Time Course of Recovery after Cycling Repeated Sprints

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[1]Post-Graduate Program in Movement Science, Laboratory of Physiology and Sport Performance (LAFIDE), São Paulo State University (UNESP), Bauru, SP, BRAZIL; [2]Human Performance Laboratory, Faculty of Kinesiology, University of Calgary, Calgary, Alberta, CANADA; [3]Laboratoire Interuniversitaire de Biologie de la Motricité, UJM Saint-Etienne, Université de Lyon, EA 7424, Saint-Etienne, FRANCE; and [4]Institut Universitaire de France, Paris, FRANCE

ABSTRACT

MILIONI, F., R. A. AZEVEDO, A. M. ZAGATTO, and G. Y. MILLET. Time Course of Recovery after Cycling Repeated Sprints. *Med. Sci. Sports Exerc.*, Vol. 53, No. 2, pp. 413–420, 2021. **Purpose:** The present study investigated the recovery of performance and neuromuscular fatigue after cycling repeated sprints. **Methods:** Ten participants performed two sessions of repeated sprints (one session: 10 × 10-s sprints, 30-s recovery) separated by 24 h (R24-S1 and R24-S2) and two sessions separated by 48 h (R48-S1 and R48-S2). The recovery condition (i.e., 24 or 48 h) was randomized and separated by 1 wk. All sessions were performed on a recumbent bike, allowing minimal delay between sprints termination and neuromuscular measurements. Neuromuscular function of knee extensors (neuromuscular assessment [NMA]) was assessed before sessions (presession), after the fifth sprint (midsession), and immediately after (postsession). Before sessions, baseline NMA was also carried out on an isometric chair. The NMA (bike and chair) was composed of maximal voluntary contraction (MVC) of knee extension and peripheral neuromuscular stimulation during the MVC and on relaxed muscle. **Results:** The sprints performance was not significantly different between sessions and did not presented significant interaction between recovery conditions. MVC was significantly lower at R24-S2 compared with R24-S1 (−6.5% ± 8.8%, $P = 0.038$) and R48-S2 (−5.6% ± 8.2%, $P = 0.048$), whereas resting potentiated high-frequency doublet (Db100) was lower at R24-S2 compared with R24-S1 (−10.4 ± 8.3, $P = 0.01$) (NMA on chair). There were significant reductions in MVC (>30%, $P < 0.001$) and Db100 (>38%, $P < 0.001$) from pre- to postsession in all sessions, without significant interactions between recovery conditions (NMA on bike). **Conclusion:** Cycling repeated sprints induce significant fatigue, particularly at the peripheral level, which is fully restored after 48 h, but not 24 h, of recovery. One versus two days of recovery does not affect neuromuscular fatigue appearance during cycling repeated-sprint sessions. **Key Words:** FATIGUE, CENTRAL FATIGUE, PERIPHERAL FATIGUE, FORCE, POWER, PERFORMANCE

The capacity to perform intermittent all-out efforts with short recovery periods (i.e., <30 s) is determinant of success in many sports (1). However, performing repeated sprints has a very high energetic/metabolic cost (2) and neuromuscular demand (3,4), leading to a high level of fatigue (3,5). Fatigue can be seen as a reversible decline in neuromuscular function and/or the ability to produce power (4). Fatigue can affect the peripheral mechanisms (i.e., distal to the neuromuscular junction) and the central nervous system. Peripheral aspects of neuromuscular fatigue can be assessed by the decrement in potentiated evoked forces, such as high-frequency doublet (Db100) and maximal M-wave amplitude evoked by peripheral neuromuscular stimulation (PNS) (6). The central aspects of neuromuscular fatigue are usually evaluated through changes in normalized electromyographic signal amplitude during maximal voluntary contraction (MVC) (7,8) and level of maximal voluntary activation (VA) (3,5,9).

After repeated sprints, neuromuscular function appears to remain substantially impaired over long periods of time (1,4,5) so that force production (5,9) and sprint performance (5,10) do not fully recover within a few hours. This residual fatigue is a factor that must be taken into account for the subsequent training session or competition because incomplete recovery can lead to deteriorated performance, chronic fatigue (i.e., overtraining), and injuries (11).

Despite the increased research interest in recovery strategies after repeated sprints, the time course to return to initial performance and neuromuscular function is still poorly understood (4,5). The few studies that have investigated the time course of performance and neuromuscular recovery after cycling repeated sprints showed that MVC, Db100 (i.e., peripheral fatigue), VA (i.e., central fatigue), and sprint performance were still depreciated 24 h after a repeated-sprint protocol (4,5,12); however, no studies performed monitoring for more than 24 h. In addition, the usual time delay for neuromuscular fatigue assessment (1–3 min after effort termination) is a problematic issue that can largely affect the measured level of central

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and peripheral fatigue (13) because significant recovery has been shown to occur within 1–2 min (13–15).

Because of the few/inconclusive data available about how long the residual effects of fatigue can last, and how these can affect cycling repeated-sprint performance, the aim of the present study was to investigate the time course of repeated-sprint performance and neuromuscular function recovery after cycling repeated sprints by comparing 24 versus 48 h of recovery. Using an innovative cycling ergometer that allows neuromuscular fatigue assessment within 1 s after cycling (13), we hypothesized that repeated-sprint performance in cycling and neuromuscular function would remain impaired after 24 h but would be fully restored after 48 h of recovery.

METHODS

Participants

The sample size was calculated using G*Power software (University of Düsseldorf, Germany), based on the assumption that ten 10-s all-out cycling sprints can induce significant MVC decrement with an effect size (ES) of 1.7 (16). Using a statistical power of 95% and an alpha level of 0.05, the sample size calculated was $n = 7$ participants. Thus, 10 healthy men participated in the study (age, 28 ± 4 yr; body weight, 69.8 ± 5.6 kg; $\dot{V}O_{2max}$, 45.7 ± 7.3 $mL \cdot kg^{-1} \cdot min^{-1}$). The participants were asked to refrain from alcohol and caffeine for at least 24 h and eat a light meal about 2 h before the experimental procedures.

All participants were nonsmokers and did not present musculoskeletal or cardiorespiratory issues. Participants were informed of the experimental protocol and all associated risks and completed the PAR-Q+ questionnaire before giving written informed consent. All procedures were in accordance with the Declaration of Helsinki and were approved by the University of Calgary Conjoint Health Research Ethics Board (REB17-1032).

Experimental Design

Five environmentally controlled experimental visits were carried out in separate days (temperature ~21°C and relative humidity ~40%). In the first visit (day 1), the participants performed an incremental test to determine the maximal oxygen uptake ($\dot{V}O_{2max}$) and peak power output. Next, the participants were familiarized with the repeated-sprint protocol on the recumbent cycle ergometer, MVC, and PNS. From days 2 to 5, repeated-sprint sessions and neuromuscular assessment (NMA) were performed. The participants randomly performed two repeated-sprint sessions separated by 24 h (R24-S1 and R24-S2) and two repeated sprints sessions separated by 48 h (R48-S1 and R48-S2). The recovery conditions (i.e., 24 h recovery or 48 h recovery) were separated by 1 wk. The participants were requested to avoid heavy physical activities and maintain their usual nutritional habits during the recovery conditions. The repeated-sprint sessions were composed of ten 10-s all-out sprints with 30 s of active recovery at 20 W and ~60–70 rpm. Before all repeated-sprint sessions, the baseline neuromuscular function was measured on an isometric chair. Subsequently, the participants were transferred to the recumbent bike and performed two MVC with PNS before (presession) and immediately after the repeated sprints (postsession), as well as one MVC with PNS immediately after the fifth sprint (midsession) (Fig. 1).

Incremental Test

The incremental test started with a 5-min warm-up at 20 W, and the power output was increased by 25 $W \cdot min^{-1}$ until task failure, defined as the inability to maintain the pedal cadence at 80 ± 5 rpm. Oxygen uptake, carbon dioxide production, and ventilation were monitored breath by breath, using a metabolic cart (Quark CPET, Cosmed, Rome, Italy). Heart rate was measured by a heart rate monitor (GarminInternational, Schaffhausen, Switzerland) synchronized with the metabolic cart. Calibration was performed before each test following the manufacturer's instructions, and the points obtained were smoothed (each 10 points) and interpolated to consider 1 value per second (OriginPro 8.0; OriginLab Corporation, Northampton, MA). $\dot{V}O_{2max}$ was determined as the highest $\dot{V}O_2$ average obtained in the final 30 s of the exercise stage, when at least two of the following criteria were met: 1) $\dot{V}O_2$ stabilization of the final two stages of exercise (<2.1 $mL \cdot kg^{-1} \cdot min^{-1}$); 2) respiratory exchange ratio >1.1; and 3) maximal heart rate >90% of maximal predicted heart rate (17). In all cases, the criteria were met and the $\dot{V}O_{2max}$ was successfully determined.

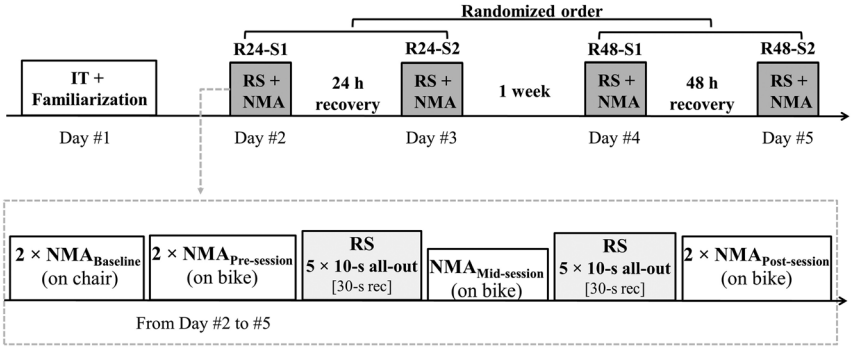

FIGURE 1—Representation of the experimental design. IT, Incremental test; RS, Cycling repeated-sprint; NMA, Neuromuscular assessment.

Cycling Repeated Sprints

The repeated-sprint sessions started with a 5-min warm-up at 20 W followed by two 5- to 7-s submaximal sprints. The participants performed ten 10-s all-out sprints with 10 s of passive recovery and 20 s of active recovery (20 W at ~60–70 rpm) between sprints, totaling 30 s of recovery. The participants were instructed to begin increasing the pedal cadence 3 s before starting the sprint and then pedal as fast as possible during the entire sprint (without any pacing strategy). Strong verbal encouragement was provided by the evaluators throughout the repeated-sprint sessions. The sprint torque factor was set at 6% of individual body weight. Peak blood lactate concentration was measured in all repeated-sprint sessions. Blood samples (2 μL) were collected from the fingertips at 3 and 5 min after sprints and analyzed using a portable lactate analyzer (Lactate Scout; SensLabGmb, Leipzig, Germany). RPE was recorded using Borg's RPE scale (18) to assess subjective perception of effort immediately after the termination of the repeated-sprint sessions.

NMA

Before each session, a 1-ms rectangular single stimulus was delivered incrementally until twitch plateau and maximal M-wave amplitudes were reached. The stimulation intensities were adjusted to 130% to ensure maximal twitch and M-wave amplitudes. The stimulation intensities were as follows: R24-S1, 117 ± 61 mA; R24-S2, 111 ± 63 mA; R48-S1, 121 ± 55 mA; and R48-S2, 109 ± 52 mA. The NMA started with an approximately 5-min warm-up during which the participants performed submaximal MVC (3 at 25%, 3 at 50%, and 2 at 75% MVC). After the warm-up, the baseline neuromuscular measurements were carried out both on an isometric chair and on a recumbent bike (presession). NMA was also carried out on the bike after the fifth sprint (midsession) and immediately after the repeated sprints (postsession). NMA (i.e., chair for baseline measurements and recumbent bike during repeated sprints) was composed of a 5-s MVC superimposed at peak MVC force plateau by a high-frequency doublet at 100 Hz (19). Approximately 3 s after the MVC, a sequence of stimulation was delivered on the relaxed muscle, composed of a potentiated high-frequency doublet at 100 Hz (Db100), a potentiated low-frequency doublet at 10 Hz (Db10), and a singlet (Pt), 3-s apart from each other. The EMG activity of the vastus lateralis (VL) muscle was monitored during the entire procedure. The sequence was applied twice during the baseline measurements, presession and postsession, separated by 10 s, and only once at midsession. The trial that presented the highest MVC was used for baseline on the chair and presession on the recumbent bike, whereas the first trial was considered for posttest. During all NMA, the EMG activity of the VL muscle was monitored.

Instrumentation and Data Analysis

Isometric chair. For the baseline NMA, participants were evaluated on an isometric chair secured by chest and hip straps, with knees and hips flexed at 90°. The right ankle was attached perpendicularly to a force transducer (LC101-2 K; Omegadyne, Sunbury, OH). Visual feedback was provided during the MVC attempts.

Recumbent cycle ergometer. Both incremental tests and repeated sprints were carried out on a recumbent cycle ergometer with an electromagnetically braked Velotron system (Racermate Inc., Seattle, WA). The incremental test was empowered by Velotron Coaching Software (Racermate Inc.), while during the repeated sprints the sprint power was acquired at 10 Hz using Velotron Wingate Software (Racermate Inc.). Peak power was determined as the best peak power output achieved among the 10 sprints of the session, whereas the average power output was determined as the sum of the peak power output of each sprint of the session divided by 10. The performance decrement was also calculated using a formula derived from Glaister et al. (20) [100 × (sum of sprints peak power)/(number of sprints × highest sprint peak power) − 100].

Force measurement during MVC. The recumbent bike allows the pedals to be locked instantly (2–3 s of delay after cycling interruption), maintaining the cranks parallel to the ground and hip, knee, and ankle angles at approximately 100°, 90°, and 90°, respectively. The ergometer was validated by Doyle-Baker et al. (13). Participants were firmly attached at the hip and chest with noncompliant straps. MVC of the right leg was measured during the NMA by a wireless Power Force pedal force analysis system (Model PF1.0.0; Radlabor GmbH, Freiburg, Germany) located between the pedal and the crank. Force was sampled at 500 Hz and recorded using Imago Record software (version 8.50, Radlabor GmbH) (13,21). To provide real-time visual force feedback during the MVC, the force signal was transmitted to a PowerLab system (16/35; AD Instruments, Bella Vista, Australia) using a National Instruments 16-bit A/D card (NI PCI-6229; National Instruments, Austin, TX) and a connector block (BNC-2111, National Instruments) and displayed on a large computer monitor positioned in front of the participant.

PNS. Electrical stimuli were delivered via a constant-current stimulator (DS7A; Digitimer, Welwyn Garden City, Hertfordshire, UK) to the right femoral nerve via a cathode electrode (10 mm stimulating diameter; Meditrace 100, Covidien, Mansfield, MA) in the inguinal triangle and 50 × 90 mm rectangular anode electrode (Durastick Plus; DJO Global, Vista, CA) at the femoral greater trochanter level. All stimuli lasted 1 ms; however, Db100 and Db10 were composed of doublets separated by 10 and 100 ms, respectively.

EMG data acquisition. EMG of the VL was recorded with pairs of self-adhesive surface electrodes (10 mm recording diameter) (Meditrace 100, Covidien) positioned at distal part of the muscle (22) in a bipolar configuration with a 30-mm interelectrode distance. The reference electrode was placed on the patella. A low impedance (<10 kΩ) between electrodes was obtained by shaving and gently abrading the skin with sandpaper and then cleaning with isopropyl alcohol 70%. Signals were converted from analog to digital at a sampling rate of 2000 Hz by PowerLab system (16/35, AD Instruments)

and an octal bioamplifier (ML138, AD Instruments; common mode rejection ratio = 85 dB, gain = 500) with band-pass filter (5–500 Hz).

Data processing. MVC and EMG data were analyzed offline using LabChart 8 software (AD Instruments). The MVC peak force was determined as the highest force value recorded. The amplitudes evoked by Db100 and Db10 were measured in the force signal, allowing the calculation of the Db10:100 ratio that is needed to determine the type of peripheral fatigue (i.e., low- or high-frequency fatigue) (6). The VA was calculated as VA = [1 − (superimposed twitch amplitude/Db100)] × 100; however, when superimposed twitch was evoked slightly before or after the MVC force plateau, a correction was applied: VA = [1 − (superimposed twitch amplitude × (force level at stimulation/MVC)/Db100)] × 100 (23). M-wave maximal amplitude evoked by the singlet delivered to the relaxed muscle was used to normalize the root mean square (RMS) of the 500 ms during the MVC (RMS:M).

Statistical Analyses

All data are presented as mean ± SD, and the statistical analyses were performed using the software SPSS 17.0. The Shapiro–Wilk test confirmed the data normality. The sprint-by-sprint analysis was performed according the recovery condition (24 or 48 h of recovery) using a two-way repeated-measures ANOVA (sprint [1 to 10] × session [S1 and S2]). The comparison of sprint performance variables (i.e., peak power, average power, performance decrement, peak of blood lactate concentration, and RPE) as well as the baseline neuromuscular function (on chair) were analyzed using a two-way repeated-measures ANOVA (session [S1 and S2] × recovery condition [R24 and R48]). To analyze the neuromuscular function during the repeated-sprint sessions (on bike), a three-way repeated-measures ANOVA was used (moment [presession, midsession, and postsession] × session [S1 and S2] × recovery condition [R24 and R48]). Sphericity was tested using Mauchly's test, and if violated, degrees of freedom were corrected using Greenhouse–Geisser. When significant interactions were found, Sidak's *post hoc* test was used. The partial eta square from ANOVA was used as effect size (ES), and Cohen's *d* qualitative descriptors for ES interpretation were assigned as follows: <0.2, negligible effect; 0.2–0.39, small effect; 0.40–0.75, moderate effect; and >0.75, large effect. In all cases, a significance level of 5% was considered ($P > 0.05$).

RESULTS

Sprint by sprint. The greatest peak power was always the first sprint. The analysis of 24 h of recovery reveals significant decrement of sprints peak power in both session ($F_{1, 9} = 38.0$, $P = 0.0001$, ES = 0.81) and sprint–session interaction ($F_{1, 9} = 3.5$, $P = 0.01$, ES = 0.28). In particular, the first sprint was lower in R24-S2 compared with R24-S1 (Fig. 2A). Similarly, 48 h of recovery reveals significant decrement of sprints peak power in session ($F_{1, 9} = 23.2$, $P = 0.0001$, ES = 0.72); however, there was no significant sprint–session interaction ($F_{1, 9} = 0.62$, $P = 0.62$, ES = 0.07) (Fig. 2B).

Sprint performance. There were no significant differences between sessions ($F_{1, 9} < 4.4$, $P > 0.06$, ES < 0.35) and session–recovery condition interaction ($F_{1, 9} < 3.6$, $P > 0.09$, ES < 0.28) for any sprint performance variable (i.e., peak power, average power, performance decrement, peak of blood lactate, and RPE) (Fig. 3).

Neuromuscular function at rest. The baseline MVC (on chair) presented a significant session–recovery condition interaction ($F_{1, 9} = 5.6$, $P = 0.042$, ES = 0.38) with lower values of R24-S2 compared with R24-S1 ($P = 0.038$) and R48-S2 ($P = 0.048$). Db100 also presented a significant session–recovery condition interaction ($F_{1, 9} = 18.4$, $P = 0.002$, ES = 0.67) with lower values of R24-S2 compared with R24-S1 ($P = 0.003$), and despite no statistical significance, there was a trend for lower values of R24-S2 compared with R48-S2 ($P = 0.063$). No other baseline neuromuscular variables (i.e., Db10:100, VA, M-wave, and RMS:M) presented significant differences between sessions ($F_{1, 9} < 2.1$, $P > 0.18$, ES < 0.19) and session–recovery condition interaction ($F_{1, 9} < 3.2$, $P > 0.11$, ES < 0.26) (Fig. 4).

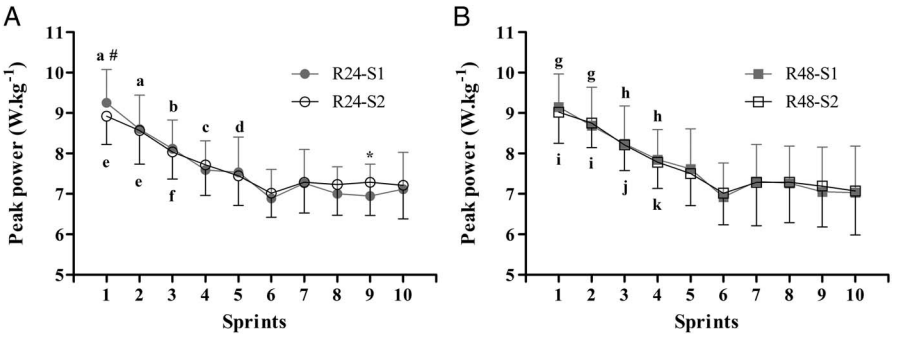

FIGURE 2—Sprint-by-sprint peak power output of the session before and after 24 h of recovery (A) and before and after 48 h of recovery (B). [a]Different compared with sprints 3 to 10 of R24-S1. [b]Different compared with sprints 6, 8, and 9 of R24-S1. [c]Different compared with sprints 6 and 9 of R24-S1. [d]Different compared with sprints 7, 9, and 10 of R24-S1. [e]Different compared with sprints 3 to 10 of R24-S2. [f]Different compared with sprints 6, 7, and 8 of R24-S2. [g]Different compared with sprints 5 to 10 of R48-S1. [h]Different compared with sprint 6 of R48-S1. [i]Different compared with sprints 5 to 10 of R48-S2. [j]Different compared with sprints 4, 5, 6, 9, and 10 of R48-S2. [k]Different compared with sprint 6 of R48-S2. #Different compared with sprint 1 of R24-S2. *Different compared with sprint 9 of R24-S2.

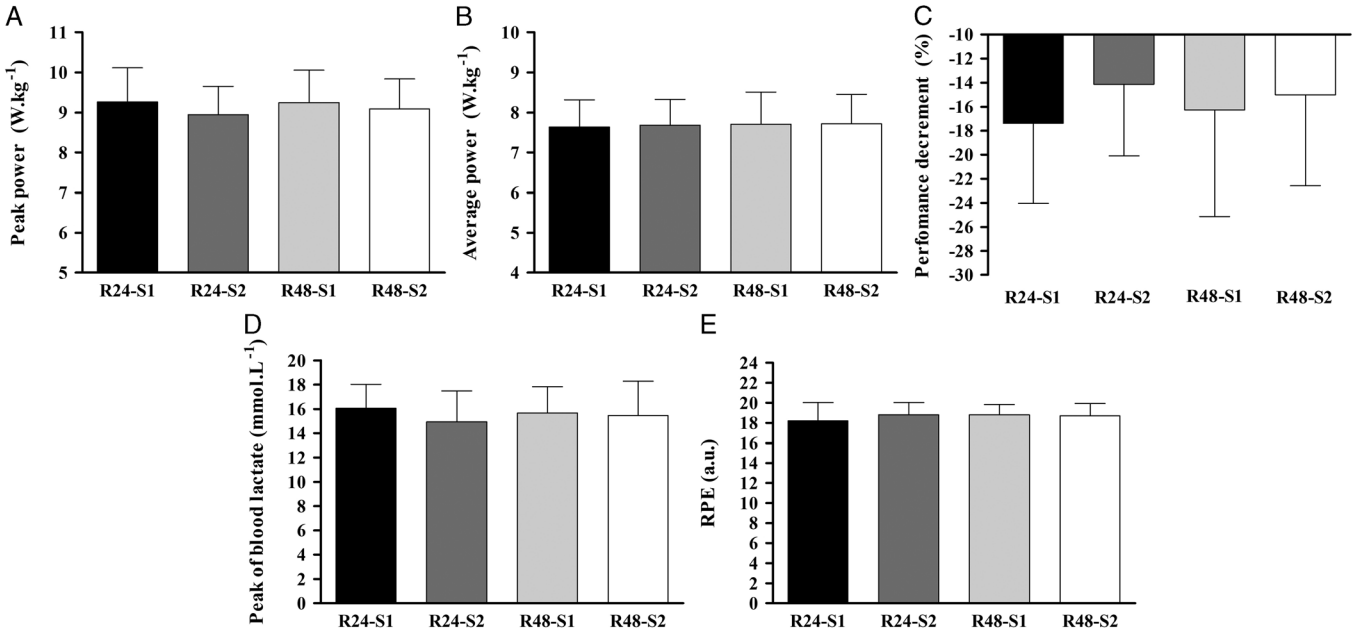

FIGURE 3—Raw sprint performance data. Peak power (A); average power (B); sprint performance decrement (C); peak of blood lactate concentration (D); RPE (E). *Different compared with R24-S1, $P < 0.05$.

Neuromuscular fatigue during the repeated-sprint sessions. Regarding neuromuscular fatigue during the repeated-sprint sessions, there were significant decrements in MVC ($F_{1.2,\ 10.5} = 39.5$, $P = 0.0001$, ES = 0.82), Db100 ($F_{1.1,\ 9.5} = 43.7$, $P = 0.0001$, ES = 0.83), and Db10:100 ($F_{2,\ 18} < 46.1$, $P = 0.0001$, ES = 0.84), with no moment–session–recovery condition interactions ($F_{2,\ 18} < 1.8$, $P > 0.20$, ES < 0.16). VA, M-wave, and RMS:M were not significantly altered ($F_{2,\ 18} < 3.4$, $P > 0.06$, ES < 0.27) and did not present significant interactions ($F_{1.2,\ 11.2} < 1.8$, $P > 0.21$, ES < 0.18) (Table 1).

DISCUSSION

The main purpose of the present study was to investigate the time course of cycling repeated-sprint performance and neuromuscular function recovery after cycling repeated sprints. Despite no significant differences in sprint performance between sessions, the cycling repeated sprints induced neuromuscular fatigue, including peripheral fatigue, which remained depressed after 24 h and was fully recovered in 48 h. No residual central fatigue was observed between sessions. These findings

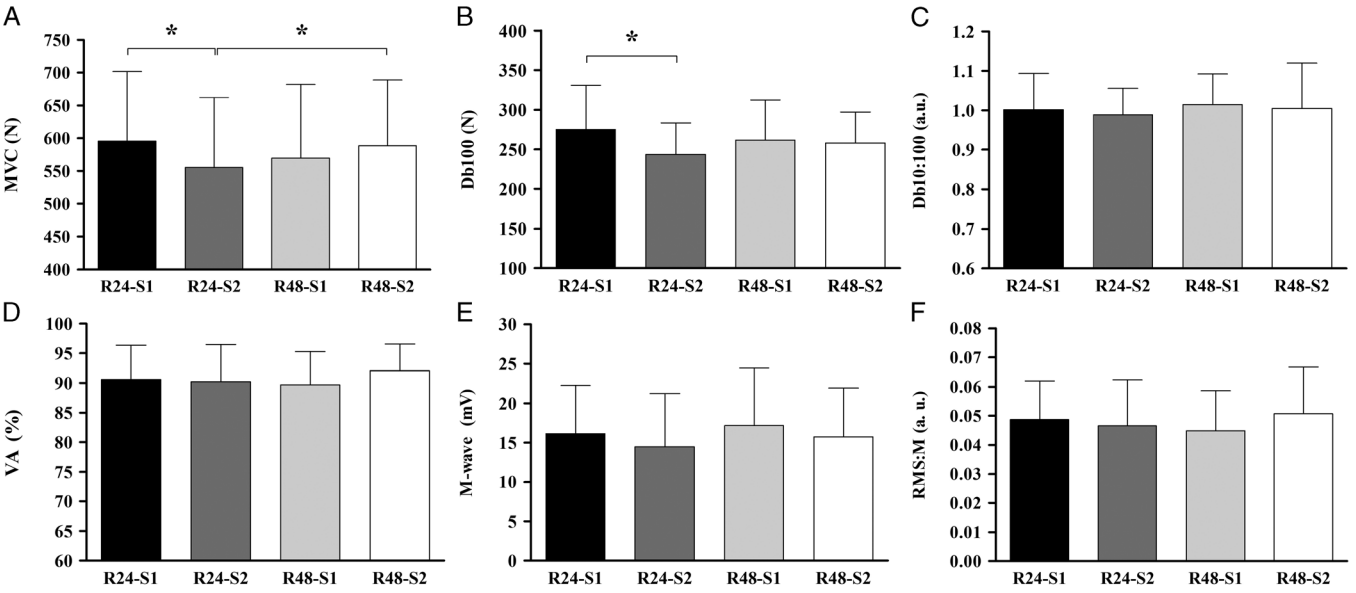

FIGURE 4—Raw neuromuscular function data at baseline. A, MVC of knee extension. B, Maximal amplitude of potentiated doublet 100 Hz (Db100). C, Ratio between the amplitude of potentiated doublet at 10 Hz and the potentiated doublet at 100 Hz (Db10:100). D, VA, E, Peak-to-peak maximal M-wave amplitude. F, Ratio between RMS and M-wave amplitude. *Different compared with R24-S1, $P < 0.05$.

TABLE 1. Neuromuscular measurements during the repeated-sprint sessions.

	R24-S1				R24-S2				R48-S1				R48-S2			
	Presession	Midsession	Postsession	Δ%	Presession	Midsession	Postsession	Δ%	Presession	Midsession	Postsession	Δ%	Presession	Midsession	Postsession	Δ%
MVC (*N*)	351.8 ± 72.1	249.6 ± 35.4[a]	232.3 ± 40.5[a,b]	−32.9 ± 10.8	345.5 ± 69.6	250.6 ± 35.4[a]	225.3 ± 43.2[a,b]	−34.0 ± 10.7	337.1 ± 69.1	250.1 ± 31.5[a]	220.8 ± 44.1[a,b]	−33.3 ± 12.7	342.3 ± 54.7	248.8 ± 33.6[a]	223.8 ± 44.5[a,b]	−33.9 ± 13.4
Db100 (*N*)	171.8 ± 37.3	109.2 ± 26.3[a]	105.5 ± 29.3[a]	−37.1 ± 19.0	175.5 ± 31.7	117.5 ± 28.1[a]	107.5 ± 24.2[a]	−38.0 ± 13.4	174.3 ± 40.6	106.4 ± 25.3[a]	100.2 ± 24.3[a]	−41.0 ± 14.4	173.0 ± 30.9	111.7 ± 24.3[a]	101.9 ± 23.2[a]	−39.4 ± 16.9
Db10:100	1.01 ± 0.13	0.78 ± 0.09[a]	0.76 ± 0.11[a]	−23.4 ± 14.0	0.99 ± 0.07	0.78 ± 0.13[a]	0.74 ± 0.11[a]	−24.7 ± 12.5	0.93 ± 0.15	0.81 ± 0.12[a]	0.75 ± 0.12[a]	−18.3 ± 12.8	0.97 ± 0.12	0.78 ± 0.13[a]	0.74 ± 0.10[a]	−22.7 ± 12.0
VA (%)	97.0 ± 3.4	95.5 ± 4.5	94.4 ± 4.8	−2.6 ± 5.5	96.5 ± 2.6	94.9 ± 8.5	93.1 ± 6.1	−3.1 ± 3.8	96.6 ± 2.6	95.1 ± 6.2	94.1 ± 5.0	−2.5 ± 4.5	96.9 ± 2.6	95.5 ± 5.1	94.2 ± 5.9	−2.8 ± 5.1
M-wave (mV)	14.0 ± 4.9	14.8 ± 4.5	15.3 ± 4.2	14.3 ± 24.4	15.4 ± 5.3	15.4 ± 5.3	15.3 ± 5.4	−0.3 ± 16.2	16.2 ± 6.2	16.6 ± 6.5	16.8 ± 6.5	3.9 ± 7.6	15.3 ± 5.2	15.3 ± 5.2	15.7 ± 5.5	2.9 ± 7.3
RMS:M	0.047 ± 0.017	0.038 ± 0.016	0.035 ± 0.010	−21.1 ± 24.2	0.042 ± 0.010	0.040 ± 0.016	0.037 ± 0.017	−14.0 ± 26.3	0.042 ± 0.013	0.038 ± 0.014	0.035 ± 0.017	−18.2 ± 24.9	0.043 ± 0.015	0.044 ± 0.019	0.041 ± 0.021	−7.6 ± 36.2

[a]Different of respective presession, $P < 0.05$.
[b]Different of respective midsession, $P < 0.05$.
Δ%, percentage of alteration between presession and postsession of the respective session; Db100, maximal amplitude of potentiated doublet 100 Hz; Db10:100, ratio between the maximal amplitude of potentiated doublet 10 Hz and doublet 100 Hz (Db10:100); M-wave, peak-to-peak maximal amplitude evoked by the singlet; RMS:M, ratio between RMS and M-wave amplitude.

provide the first indications about the recovery time course after a repeated-sprint session in cycling.

Fatigue induced by the repeated-sprint sessions. Fatigue is classically defined as an exercise-induced reduction in force generating capacity of the muscle (5), and its etiology can be attributed to several factors, such as peripheral failure, impaired motor drive, and altered afferent inputs innervating the active muscles (24). In the present study, the cycling repeated sprints induced peripheral fatigue (but no central fatigue) as evidenced by significant decrements in Db100 and Db10:100 from pre to mid- and postsession in all sessions. In line with the present study, Girard et al. (25) and Tomazin et al. (26) reported only peripheral fatigue (i.e., no VA deficit) after cycling repeated sprints. Isolated peripheral fatigue may be related to the accumulation of metabolic waste such as inorganic phosphate (due to PCr breakdown) and H^+ ions, which are known to diminish sensitivity of muscle fibers to Ca^{2+} and induce excitation–contraction coupling failure (27).

The absence of VA deficit in Girard et al. (25) and Tomazin et al. (26) might be related to the time delay between the sprint termination and the neuromuscular measurements (13). Several authors have shown the occurrence of both peripheral and central fatigue after cycling repeated sprints (28,29). For instance, Pearcey et al. (16) assessed neuromuscular fatigue at baseline, after the 5th sprint, and after the 10th sprint of a set composed of 10 × 10-s sprints with 180 s of recovery and showed that neuromuscular fatigue in the first five sprints was mainly peripheral, whereas in the final five sprints it was both peripheral and central. In the present study, the absence of VA decrements in all sessions, confirmed by the same behavior for RMS:M, suggests that the ability of the central nervous system to drive the knee extensors maximally during quadriceps isometric contractions was not significantly affected by the repeated sprints. The discrepancy between studies in the appearance of central fatigue after repeated sprints may be linked to participant characteristics (age, training background) and repeated sprints setup (time delay before neuromuscular measurements, sprints number, recovery duration, and characteristics) (25).

Interestingly, the magnitude of peripheral fatigue induced by the repeated sprints was similar between all sessions (i.e., R24-S1, R24-S2, R48-S1, and R48-S2), regardless of the recovery time (Δ% of Db100 ~ 39% in all sessions and Db10:100 ~ 21% in all sessions; Table 1). Hureau et al. (3) performed two sets of 10 × 10-s all-out sprints with 30-s recovery, one in a prefatigued condition induced by tetanic electrical stimulation and another in a control condition (i.e., nonfatigued), and found that peripheral fatigue was similar in both conditions (e.g., decrease in Db100 amplitude of ~51%) after the 10th sprint. Based on these outcomes, it is possible to speculate that independent of the preexisting fatigue level (induced by tetanic electrical stimulation in Hureau et al. [3] or incomplete recovery in the present study), there is a fine adjustment to limit the peripheral fatigue so as not to surpass the sensory threshold tolerance, preventing severe impairment in the locomotor muscle system (3,4,30). Although peak power

output of the first sprint of R24-S2 was significantly lower than for R24-S1 (Fig. 2), this was unlikely to happen in the present study. Indeed, if the same fine adjustment to limit the peripheral fatigue had been observed in the present study, one would have expected a significantly lower power output and a lower ΔDb100 during the session in R24-S2 compared with R24-S1. This was not the case.

Time course of repeated sprints and neuromuscular recovery. Incomplete recovery before the subsequent sprints session can affect performance and lead to fatigue accumulation (5). In the present study, the residual fatigue after repeated sprints was predominantly related to peripheral factors. With the measurements performed in the present study, we can only speculate that it is linked to glycogen depletion (31) and/or muscle damage (32). Indeed, unlike metabolite accumulation, which recovers quickly (i.e., within hours) (33), glycogen storage (34) and muscle damage (32) can take several days to return to baseline values and are associated with a reduction in force production, excitation–contraction coupling (E-C coupling), and intramuscular Ca^{2+} homeostasis (5).

Gavin et al. (35) induced glycogen depletion (exercise–diet intervention) and muscle damage (eccentric contractions) in the right leg of healthy males, with the left leg serving as a control. The authors found that MVC and twitch force were reduced up to 48 and 12 h, respectively, for the reduced glycogen leg, but not for the control leg. In addition, low-frequency fatigue (assessed as the decrease in the ratio between 20 and 50 Hz electrical stimulations) was found only for the reduced glycogen leg at 12 h after the protocol and was fully recovered at 24 h. Similarly, impaired MVC and a trend of Db100 impairment ($P = 0.063$) were found in the present study after 24 h of recovery (fully recovered at 48 h). Furthermore, in line with Gavin et al. (35), although the Db10:100 dropped during all repeated-sprint sessions (see Table 1), it returned to baseline values after 24 h of recovery.

It is however important to note that glycogen depletion and muscle damage levels are directly related to exercise characteristics. In particular, muscle damage depends on the amount of eccentric work. Thus, the results of the present study must be interpreted in the context of cycling repeated sprints because running and team-based sports present a substantially larger eccentric component (5). Assessments of muscle glycogen storage and muscle damage are extremely invasive, so that the absence of measurements of these variables is the major limitation of the present study. Thus, it is recommended that future investigations address direct measurements of glycogen depletion and muscle damage (not only blood-related markers) to elucidate the kinetics of physiological and performance recovery after cycling repeated sprints.

In addition, two points require caution when interpreting the present data: i) despite supported by the sample size calculation, a bigger sample size would strengthen the findings of the present study; ii) even with higher power output during sprints 1 and 2 compared with all other eight sprints in all sessions, as well as the constant recommendation to the participants to avoid any pacing strategy, it is not possible to confirm an total absence of unconsciously pacing. A single maximal effort measurement before performing repeated sprints would have allowed control the pacing effect.

The present study provides new information on time course of performance and neuromuscular function recovery after cycling repeated sprints. This information may have practical value because residual fatigue can affect the subsequent session of training and/or competition. In addition to being directly related to depressed performance, incomplete recovery also increases susceptibility to injury (11).

CONCLUSIONS

It is concluded that cycling repeated sprints induce high levels of neuromuscular fatigue, particularly peripheral fatigue, which seems to be limited to a certain level so as not to surpass a critical threshold. Neuromuscular function is negatively affected by residual fatigue up to 24 h; however, all neuromuscular variables returned to baseline values after 48 h of recovery. These results might be explained by muscle glycogen depletion induced by cycling repeated sprints and muscle damage, but the exact mechanisms should be investigated in future research.

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Author contribution: F. M., A. M. Z., and G. M. developed the study methodology; F. M. and R. A. A. collected the data; F. M. analyzed the results; and F. M., A. M. Z., and G. M. drafted the manuscript. All authors reviewed, revised, and approved the final version of the manuscript for submission.

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