Muscle Deoxygenation and Neural Drive to the Muscle during Repeated Sprint Cycling

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ABSTRACT

RACINAIS, S., D. BISHOP, R. DENIS, G. LATTIER, A. MENDEZ-VILLANEUVA, and S. PERREY. Muscle Deoxygenation and Neural Drive to the Muscle during Repeated Sprint Cycling. *Med. Sci. Sports Exerc.*, Vol. 39, No. 2, pp. 268–274, 2007. **Purpose:** To investigate muscle deoxygenation and neural drive–related changes during repeated cycling sprints in a fatiguing context. **Methods:** Nine healthy male subjects performed a repeated-sprint test (consisting of 10 × 6-s maximal sprints interspaced by 30 s of recovery). Oxygen uptake was measured breath-by-breath; muscle deoxygenation of the vastus lateralis was assessed continuously using the near-infrared spectroscopy technique. Surface electromyograms (RMS) of both vastus lateralis and biceps femoris were also recorded. Furthermore, before and after the repeated-sprint test, the percentage of muscle activation by voluntary drive (twitch-interpolated method) was measured during a maximal voluntary contraction. **Results and Discussion:** Consistent with previous research, our data showed a significant power decrement during repeated-sprint exercise. There was also a progressive muscle deoxygenation, but our data showed that the ability of the subjects to use available O_2 throughout the entire repeated-sprint test was well preserved. Our data displayed a significant decrement in the RMS activity during the acceleration phase of each sprint across the repeated-sprint exercise. Moreover, decrement in motor drive was confirmed after exercise by a significant decrease in both percentage of voluntary activation and RMS/M-wave ratio during a maximal voluntary contraction. **Conclusion:** In this experimental design, our findings suggest that the ability to repeat short-duration (6 s) sprints was associated with the occurrence of both peripheral and central fatigue. **Key Words:** CENTRAL ACTIVATION, METABOLIC ANAEROBIOSIS, NEAR-INFRARED SPECTROSCOPY, NEUROMUSCULAR EFFICIENCY, MUSCULAR POWER OUTPUT

BASIC SCIENCES

Previous studies have shown that repeated bouts of brief, maximal-intensity exercise (< 6 s) with incomplete recovery periods (< 60 s) cause muscular fatigue (i.e., a decrease in power output) (7,11). Limitations in energy supply, intramuscular accumulation of metabolic by-products, impairment of Ca^{2+} kinetics, and ion shifts have often been implicated as causes of muscular fatigue during such intense exercise (13). However, despite increasing research suggesting that central mechanisms may contribute to fatigue (12), there has been little research investigating this source of fatigue during repeated-sprint exercise, which represents an important component of many popular sports such as soccer, tennis, or hockey.

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Gaitanos et al. (11) have previously observed, in muscle tissue, a dramatic increase in metabolic by-products (i.e., lactate and P_i), a lowered pH, and a decrement in phosphocreatine (PCr) concentration from the first to the last sprint of a repeated-sprint exercise. These changes in the intracellular environment of the recruited fibers have been associated with decrements in the power-generating capacity of the skeletal muscle (13) and suggest that peripheral mechanisms contribute to fatigue during such tasks. The results of the Gaitanos et al. (11) study also showed that the decrease in ATP production from anaerobic sources during a sprint from sprint 1 to sprint 10 was greater than the decrease in power output (11). The authors suggested that this indicates an increase in aerobic contribution to the latter sprints. However, these assumptions were based solely on postexercise muscle metabolite measures, because oxygen consumption was not measured. To our knowledge, no studies have tracked changes in peripheral muscle deoxygenation during repeated-sprint exercise. Because a diminished oxygen availability (as a result of hypoxia) results in a greater accumulation of blood lactate and an impaired ability to repeat sprint exercise (4), and because PCr recovery is strongly dependent on oxygen availability (14), we hypothesized that muscle deoxygenation may also be associated with the power decrements observed during repeated exercise.

Fatigue induced by exercise is known to not only be related to peripheral factors but to also have a central component as demonstrated by a voluntary activation deficit (12). It has been suggested that a central component was preponderant in the force decrement of respiratory muscles after exercise (27) and represents a protective mechanism to prevent myocardial ischemia during high-intensity exercise (22). Central fatigue has also been suggested to "protect" the muscle from further peripheral fatigue (and damage), but at the expense of a truly maximal performance (12). This deficit in muscle activation has previously been observed after prolonged continuous exercise (19,20) but, to the best of our knowledge, has never been investigated during repeated, short-duration sprints. Consequently, the purpose of this work was to investigate whether neural drive changes occur in parallel to muscle deoxygenation during this type of exercise. We hypothesized that, in parallel with muscle deoxygenation, the decline in maximal power output during repeated-sprint exercise might be associated with a decrement in muscle activation.

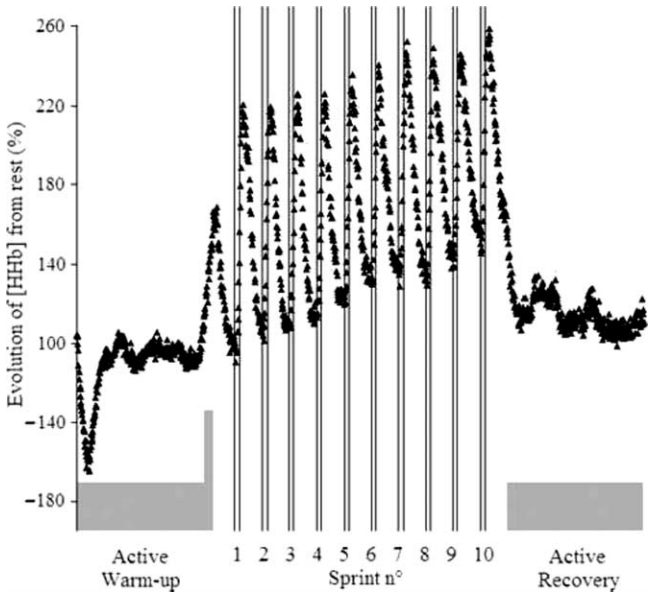

FIGURE 1—Evolution of muscle deoxyhemoglobin (HHb) during repeated-sprint exercise as a percentage of the resting value. Values are the means for all subjects (*N* = 9); *vertical bars* represent the sprints.

MATERIALS AND METHODS

Subjects. Nine males gave written informed consent to participate in this study. All of the subjects practiced regular physical training (6 ± 3 h·wk^{-1}), and none of them suffered from muscle soreness, lower-limb injuries, or peripheral vascular disorders. Their mean age, height, and body mass were 25 ± 2 yr, 178 ± 6 cm, and 69.2 ± 8.4 kg, respectively. Subjects were asked to avoid caffeine intake during the 8 h preceding the test and to avoid all vigorous activity during the 24 h preceding the test. The study was approved by the local ethics committee, and the study protocol complied with the Helsinki Declaration for human experimentation. All experimental procedures were performed in accordance with the policy statement of the American College of Sports Medicine on research with human subjects as published by *Medicine and Science in Sports and Exercise*®.

Experimental procedure. Two to three days before starting the experiment, subjects were asked to come to the laboratory for a familiarization session to be familiar with the testing procedures and to allow determination of the optimal electric stimulation intensity for the superimposed twitch. During these preliminary tests, subjects performed maximal voluntary contractions and a number of sprints at their desired intensity, enabling them to be accustomed with the equipment and allowing the experimenters to determine appropriate individual adjustments for the ergocycle and knee extensor device. The subsequent test session was conducted as follows: 30 min of rest, including preparation of the subject; 3 min of active warm-up on the ergocycle (frequency: 60 rpm, load: 14 N); 30 s of rest; and 10×6-s sprints interspersed with 30 s of rest and 3 min of active recovery (frequency: 60 rpm, load: 14 N). Active warm-up and active recovery were performed for safety reasons. Active warm-up, active recovery, and the repeated-sprint tests were conducted on a modified friction-loaded cycle ergometer (Monark type 818E, Stockholm, Sweden). The braking resistive force applied on the flywheel was set at 14 N for both the warm-up and posttest recovery, and at 0.9 N·kg^{-1} of body mass for the maximal sprints. The load for the sprint test was applied on the flywheel during the final 6 s of the warm-up. The protocol is represented at the bottom of Figure 1.

Cycling repeated-sprint test. The standard friction-loaded cycle ergometer was specifically equipped with both a strain gauge (Interface MFG type, Scottsdale, AZ) and an optical encoder (Hengstler type RIS IP50, Aldingen, Germany) (3). The strain gauge and the optical encoder measured the friction force applied to the belt and the flywheel displacement, respectively. The flywheel velocity was determined using a first-order derivation of the flywheel displacement, and the power output was calculated from the product of total force and flywheel velocity. The values of power output were collected at 50 Hz and were sent to a personal computer for subsequent analysis. The cycle was equipped with toe clips to prevent the subjects' feet from slipping. For all tests, subjects were in the seated position during rest, exercise, and subsequent recovery. All tests started from a dead stop, with the front pedal crank at approximately 45° to the horizontal to facilitate the best starting push.

The repeated-sprint test consisted of 10×6-s maximal sprints interspaced by 30 s of recovery (11). During the 30-s recovery between sprints, subjects remained seated on the bicycle and were allowed to follow the countdown. Five seconds before starting each sprint, subjects were asked to assume the ready position and to wait for the start signal, which was announced by a countdown given by the experimenter. Strong verbal encouragement was provided to each subject during all sprints.

Alveolar oxygen uptake and muscle oxygenation. Oxygen uptake was recorded during the entire cycling procedure (from warm-up to recovery). Expired gases were collected by a mask enclosing both the mouth and nose and were measured breath-by-breath (ZAN 680, Oberthulba, Germany). The values of oxygen consumption were averaged to obtain one value for each sprint-recovery cycle. Muscle deoxygenation was assessed using the near-infrared spectroscopy (NIRS) technique (8). This technique provides continuous, noninvasive monitoring of the relative concentration changes in deoxyhemoglobin concentration ([HHb]). In the present study, changes in muscle oxygenation of the right vastus lateralis (VL) were continuously monitored at 2 Hz using a near-infrared spatially resolved spectroscopy oximeter (NIRO-300, Hamamatsu Photonics, Japan). Data were simultaneously transmitted to a personal computer using an RS-232C wire. The optodes were housed in an optically dense plastic holder, thus ensuring that the positions of the optodes, relative to each other, were fixed and invariant. The optode assembly was secured on the cleaned skin surface with tape and then covered with a black homemade cotton tissue, thus minimizing the intrusion of extraneous light and loss of infrared light from the field of interrogation. The probe was placed overlying the lower third of the VL muscle of the right limb, parallel to the major axis of the thigh. The detector in the NIRS probe was separated from the light source by 40 mm. The light emitted by the infrared probe is assumed to reach a tissue depth of 50% of the interoptode spacing (space between emitting and receiving probe) (17). Skinfold thickness was measured between the NIRS optodes using a skinfold caliper (Holtain Ltd., Crymmych, UK) and was divided by two to determine the adipose tissue thickness (fat + skin layer) covering the muscle. The obtained value was 4.6 ± 1.9 mm, which is well below the 1.5 cm necessary to allow the NIRS photons to penetrate through to the muscle (17). The absorption of light at different wavelengths (775, 810, 850, and 910 nm) was analyzed according to the modified Beer–Lambert law (8). A differential pathlength factor (DPF) of 3.8 was used for the VL muscle (9). Thus, changes in [HHb] are reported as an absolute change from baseline. It has been proposed that HHb is less sensitive to blood volume changes than its counterpart, HbO_2 (10). Thus, we used HHb as an estimator of changes in intramuscular oxygenation.

Electromyography acquisition and analysis. Electromyograms (EMG) were obtained using MP30 hardware (Biopac Systems Inc., Santa Barbara, CA) and dedicated software (BSL Pro Version 3.6.7, Biopac Systems Inc., Santa Barbara, CA). Surface EMG from the muscle bellies of the vastus lateralis (VL) and biceps femoris (BF) were recorded during the repeated-sprint exercise. We used bipolar Ag/AgCl electrodes (Contrôle Graphique Medical, Brie-Comte-Robert, France) with a diameter of 9 mm and an interelectrode distance of 25 mm. The ground electrode was placed on the wrist. Before electrode placement, the skin was lightly abraded and washed to remove surface layers of dead skin, hair, and oil. The impedance of the EMG collector was always less than 5 kΩ. The myoelectric signal was amplified and filtered (band pass 30–500 Hz, gain = 1000), and the frequency of data collection was 2000 Hz. Subjects wore a muff net to prevent cable movement during cycling.

During the cycling sprints, the activity of each muscle was determined by measuring the mean value of the root mean square (RMS) of the signal between the onset and the end of the burst. Burst onsets and offsets were determined using a constant electric threshold of ± 0.2 mV (6). Values of RMS for the burst of each pedal revolution were averaged within each sprint to obtain one value for each of the 10 sprints. Because maximal power is generally attained in 1.8 s, during the third pedal revolution (16), values of RMS for the second and the third pedal revolutions were also averaged to obtain one value for the peak power development phase of each sprint. Data from warm-up and recovery were not considered because of the poor signal-to-noise ratio.

Maximal voluntary force. Before (at rest) and after (2 min after the end of the recovery period) the cycling exercise, subject performed a 5-s isometric maximal voluntary contraction (MVC) to measure the maximal voluntary force of the knee extensor muscles. The subjects were seated on the edge of a custom-made padded bench approximately 1 m in height with a 2- to 3-cm gap between the back of the knee and the edge of the bench. The knee angle was set at 80° (full extension represents 0°). The knee extension force was measured using a strain gauge connected between the stationary bench and the subject's ankle. Verbal encouragements were given throughout the contraction time.

Preexercise versus postexercise investigation of muscle contractility was supplemented by recording both mechanical twitch and M-wave (see below) signals at rest after electric stimulation of the femoral nerve. The femoral nerve was stimulated using a monopolar cathode electrode located in the femoral triangle. The anode was positioned in the gluteal fold. Electric stimulations were delivered by a high-voltage stimulator (Digitimer DS7AH, Digitimer, Hertfordshire, England). Three stimuli, each separated by a 5-s interval, were delivered. Each twitch duration was 0.2 ms, the tension was 400 V, and the amperage of stimulation was adjusted for each subject during the learning session to the amperage needed to obtain a maximal mechanical response. The peak twitch force production was obtained from the mechanical response, and the peak-to-peak amplitudes of the concomitantly electrically evoked compound action potentials were recorded as M-waves. The data from the three stimuli were averaged.

During the MVC, we calculated the neuromuscular efficiency as the ratio of the force developed divided by RMS activity of the VL (NME = force/RMS activity) for a duration of 1 s during the plateau of force production. This ratio was considered to represent an indicator of the peripheral muscle contractility (24). Furthermore, a superimposed twitch during the MVC plateau and a potentate twitch 4 s after the end of the MVC were elicited to

estimate the percentage of voluntary activation (1). According to the twitch-interpolated method (1), the percentage of muscle activation by voluntary drive was calculated as follows: voluntary activation (%) = (1 − superimposed twitch/control twitch) × 100.

Statistical analysis. Each variable was tested for normality using the kurtosis test with acceptable Z values not exceeding ± 1.5. With the assumption of normality confirmed, parametric tests were performed. The effect of the repeated exercise was analyzed for each variable by a one-way analysis of variance with repeated measures for sprint number. A *post hoc* Fisher least significant difference test was used when necessary to determine where significant differences occurred. Data are reported as means ± SD, and statistical significance was set at $P < 0.05$.

RESULTS

Global changes during repeated-sprint exercise. During the repeated-sprint test, there was a significant peak power decrement across the sprint repetitions (from 23.3 ± 2 W·kg^{-1} during sprint 1 to 20.7 ± 2 W·kg^{-1} during sprint 10, $F_{9,72} = 11.39$, $P < 0.0001$, Fig. 2). Concomitantly, statistical analyses also revealed a global increase in oxygen uptake (from 2.1 ± 0.4 L·min^{-1} during sprint 1 to 2.8 ± 0.5 L·min^{-1} during sprint 10, $F_{9,72} = 12.45$, $P < 0.0001$, Fig. 2).

Muscle deoxygenation during repeated-sprint exercise. The increase in [HHb] engendered by each sprint (mean increase of +21.34 ± 1.56 μmol·L^{-1} during the sprint) remained constant throughout the sprint repetitions ($F_{9,72} = 0.58$, $P = 0.81$, Fig. 1). However, at the end of each recovery period, [HHb] failed to return to the end-recovery value of the previous sprint ($F_{9,72} = 3.43$, $P < 0.002$, Fig. 1). This incomplete recuperation led to a further increase in the value of [HHb] attained at the end of each sprint with the repetition of the sprints ($F_{9,72} = 5.30$, $P < 0.0001$, Fig. 1).

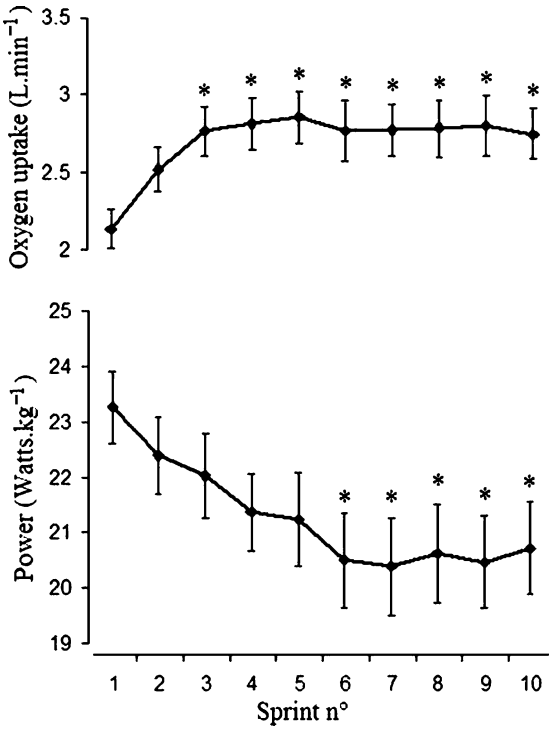

FIGURE 2—Oxygen uptake and maximal power output across repeated-sprint cycling exercise. Values are means ± SEM. * Significantly different from the first sprint.

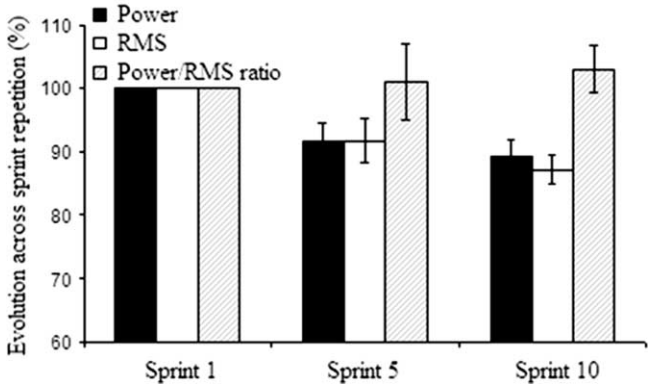

FIGURE 3—Evolution of maximal power output (*black column*), RMS activity of the vastus lateralis during the second and third pedal revolutions (*white column*), and power/RMS ratio (*cross-hatch column*). The data are displayed as percentages of the values recorded during the first sprint.

Electromyographic activity during repeated-sprint exercise. The RMS activity of the agonist muscle (VL) during each sprint did not significantly change across the sprint repetitions ($F_{9,72} = 0.42$, $P = 0.92$; mean value of 0.52 ± 0.04 mV during sprint 1 vs 0.51 ± 0.05 mV during sprint 10). In contrast, the RMS activity of the BF decreased significantly with the repetition of sprints ($F_{9,72} = 2.59$, $P < 0.02$; mean value of 0.29 ± 0.03 mV during sprint 1 vs 0.26 ± 0.03 mV during sprint 10). Whereas global RMS activity of the VL during the 6-s sprint failed to fluctuate across the sprint repetitions, this parameter decreased significantly during the second and the third pedal revolutions with sprint repetitions ($F_{9,72} = 2.69$, $P < 0.01$; mean value of 0.70 ± 0.06 mV during sprint 1 vs 0.60 ± 0.05 mV during sprint 10), resulting in a constant peak power/RMS ratio ($F_{9,72} = 0.87$, NS, Fig. 3).

Maximal voluntary contractions. Despite the active recovery period allowed after the repeated-sprint exercise, our results showed a significant decrease of MVC from rest to postexercise (from 747 ± 177 N to 624 ± 152 N, −16.5%, $F_{1,8} = 46.80$, $P < 0.0001$). This force decrement was associated with a significant impairment in NME (force/RMS activity, −11.4%, $F_{1,8} = 5.37$, $P < 0.05$). Furthermore, despite an increment in muscle excitability during electric stimulation (estimated from M-wave amplitude, +13.7%, $F_{1,8} = 20.45$, $P < 0.002$), there was a decrement in peak twitch force production (−9%, $F_{1,8} = 2.08$, NS). This was associated with a significant decrement of the percentage of voluntary activation estimated by the twitch-interpolation method (from 94.0% before exercise to 91.5% after exercise, $F_{1,7} = 8.57$, $P < 0.02$). Finally, there was also a significant decrement of the RMS/M-wave ratio (−14.5%, $F_{1,8} = 5.953$, $P < 0.05$).

DISCUSSION

The purpose of this study was to investigate fatigue-related changes (i.e., power output decrement) during repeated high-intensity sprints. Consistent with previous research (7,11), our data showed a significant power decrement during repeated-sprint exercise. For the first time, however, our data indicate that there is an increase in muscle deoxygenation during repeated sprints of short duration (6 s) interspersed with brief (30 s) recovery. According to our hypothesis, we also demonstrated evidence of a concomitant decrease in neural drive to the muscle that could be partly responsible for the observed decrement in power output.

Oxygen uptake and muscle deoxygenation. In this study, we observed a significant increase in $\dot{V}O_2$ with the repetition of sprints, whereas the maximal power output decreased (Fig. 2). It is interesting to note that this increased aerobic contribution lead a mean relative $\dot{V}O_2$ of 39.7 $mL \cdot min^{-1} \cdot kg^{-1}$ during the last sprint recovery period. Thus, although an increase in aerobic metabolism ($\dot{V}O_2$) occurred when subjects performed repeated sprints, this increase did not allow them to maintain their initial power output. The decrease in power output observed in this study, despite an increase in aerobic metabolism, can largely be attributed to the previously reported decrease in anaerobic metabolism that occurs during the latter sprints of a repeated-sprint test (11) (as well as changes in neural drive to the muscle, as explained below).

Accompanying this rise in $\dot{V}O_2$, our data demonstrate a significant increase in muscle deoxygenation during the exercise repetitions. This suggests that homeostatic adjustments were insufficient to maintain the capillary O_2 concentration during repeated-sprint exercise, resulting in a mismatch between O_2 supply and the increasing muscle demand. At the onset of a bout of intense exercise, there is a delay in O_2 delivery to the working muscles (5). However, if the duration of the work period is limited to a few seconds, oxygen bound to myoglobin may buffer the initial oxygen demand of the exercise (25,28). During recovery, oxygen bound to myoglobin is fully replenished within 20 s of the cessation of a submaximal exercise (26). Theoretically, with such a rapid rate of restoration, it is unlikely that the availability of oxygen from myoglobin would be a limiting factor during repeated exercises. However, despite a recovery time of 30 s, our data displayed a significant increase in [HHb], suggesting a progressive anaerobiosis in the muscle.

This progressive muscle anaerobiosis seems to be the result of an inability to restore presprint levels of oxygen saturation during the 30 s of recovery after each sprint. Indeed, the amplitude of [HHb] decrement during recovery tends to be lower than the [HHb] increment during the preceding sprint (average elimination of 19 $\mu mol \cdot L^{-1}$ during recovery vs an average increase of 21 $\mu mol \cdot L^{-1}$ during sprints, $F_{1,8} = 4.61$, $P = 0.064$). However, from a practical point of view, our results showed that the increase in [HHb] engendered by each sprint remained fairly constant throughout each sprint repetition (+21.34 ± 1.56 $\mu mol \cdot L^{-1}$). This suggests that despite a progressive muscle deoxygenation, the ability of the subjects to use available O_2 throughout the entire repeated-sprint test was well preserved. In consequence, our data failed to support the hypothesis that muscle deoxygenation could be responsible for the power decrement observed across repetitions.

Changes in electromyography. Similar to the present study, Hautier et al. (15) examined changes in the EMG signal during brief, maximal, intermittent cycling exercise (15 maximal 5-s sprints with 25 s of rest between each sprint). The mean peak power output recorded in the last sprint by Hautier et al. (15) represented 88.7% of the value achieved during the first sprint; this value is close to the value of 89.2% observed in this study, suggesting a similar level of fatigue. They also observed that mean EMG activity (i.e., RMS) of the prime mover muscles (gluteus maximus and vastus lateralis) remained unchanged during the repeated sprints, despite the significant loss of power observed in the last sprint, suggesting a peripheral fatigue. Although consistent with the results of the present study, Hautier et al. (15) have argued that their observed 13.3% decrement in the activity of the BF could be attributed to an efficient adaptation of the intermuscular coordination to transfer reduced force and power to the pedal in the trained subject (15). Thus, Hautier et al. (15) propose that peripheral, rather than central, mechanisms of fatigue explain the decrease in repeated-sprint performance observed in their study.

Several limitations exist to the conclusion of Hautier et al. (15). First, although they consider the BF to be an antagonist, it could be considered an agonist during the upstroke of the pedal revolution if the cycloergometer is equipped with toe clips, as in the present study; however, this was not the case in the study by Hautier et al. Second, the maximal power output is mainly dependent on the early pedal revolutions, which could present a different motor pattern than a mean EMG value obtained during a 6-s exercise. Indeed, Martin et al. (16) have shown that when flywheel inertia is taken into account in the power calculation, maximal power is generally attained in 1.8 s during the third pedal revolution. If we specifically analyze the second and third pedal revolutions, our data show that the decrement in power output was accompanied by a decrease in EMG activity, namely, a reduction of EMG amplitude (i.e., RMS) of the VL muscle during repeated maximal sprints. As showed in Figure 3, power output represented 91.5 and 89.2% of the initial value for the 5th and the 10th sprints, respectively. At the same time, RMS activity of the VL represented 91.8 and 87.2% of the initial value for the 5th and the 10th sprints, respectively, without any variation of the power/RMS ratio (Fig. 3). This suggests that, in contrast to the results of Hautier et al. (15), there was also a possible decrement in neural drive to the muscle in the present study.

MVC and twitch-interpolation changes. Reduced neuromuscular activity can occur from a reduced neural

drive to the motoneurons, reflecting a central nervous system role in fatigue development during the present experimental conditions (12). However, a decrease in EMG amplitude might also be related to changes in sarcolemma excitability (23). To avoid this methodologic limitation, we calculated the RMS/M-wave ratio before and after the repeated-sprint exercise. This parameter showed a significant decrease (−14.5%, $P < 0.05$), suggesting a decrement in muscle activation. Furthermore, we also evaluated muscle activation by the twitch-interpolation method (1). Supporting our hypothesis, data from this method showed a significant decrease in voluntary activation from the pre- to postexercise MVC (−3.0%, $P < 0.05$).

This decrement in muscle activation has previously been observed after prolonged running and seems to depend on the race duration. Millet et al. (19) observed a decrement in voluntary activation of 27.7% after a 65-km race, but, according to the same authors, this decrement was only 7.6% after a 30-km race (20). For the first time, our data show a reduced maximum voluntary activation after repeated cycling sprints of short duration. In this study, the decrement in voluntary activation observed after 10 cycling sprints was functionally weak (i.e., 3%); this can likely be attributed to the short duration of the fatiguing exercise (i.e., 10 × 6 s). Although this small decrement may seem to be of only minor practical relevance, this decrement may increase and become more important during cycling road races and team sports, in which athletes perform many more sprints. The weakness of this decrement can also possibly be attributed to the controversy that exists regarding train versus single stimulation techniques. In fact, Miller et al. (18) have shown that a stimulation train elicits higher torque increment and improves the detection of a central activation failure compared with a single stimulation. However, Allen et al. (2) failed to observe differences in the responses to single, paired, and trains of four stimuli at high voluntary torques, and they suggested that single stimuli are adequate for estimating voluntary drive. We choose single stimuli because this protocol causes less discomfort for the subjects than multiple stimulations, and this method provides reliable measurements. Furthermore, if voluntary activation were measured immediately after the last sprint, rather than after the recovery period, it is likely that we would have observed a greater failure in voluntary activation. Although the postexercise recovery may have prevented us from observing the full extent of the changes in voluntary activation, we choose to include an active recovery to reduce the risk of vagal syncope. The precise mechanisms underlying the decrease in voluntary motor drive cannot be ascertained from the results of this study. They could reside in a physiological incapacity of the motor cortex to generate an appropriate command, a physiological failure in motoneuron pool excitability, and/or in both the conscious and unconscious will of the subject to reduce the exercise intensity.

CONCLUSION

In this study, muscle deoxygenation was used as a surrogate measure of peripheral metabolic perturbations. For the first time, our data show that despite progressive muscle deoxygenation, the ability of the subjects to use available O_2 throughout the entire repeated-sprint test was well preserved. In addition to possible decrease in anaerobic contribution and/or perturbations in contractile processes, our results showed that the decrement in power output was associated with a decrease in neural drive to the muscle. Indeed, data showed a significant decrement in the RMS activity during the acceleration phase of each sprint across repeated-sprint exercise. Moreover, the decrement in neural drive was confirmed after exercise by a significant decrease in both the percentage of voluntary activation and RMS/M-wave ratio. This is the first time that a neural drive failure has been demonstrated for repeated short-duration sprints. This voluntary activation deficit could represent a protective mechanism to the progressive muscle deoxygenation observed in this study (12,21,22) mediated by III and IV afferences (12). Further research is needed to investigate the cause of the activation failure observed in this study. This information may be useful in designing training protocols to help delay this failure and to enhance athletes' repeated-sprint performance.

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