to post-test design using one experimental (HIIT) group and a control group to examine whether a short-term (4 weeks) Wingate-based HIIT program could alter motoneuronal excitability (H-reflex), volitional drive (V-wave) and Maximal Voluntary Contraction (MVC) of the plantar-flexor muscles (soleus), as well as the peak power achieved during a Wingate test. Before data collection, all participants took part in a familiarization session with the Wingate test, peripheral nerve stimulation and MVCs. One week later, the dependent variables were

tested as described below. In session one, H-reflexes were measured at rest. Then, subjects were required to perform isometric MVCs with and without electrical stimulation on a soleus isolation machine to assess the MVC and V-waves. Two to three days apart, in a second experimental session, subjects were asked to perform a Wingate test to evaluate peak power. Then, during the following 4 weeks, the experimental group performed Wingate-based HIIT training in an incremental periodized program (i.e., three to six sets) three times per week (Monday, Wednesday and Friday). The control group did not train. After this four-week period, both groups were tested again in similar conditions (same researcher, protocol, time of day and a temperature of 22-24°C). For all experimental protocols, the subjects were instructed to fast for 2 h beforehand and not to consume alcohol, caffeine or taurine-containing drinks within 12 h. They were also asked to avoid strenuous physical activities the day before each session. During the 4-week training period, all subjects were asked to maintain their normal daily routines and eating habits, not to take nutritional supplements, and to refrain from commencing new exercise programs during the study.

### Neuromuscular assessment

The soleus H-reflex was obtained by posterior tibial nerve stimulation with the cathode (1cm diameter) located in the popliteal fossa and the anode (4 x 4 cm electrode) placed just under the patella. The optimal stimulation point was firstly located using a hand-held electrode, using various test stimuli to find the point of greatest Ia afferent recruitment. Rectangular stimuli of 0.2 ms duration were applied with a constant-current stimulator (DS7AH; Digitimer; Hertfordshire, UK). The initial intensity of the electric stimulation, which corresponded to the H-reflex threshold, was gradually increased (0.5 mA steps) to obtain a maximum H-wave ( $H_{max}$ ). The intensity was then increased at intervals of 1 mA until a maximum direct muscle response ( $M_{max}$ ) was obtained and further increases in intensity did not produce increments in M-wave amplitude. During the whole process, the subjects remained seated and relaxed, with hip, knee and ankle joints in 90° of flexion. Peak-to-peak amplitudes were computed offline from the unrectified electromyographic (EMG) signals. Then, the maximal H-reflex was normalized to the corresponding maximal M-wave ( $H_{max}/M_{max}$  ratio; see Figure 1A). For the MVC measurements, subjects were seated in a soleus isolation machine (Technogym SpA, Cesena, Italy) in the same position used for the H-M recruitment curve measurements. The lever of the isolation machine was anchored with a metal chain in order to restrict all movements and allow a purely isometric soleus contraction. All subjects performed 3-4 MVC of 3 seconds duration with 90 seconds of rest in between. In each trial, subjects were firmly encouraged to ensure maximal effort. A force transducer (model 520 -500 lb-DTS, Noraxon, Scottsdale, AZ), anchored to the metal chain that restricted the movement of the lever, was used to record MVCs. After the completion of three valid attempts, the arithmetic mean



**Figure 1.** Experimental set-up. A) Example of overdrawn M- and H- responses from a single subject, which were then analyzed (peak-to-peak amplitude in mV) to compute the  $H_{\max}/M_{\max}$  ratio. B) Example of overdrawn M- and V-waves recorded during the performance of MVCs, which were then used to compute the  $V_{\text{wave}}/M_{\text{wave}}$  ratio.

of the two highest contractions was calculated and used as a reference for subsequent contractions in which the V-wave was obtained.

For the V-wave recordings, a supramaximal stimulus (120%  $M_{\max}$  intensity, 0.2 ms rectangular pulse) was delivered over the posterior tibial nerve (at the same point as for the H-M recruitment curves) during the performance of MVCs. Five valid MVCs (3 s contraction; 90 s rest) were recorded. Stimulation was automatically applied 2 s after the beginning of the contraction, when the force reached the plateau area. Attempts were discarded if the amplitude of the MVC and M-wave did not reach at least 90% of previously recorded values of MVC and  $M_{\max}$ , respectively. Then, peak-to-peak amplitudes of the V- and M-waves were computed offline from the unrectified EMG signals and expressed as a ratio (i.e.:  $V_{\text{wave}}/M_{\text{wave}}$  ratio; see Figure 1B). We also computed the integrated EMG (iEMG) amplitude in a time window of 500 ms prior to the electrical stimulation, and it was then normalized to the amplitude of the  $M_{\max}$  in a trial-by-trial fashion ( $iEMG/M_{\max}$ ).

All the EMG recordings were obtained from the belly of the soleus muscle using Ag-AgCl circular electrodes (10 mm diameter, 3 cm inter-electrode distance) with a tendon-belly assembly<sup>5</sup>. After skin preparation (shaved, abraded and cleaned with alcohol), electrodes were attached to the skin and secured with adhesive tape to prevent possible artefacts. EMG signals were amplified (x500), bandpass-filtered (10-500 Hz) and sampled at 1500 Hz with a DTS Desktop (Noraxon, Scottsdale, AZ) connected to an analog-digital CED Micro 1401-3 board (Cambridge Electronic Design, Cambridge, UK).

### Wingate test

The Wingate test was conducted on a Technogym Bike Med cycle ergometer (Technogym SpA, Cesena, Italy). This test consisted of a 30 s maximal sprint against a constant braking resistance dependent on the subjects' body mass (0.075  $\text{kg}\cdot\text{kg}^{-1}$  body mass) according to the optimization tables of Bar-Or<sup>26</sup>. The test began from a rolling start, at 60 rpm against minimal resistance. When a constant pedal rate of 60 rpm was achieved, the investigator gave a countdown of "3-2-1-go!". Subjects were instructed to pedal as fast as they could for 30 s. During the test, they were strongly and vigorously encouraged to sprint maximally throughout the 30 s. The power produced was calculated as the highest value chosen for maximal power. Results were then expressed as absolute (W) and relative ( $\text{W}\cdot\text{kg}^{-1}$ ) values.

### Training program

Subjects in the experimental group performed HIIT three times per week for four weeks with at least one day of rest between each training session. The training volume increased from three to six sets during the training period, with the addition of one set every week. At the beginning of each session, subjects performed a general warm-up that consisted of 10 min of cycling at 60-75 rpm (50 W). After that, subjects performed every maximal sprint for 30 s against a constant braking resistance (0.075  $\text{kg}\cdot\text{kg}^{-1}$  body mass), with 4 min of active rest (60-75 rpm with minimal resistance) between sprints. After completing all sprints in each session, subjects performed 5 min of low intensity cycling to return to baseline.

**Table 1.** Mean (SD) values achieved by HIIT and control groups in the Wingate and MVC tests before and after a 4-week training period.

Variable	Group	Pre-test	Post-Test	$\Delta\%$ (90%CI)	ANOVA	
					Time	Time x Group
<b>ABS_P<sub>peak</sub></b> (W)	HIIT	781 (133)	890 (129)*	14.7 (7.4; 22.1)#	$F_{1,12} = 14.5, P = 0.003,$ $\eta_p^2 = 0.55$	$F_{1,12} = 7.5, P = 0.018,$ $\eta_p^2 = 0.38$
	control	721 (105)	739 (127)	2.4 (-3.2; 8.0)		
<b>REL_P<sub>peak</sub></b> (W/Kg)	HIIT	10.4 (0.8)	12.0 (1.0) *	15.0 (7.7; 22.3) #	$F_{1,12} = 13.3, P = 0.003,$ $\eta_p^2 = 0.53$	$F_{1,12} = 6.9, P = 0.021,$ $\eta_p^2 = 0.37$
	control	10.5 (0.7)	10.8 (1.1)	2.3 (-3.4; 8.1)		
<b>MVC</b> (N)	HIIT	723 (189)	778 (212)	10.9 (-9.0; 30.8)	$F_{1,12} = 0.55, P = 0.475,$ $\eta_p^2 = 0.04$	$F_{1,12} = 0.41, P = 0.536,$ $\eta_p^2 = 0.03$
	control	748 (141)	752 (210)	-0.8 (-14.3; 12.7)		

*ABS\_P<sub>peak</sub>*: absolute peak power; *REL\_P<sub>peak</sub>*: relative peak power; *MVC*: maximal voluntary contraction; *HIIT*: high intensity interval training; *control*: control.  $\Delta$  (%): percent change from pre- to post-test; \* differences between pre- and post-test ( $P < 0.05$ ); # differences between HIIT and control groups ( $P < 0.05$ ).

**Table 2.** Mean (SD) values achieved by HIIT and control groups in the neuromuscular evaluation before and after a 4-week training period.

Variable	Group	Pre-Test	Post-Test	$\Delta\%$ (90%CI)	ANOVA	
					Time	Time x Group
<b>M<sub>max</sub></b>	HIIT	7.0 (0.8)	7.2 (0.8)	3.0 (-9.3; 15.4)	$F_{1,12} = 0.041, P = 0.843,$ $\eta_p^2 = 0.003$	$F_{1,12} = 0.08, P = 0.782,$ $\eta_p^2 = 0.007$
	control	6.2 (1.0)	6.1 (1.4)	-0.4 (-12.2; 11.5)		
<b>H<sub>max</sub></b>	HIIT	3.3 (0.9)	4.0 (0.8)*	24.5 (4.4; 44.6)#	$F_{1,12} = 1.9, P = 0.196,$ $\eta_p^2 = 0.14$	$F_{1,12} = 13.2, P = 0.003,$ $\eta_p^2 = 0.525$
	control	3.3 (1.0)	3.0 (1.0)	-10.3 (-16.0; -4.6)		
<b>H<sub>max</sub>/M<sub>max</sub></b> Ratio	HIIT	0.47 (0.09)	0.57 (0.10)*	23.0 (-1.0; 47.1) #	$F_{1,12} = 1.0, P = 0.333,$ $\eta_p^2 = 0.08$	$F_{1,12} = 4.7, P = 0.050,$ $\eta_p^2 = 0.28$
	control	0.52 (0.10)	0.49 (0.15)	-7.9 (-20.6; 4.9)		
<b>M<sub>wave</sub></b>	HIIT	7.0 (0.2)	7.2 (0.5)	2.7 (-2.0; 7.4)	$F_{1,12} = 1.0, P = 0.330,$ $\eta_p^2 = 0.08$	$F_{1,12} = 0.4, P = 0.544,$ $\eta_p^2 = 0.03$
	control	6.7 (1.0)	6.8 (1.0)	0.7 (-4.2; 5.6)		
<b>V<sub>wave</sub></b>	HIIT	2.4 (0.8)	2.6 (0.5)	14.8 (-3.8; 33.4)	$F_{1,12} = 0.5, P = 0.485,$ $\eta_p^2 = 0.02$	$F_{1,12} = 0.3, P = 0.603,$ $\eta_p^2 = 0.04$
	control	2.6 (0.9)	2.6 (1.2)	-1.7 (-24.4; 21.0)		
<b>V<sub>wave</sub>/M<sub>wave</sub></b> Ratio	HIIT	0.34 (0.11)	0.36 (0.06)	11.5 (-4.6; 27.6)	$F_{1,12} = 0.1, P = 0.721,$ $\eta_p^2 = 0.01$	$F_{1,12} = 0.4, P = 0.515,$ $\eta_p^2 = 0.04$
	control	0.37 (0.08)	0.37 (0.14)	-2.5 (-24.3; 19.2)		
<b>iEMG/M<sub>max</sub></b> Ratio	HIIT	23.2 (7.3)	22.1 (4.7)	-1.5 (-15.9; 13.0)	$F_{1,12} = 2.7, P = 0.129,$ $\eta_p^2 = 0.18$	$F_{1,12} = 0.07, P = 0.798,$ $\eta_p^2 = 0.006$
	control	25.8 (5.8)	23.8 (6.4)	-7.0 (-13.6; -0.4)		

*ABS\_P<sub>peak</sub>*: absolute peak power; *REL\_P<sub>peak</sub>*: relative peak power; *MVC*: maximal voluntary contraction; *HIIT*: high intensity interval training; *control*: control.  $\Delta$  (%): percent change from pre- to post-test; \* differences between pre- and post-test ( $P < 0.05$ ); # differences between HIIT and control groups ( $P < 0.05$ ).

Each training session was supervised by an experienced instructor, who strongly encouraged the subjects in each “all-out” sprint, and ensured that all rest periods were observed. The total training time ranged between 25 min (three sprints) and 38 min (six sprints).

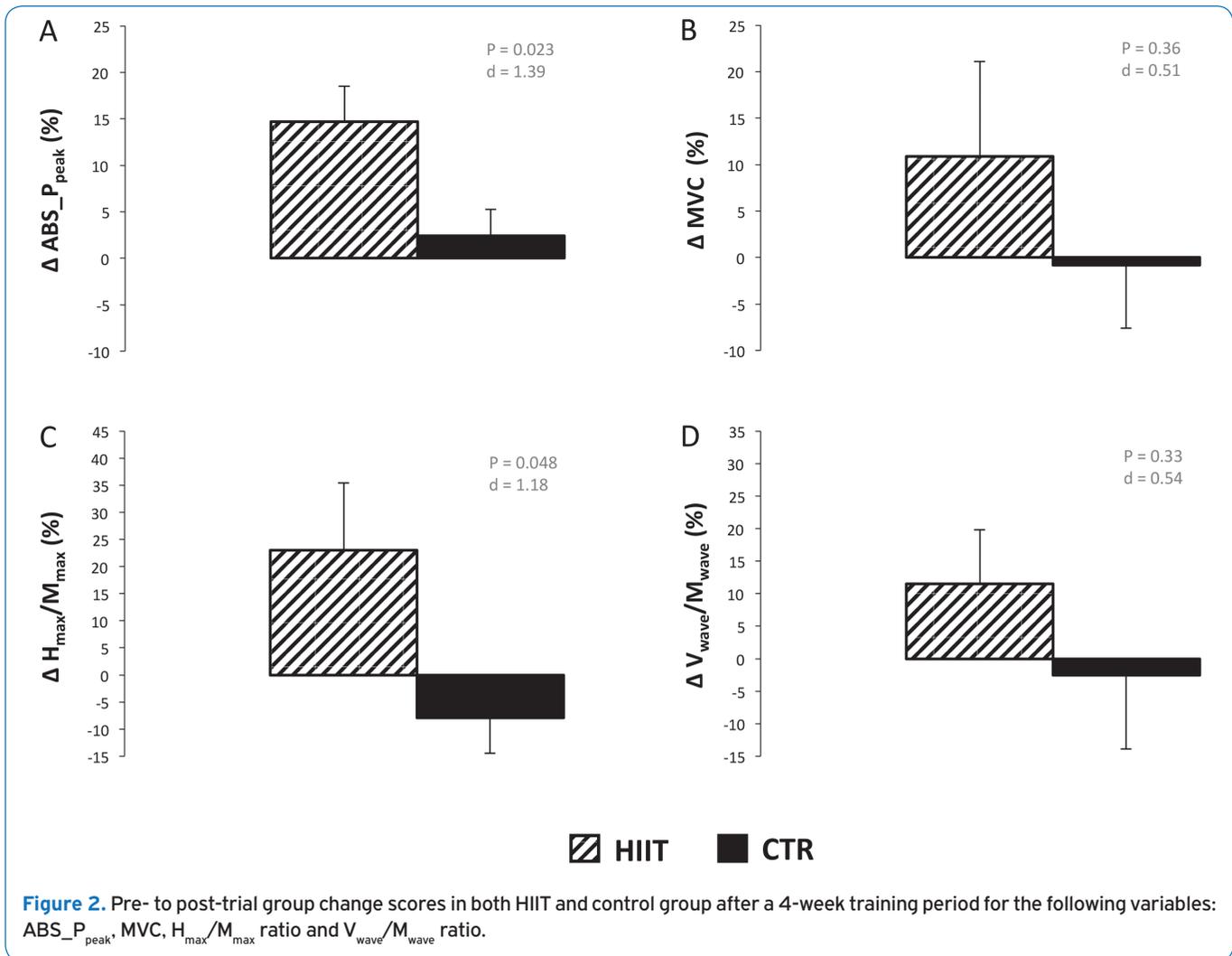
### Statistical analysis

Firstly, the normal distribution and homogeneity were checked using Shapiro-Wilk and Levene tests, respectively. Then, a mixed model analysis of variance (ANOVA) with the group (HIIT vs Control) as between-subjects and time (pre- to post-test) as within-subjects factor was performed for the following variables:  $ABS\_P_{peak}$ ,  $REL\_P_{peak}$ , MVC, soleus V- and M-waves, soleus H-reflex and  $M_{max}$ , the  $V_{wave}/M_{wave}$  ratio, the  $H_{max}/M_{max}$  ratio, and the soleus  $iEMG/M_{max}$  ratio. An independent “t” test was firstly performed to discard between-group differences in the pre-test values. When a

TIME effect was observed, pairwise comparisons for each individual group were performed to assess pre- to post-test differences. When significant interactions of both factors (TIME x GROUP) were found, independent “t” tests were computed on the pre- to post-trial change scores [ $\Delta$  (%)]. The calculation of  $\Delta$  (%) used the pre- to post-test differences divided by the pre-test values and then multiplied by 100 and was carried out for each subject. Significance was accepted when  $p < 0.05$ . Effect sizes (ES) were reported as partial eta square ( $\eta_p^2$ ) for the ANOVA (small  $\leq 0.06$ , medium 0.07-0.14, large  $> 0.14$ ) and Cohen’s  $d$  for paired comparisons (small  $\leq 0.2$ , medium  $> 0.2$  and  $\leq 0.5$ , large  $\geq 0.8$ )<sup>27</sup>.

### Results

No significant differences were found between HIIT and control group during the pre-tests in any of the variables studied.



### Wingate and MVC

Both absolute and relative peak power were statistically higher in the post-test than in the pre-test for the HIIT group ( $\text{ABS\_P}_{\text{peak}}$ :  $P=0.001$ ,  $d=0.83$ ; and  $\text{REL\_P}_{\text{peak}}$ :  $P=0.001$ ,  $d=1.67$ ). There were no statistically significant changes in the control group ( $\text{ABS\_P}_{\text{peak}}$ :  $P=0.466$ ,  $d=0.15$ ; and  $\text{REL\_P}_{\text{peak}}$ :  $P=0.493$ ,  $d=0.26$ ). The post-hoc analysis revealed that the  $\text{ABS\_P}_{\text{peak}}$  and  $\text{REL\_P}_{\text{peak}}$  changes in the HIIT group were much higher than those observed in the control group ( $\text{ABS\_P}_{\text{peak}}$ : 14.7% vs. 2.4%,  $P=0.023$ ,  $d=1.39$ , see Figure 2A;  $\text{REL\_P}_{\text{peak}}$ : 15.0% vs. 2.3%;  $P=0.021$ ,  $d=1.41$ ).

For MVC, the ANOVA did not display either a TIME effect or a TIME\*GROUP interaction (detailed information is displayed in Table 1 and Figure 2B). Although MVC increased by 10% after the Wingate-based HIIT, the magnitude of the observed change was small (pre- to post-test effect size=0.27).

### Neurophysiological parameters

The results revealed a significant increase in the size of the H-reflex after HIIT ( $P=0.004$ ,  $d=0.77$ ), while this remained

unchanged in the control group ( $P=0.134$ ,  $d=0.31$ ). The post-hoc analysis also revealed that pre- to post-test trial changes in the HIIT group were statistically different from those observed in the control group (24% vs. -10%, respectively;  $P=0.003$ ,  $d=1.73$ ). However, the  $\text{M}_{\text{max}}$  associated with the H-reflex remained constant across the testing sessions (see detailed information in Table 1). This led to an increased  $\text{H}_{\text{max}}/\text{M}_{\text{max}}$  ratio after HIIT ( $P=0.044$ ,  $d=0.96$ , see Fig. 2C) only, because no significant differences were observed in the control group ( $P=0.428$ ,  $d=0.27$ , see Fig. 2C). The post hoc analysis of the  $\text{H}_{\text{max}}/\text{M}_{\text{max}}$  ratio also revealed that pre- to post-test trial changes after HIIT were statistically different from those observed in the control group (23% vs. -7.9%, respectively;  $P=0.048$ ,  $d=1.18$ ; see Fig. 2C).

Contrary to our hypothesis, no significant changes were observed either in the V-wave or in the  $\text{V}_{\text{wave}}/\text{M}_{\text{wave}}$  ratio (see Fig. 2D). The M-wave associated with the V-waves and soleus iEMG/ $\text{M}_{\text{max}}$  ratio also remained unchanged (detailed information on ANOVA outcomes is displayed in Table 2).

## Discussion

Following four weeks of Wingate-based HIIT there was a significant increase in peak power (~15%). This change in power output during the Wingate test was accompanied by an increase (~24%) in spinal excitability (i.e., a higher H-reflex amplitude). However, in contrast with our hypothesis, no significant improvements were observed in either the central efferent drive (i.e., V-wave) or MVC.

### *Improved power performance in absence of changes in volitional drive*

Several studies have previously demonstrated that HIIT produces similar, or even greater, physiological adaptations than high-volume endurance training in aerobic<sup>28</sup> and anaerobic<sup>23</sup> metabolism, exercise performance and cardiorespiratory fitness (for example, see the review by Sloth et al.<sup>29</sup>) despite large differences in exercise volume and intensity. Therefore, we used a previously validated and well-studied protocol, the Wingate-based HIIT, to verify whether similar neuromuscular adaptations could be observed despite the divergent nature of this training compared with that previously used (traditional endurance training). As expected, four weeks of Wingate-based HIIT resulted in an increase in peak power output, in agreement with previous reports<sup>20,21</sup>. This power improvement could be attributed to several factors such as increments in muscular phosphocreatine reserves<sup>30</sup>, anaerobic enzyme activity<sup>23</sup>, improved glycolytic capacity<sup>31</sup>, or improved Ca<sup>+</sup> kinetics. However, another plausible explanation is based on improvements in neuromuscular factors, such as central drive and motor unit behavior. Previous studies have established that strength and power training produces an increase in the volitional drive<sup>5</sup> together with higher motor unit discharge rates<sup>3</sup> and synchronization<sup>32</sup>, leading to higher muscular power. Therefore, after a Wingate-based training we would expect similar adaptations as a result of the nature of the exercise (maximal power requirements). However, despite the changes observed in the Wingate performance, our HIIT protocol did not induce significant changes in either MVC or the V-wave. This lack of change in these neuromuscular markers is in accordance with previous reports that also found no changes in maximal isometric voluntary contraction after short-term HIIT<sup>33</sup>. However, Martinez-Valdes et al.<sup>34</sup> have recently demonstrated that six sessions of HIIT increased maximal knee-extensor torque and produced higher surface EMG activation at different submaximal torque levels. They also reported an increase in motor unit discharge rate at 50 and 70% of MVC, suggesting that HIIT induces a preferential change in high threshold motor units<sup>34</sup>. Nevertheless, the observed differences between our results and those mentioned above<sup>34</sup> may lie in the protocol (Wingate-based HIIT vs. HIIT based on 60s bouts against 100% peak power reached during an incremental test) and the duration of the training (12 vs. 6 sessions).

### *Increased soleus motoneuronal excitability after HIIT*

The current findings revealed that 4 weeks of Wingate-based HIIT induced an increase in the H-reflex amplitude, confirming plastic adaptations in the Ia spinal reflex pathway. These alterations suggest that HIIT can lower the recruitment threshold of motor neurons to Ia afferent input, as has been previously observed after high-volume low-load endurance training<sup>9</sup>. Moreover, previous cross-sectional studies have shown higher H-reflex amplitudes in endurance-trained athletes compared with resistance and power-trained athletes<sup>8,35,36</sup>. These changes were partially attributed to differences in the muscle fiber type distribution between the two groups of athletes. Endurance-trained athletes have a higher percentage of slow-twitch fibers<sup>37</sup>, and the excitatory postsynaptic potentials are largest in small motor neurons innervating slow-twitch motor units<sup>38</sup>, which would explain the greater H-reflex responses observed in endurance trained athletes<sup>35</sup>. As mentioned in the previous section, the true nature of the Wingate test is ballistic in origin (i.e., maximal power); however, when performed several times in the same session under incomplete recovery conditions, it becomes an endurance-type exercise<sup>23</sup>. This is due to the higher levels of fatigue accumulated after each trial<sup>25</sup>, which compromise fast-twitch (glycolytic) fiber recruitment<sup>23</sup>, leading to a higher recruitment of slow-twitch (aerobic) fibers<sup>23</sup>. Recent studies have shown that with as little as 6 weeks of low-volume HIIT (4 min, 170%  $\text{VO}_{2\text{peak}}$ ) or traditional endurance training (30 min, 65%  $\text{VO}_{2\text{peak}}$ ) comparable changes were observed in the intra-myocellular environment and signaling activation, which resulted in an increased proportion of Type I fibers and a lowered proportion of Type IIA/X/IIIX<sup>23</sup>. Therefore, we can argue that these rapid structural and functional changes induced by HIIT might be compensated by adjustments in motor unit behavior<sup>9</sup>. It has been proposed that an adaptive mechanism in endurance training is a reduction in motor unit firing rate<sup>3</sup>, which leads to an increased number of recruited motor units to maintain similar relative loads. This lower discharge rate and higher motor unit recruitment would increase the energy efficiency and counteract muscle fatigue without affecting force and rate of force development<sup>9</sup>. This adaptive mechanism could be caused by an increase in the excitability of alpha motor neurons and a reduction in presynaptic inhibition of these motor neurons<sup>9</sup>, which would explain the observed change in the H-reflex pathway in our study.

### *Methodological considerations and limitations*

Despite the observed increase in power values during the Wingate test after 4 weeks of Wingate-based training, in our study we did not find changes in MVC as have been recently shown in other research<sup>34,39</sup>. This apparent controversy could be due to a lack of specificity between the exercise used for training (pedaling) and the strength evaluation protocol (isolated isometric plantar-extension). We used the soleus as a target muscle because previous studies have shown differential reflex changes after endurance and resistance training<sup>3,9</sup>. However, muscular activity of the

soleus during pedaling is relatively low<sup>40</sup> which may lead to a lack of MVC adaptations. This could also explain the different results obtained by Martinez-Valdes et al.<sup>34</sup> who observed an increased MVC torque, since they tested the quadriceps muscle (*vastus lateralis and medialis*), which is the prime mover during pedaling action<sup>40</sup>.

Although the magnitude of the changes (ES) observed in the power production during the Wingate test ( $ABS\_P_{peak}$  and  $REL\_P_{peak}$ ) as well as in the H-reflex and  $H_{max}/M_{max}$  ratio can be considered as “large” (ES ranged from 1.18 to 1.71), the small sample size used for this study is an important limitation. Therefore, future research shall involve higher sample size including male and female subjects.

## Conclusion

This novel study explored the changes in spinal excitability and neural drive of the soleus muscle following 4 weeks of Wingate-based HIIT. To the best of our knowledge, no previous studies have been conducted using this approach (i.e.: electrically evoked reflex responses) to explore this issue. In summary, the Wingate-based training led to an increase in anaerobic work capacity together with a higher spinal excitability. However, no changes were found in either the volitional wave or MVC. Finally, it could be advisable using this kind of training for those subjects who need to improve both power and endurance in a time-efficient way. To sum up, this study shed new light on the neural mechanisms associated with the adaptations produced by HIIT.

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