Does Central Fatigue Explain Reduced Cycling after Complete Sleep Deprivation?

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ABSTRACT

TEMESI, J., P. J. ARNAL, K. DAVRANCHE, R. BONNEFOY, P. LEVY, S. VERGES, and G. Y. MILLET. Does Central Fatigue Explain Reduced Cycling after Complete Sleep Deprivation? *Med. Sci. Sports Exerc.*, Vol. 45, No. 12, pp. 2243–2253, 2013. **Purpose**: Sleep deprivation (SD) is characterized by reduced cognitive capabilities and endurance exercise performance and increased perceived exertion (RPE) during exercise. The combined effects of SD and exercise-induced changes in neuromuscular function and cognition are unknown. This study aimed to determine whether central fatigue is greater with SD, and if so, whether this corresponds to diminished cognitive and physical responses. **Methods**: Twelve active males performed two 2-d conditions (SD and control (CO)). On day 1, subjects performed baseline cognitive and neuromuscular testing. After one night of SD or normal sleep, subjects repeated day 1 testing and then performed 40-min submaximal cycling and a cycling test to task failure. Neuromuscular and cognitive functions were evaluated during the cycling protocol and at task failure. **Results**: After SD, exercise time to task failure was shorter (1137 ± 253 vs 1236 ± 282 s, $P = 0.013$) and RPE during 40 min submaximal cycling was greater ($P = 0.009$) than that in CO. Maximal peripheral voluntary activation decreased by 7% ($P = 0.003$) and cortical voluntary activation tended to decrease by 5% ($P = 0.059$) with exercise. No other differences in neuromuscular function or cognitive control were observed between conditions. After SD, mean reaction time was 8% longer ($P = 0.011$) and cognitive response omission rate before cycling was higher ($P < 0.05$) than that in CO. Acute submaximal exercise counteracted cognitive performance deterioration in SD. **Conclusions**: One night of complete SD resulted in decreased time to task failure and cognitive performance and higher RPE compared with the control condition. The lack of difference in neuromuscular function between CO and SD indicates that decreased SD exercise performance was probably not caused by increased muscular or central fatigue.
Key Words: TRANSCRANIAL MAGNETIC STIMULATION, ENDURANCE, NEUROMUSCULAR FATIGUE, COGNITION

Sleep deprivation (SD) is usually a condition of inadequate sleep duration. This may be complete SD such as in ultraendurance sporting events and military exercises or partial SD as with persons experiencing sleep disorders, shift workers, and individuals flying across time zones. In both complete and partial SD, affected individuals self-report feelings of tiredness, clumsiness, and fatigue.

Numerous studies have also demonstrated performance deficits during prolonged exercise under conditions of SD. Intense walking to task failure was significantly shorter after 36–50 h of SD (23,25) and distance run over 30 min after 30-min submaximal running was decreased by 2.9% after 30 h of SD (33). Results from studies examining the effect of SD on performance in shorter running or cycling exercise bouts, however, are contradictory (1,4,18), suggesting that SD-induced performance decrements may be more likely to occur in longer exercise bouts. Maximal strength loss was not observed during either isometric or isokinetic contractions of upper or lower limbs during 60 h of SD (41,42). Attempts to explain decreased exercise performance measures have failed because of the abundance of conflicting results. Oxygen consumption and HR during constant load efforts of varying intensity up to 1 h (23,25,33) were unaffected by SD, although this may not be true in longer duration exercise because

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decreased oxygen consumption was observed after 3 h, but not 1 or 2 h, of light treadmill walking after 36 h SD (24). Conversely, Scott and McNaughton (37) observed lower HR during 30 h of SD with 20 min of light exercise every 4 h, but not when exercise frequency was doubled. Results from incremental tests to task failure are equivocal about the effects of SD of at least 24 h on HR responses and maximal oxygen uptake ($\dot{V}O_{2max}$) (4,18,26,34).

RPE, a subjective measure of exertion, has been shown to be increased with SD in prolonged exercise at a given speed or intensity. This occurred in protocols involving light to intense walking and SD of at least 30 h (23,30,34). Oliver et al. (33) showed no difference in RPE during a 30-min time trial despite a reduction in distance run with SD. This suggests that at identical running speeds, SD RPE would have been greater.

Total SD and partial SD are associated with a general slowing of response speed as well as decreased alertness and attentional capacities. Disagreement remains over the effect of SD on higher level cognitive functions such as learning, memory, and executive functioning (2,16,21). The few studies investigating exercise-induced cognitive changes with SD have found exercise to have short-term alerting effects (20) and decrease reaction time (RT) to a stimulus (38). The positive effects of exercise on RT are well established in non-SD conditions (for review, see Ref. [28]), especially when evaluated after at least 20 min of exercise (3). This has been suggested to result from greater nervous system activation (28) or peripheral motor processes efficiency (8,9) during exercise than at rest.

Although central changes (e.g., augmented RPE during exercise and decrements in cognitive performance) have been observed after extended periods of SD and decreased central activation detected after endurance exercise (29), no study has examined the potential implications of increased central fatigue, i.e., decreased maximal voluntary activation, in performance decrements with SD. To our knowledge, the effects of complete SD on neuromuscular parameters have been limited to transcranial magnetic stimulation (TMS) measures in the upper limbs without exercise. In healthy subjects, De Gennaro et al. (10) observed increased resting motor threshold after 40 h of SD. This was not observed in other studies after 24 h of SD (6,19,36), possibly because of circadian effects because the 40-h period ended at midnight. The single study reporting motor-evoked potential (MEP) amplitude during muscular contractions did not observe a change with SD of at least 24 h (36). This study also reported decreased cortical silent period (CSP) (36), whereas others observed no change after 24 h of SD (6,19). Intracortical inhibition tended to decrease (6,36), whereas changes in intracortical facilitation in these studies were equivocal (6,10). Difficulty in interpreting these studies is compounded by the lack of both a control condition and pre- and post-SD testing to account for normal interday variability and that all studies included both men and women.

The present study aimed to quantify the effects of SD on central fatigue, neuromuscular responses, cognitive control, and RPE in response to whole-body exercise and to determine whether SD results in decreased endurance cycling performance. Secondary objectives were to link the cognitive, physical, and neuromuscular responses to SD together, including the assessment of whether response inhibition, a crucial aspect of human cognitive control (i.e., cognitive processes that ensure adaptive goal-directed behavior), is affected by SD. It was hypothesized that one night of SD would result in decreased neuromuscular functioning evaluated during isometric contractions after exercise and in changes in RPE, HR, and performance during cycling. Furthermore, it was anticipated that submaximal exercise would negate deterioration of information processing efficiency under SD.

METHODS

Subjects

Twelve healthy active men (age, 28 ± 9 yr; height, 1.80 ± 0.06 m; body mass, 71 ± 8 kg; maximal aerobic power output, 324 ± 31 W; $\dot{V}O_{2max}$, 60 ± 7 $mL \cdot kg^{-1} \cdot min^{-1}$ (mean ± SD)) participated in a study with randomized counterbalanced crossover design. Subjects were nonsmokers, nonepileptic, and free of cardiovascular disease and contraindications to TMS. They had 11 ± 9 yr (range, 5–35 yr) of endurance sport experience and trained 5 ± 3 sessions (range, 3–12) per week. Inclusion criteria included verification of normal sleep patterns using the French versions of the Pittsburgh Sleep Quality Index (exclusion if score was ≥5), Horne–Ostberg Morningness–Eveningness questionnaire (exclusion if score was <30 or >70), and Epworth Sleepiness Scale (exclusion if score was ≥10). Written informed consent was obtained from all subjects before their participation, and this study conformed to the standards from the latest revision of the Declaration of Helsinki. All procedures were approved by the Comité de Protection des Personnes Sud-Est 1, France. Subjects were instructed to maintain normal sleep/wake patterns the week before each condition. They were also instructed to avoid strenuous exercise for the 2 d preceding each trial and to abstain from alcohol and caffeine from a minimum of 24 h before the start of each trial until its completion. Sleep and physical activity were recorded by subjects for the 3 d before each condition and verified upon arrival at the laboratory.

Experimental Design

The subjects were required to visit the laboratory for three sessions totaling 5 d. The preliminary visit was performed 1 to 2 wk before the first experimental session and consisted of a medical inclusion, maximal incremental cycling test to task failure, and familiarization with all testing procedures. The experimental conditions were performed between 2 and 4 wk apart. These were an SD condition and a control (CO) condition. Because of the nature of complete SD, neither subjects nor investigators could be blinded. Subjects were not informed of experimental hypotheses. Each condition comprised 2 d, with the first day providing baseline cognitive and

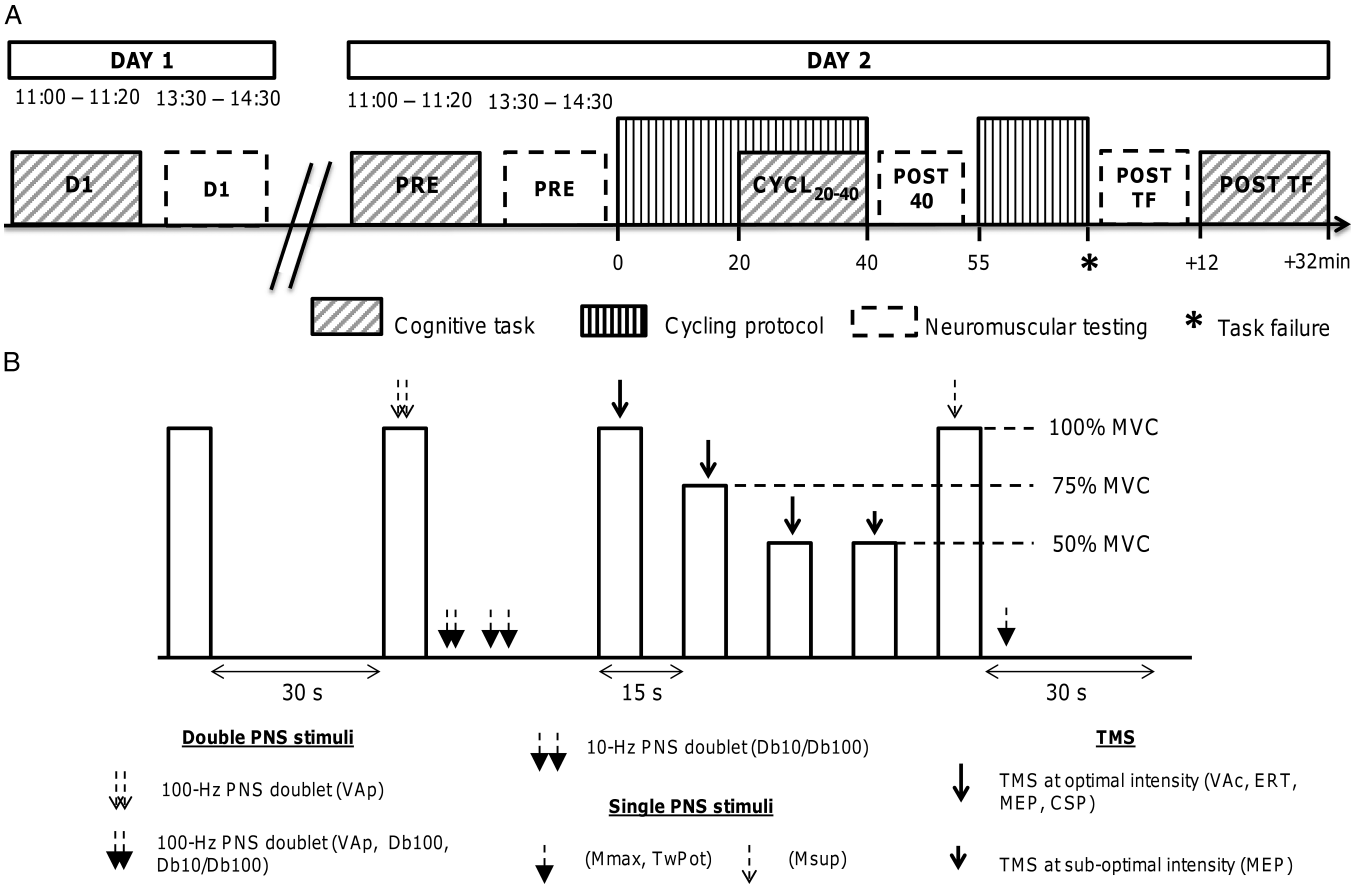

FIGURE 1—Experimental condition test order with time is indicated in minutes from the start of the exercise protocol to task failure (A) and neuromuscular testing protocol (B). The neuromuscular testing protocol began 2 min 30 s after exercise cessation at POST40 and POST TF.

neuromuscular measures from which day-to-day effects of SD and CO conditions were evaluated. On the second day, a submaximal cycling bout was followed by an incremental cycling test to task failure. Cognitive and neuromuscular measures were evaluated before, during, and after the exercise performance test (Fig. 1).

Preliminary Visit

Subjects performed a maximal cycling test to task failure on a cycle ergometer (Monark 839E; Monark Exercise AB, Vansbro, Sweden). The test commenced with 3 min of warm-up at 90 W. Power output was then increased by 15 $W \cdot min^{-1}$ until task failure. Respiratory measures were assessed breath-by-breath by an online system (Ergocard; Medisoft, Sorinnes, Belgium) and averaged every 30 s. $\dot{V}O_{2max}$ was considered as the highest 30-s mean value before task failure and maximal aerobic power output as the power output at the last completed stage. The familiarization portion of the preliminary visit included maximal and submaximal contractions of the knee extensors with and without electrical femoral nerve and transcranial magnetic stimuli (see *Neuromuscular testing* section). This included training subjects to return to the prestimulus force level as soon as possible after TMS to permit consistent measurement of the CSP. Subjects repeated trials until they were able to perform all tests consistently and as directed. Subjects also completed a session of the Simon task (see *Cognitive task* section) consisting of four blocks of 96 trials at 5-min intervals. Each block lasted approximately 3 min 40 s.

Experimental Conditions

Sleep, activity, and condition control. Subjects were instructed to maintain their normal sleep/wake and activity patterns before and during the protocol (except during the night of SD). They recorded their sleep/wake patterns and physical activity (duration and intensity) for 3 d before both experimental conditions. An Actiheart (version 2.2; CamNTech Ltd., Cambridge, UK) was used to measure HR, sleep time, and physical activity, the latter by internal accelerometer that sensed the intensity and frequency of torso movements, from 800 on the first morning of the experimental condition to the end of the protocol. During the night between days 1 and 2, subjects were permitted to return home to sleep in CO. In SD, subjects remained at the laboratory under the supervision of the investigators where they were only permitted to perform sedentary activities such as watching films and listening to music between 2300 and 0700 to limit differences in physical activity and mental stress between conditions. Only the

consumption of water *ad libitum* was permitted after lunch on day 2 (1200). Subjects rated their perception of sleepiness on the Stanford Sleepiness Scale before each cognitive test and before and after the 40-min submaximal exercise.

Force and electromyography. Knee extensor force was measured during voluntary and evoked contractions with a calibrated force transducer (Meiri F2732 200 daN; Celians, Montauban, France) with an amplifier attached by a noncompliant strap to the right leg immediately proximal to the malleoli of the ankle joint. Subjects were seated upright in a custom-built chair with both hips and right knee at 90° of flexion. The load cell was fixed to the chair and in a position that force was measured in direct line to the applied force. Electromyographic signals of the right knee extensors (vastus lateralis (VL), rectus femoris (RF), and vastus medialis (VM)) and flexors (biceps femoris) were recorded.

Electromyographic signals were recorded with pairs of self-adhesive electrodes (Meditrace 100; Covidien, Mansfield, MA) in bipolar configuration with 30-mm interelectrode distance and the reference on the patella. Low impedance (<5 kΩ) between electrodes was obtained by shaving, gently abrading the skin with sandpaper, and then cleaning it with isopropyl alcohol. Electromyographic data were analog-to-digitally converted at a sampling rate of 2000 Hz by a PowerLab system (16/30—ML880/P; ADInstruments, Bella Vista, Australia) and octal bio-amplifier (ML138, ADInstruments) with band-pass filter (5–500 Hz) and analyzed offline using Labchart 7 software (ADInstruments).

Femoral nerve stimulation. Single electrical stimuli of 1-ms duration were delivered via constant current stimulator (DS7A; Digitimer, Welwyn Garden City, Hertfordshire, UK) to the right femoral nerve (peripheral nerve stimulation (PNS)) via a 30-mm-diameter surface cathode in the femoral triangle (Meditrace 100, Covidien) and 50 × 90-mm rectangular anode (Durastick Plus; DJO Global, Vista, CA) on the gluteus maximus. Single stimuli were delivered in the relaxed muscle incrementally until plateaus in maximal M-wave (Mmax) and peak-evoked force were reached. Stimulus intensity throughout the protocol was maintained at 130% of the intensity to produce maximal Mmax and twitch responses to ensure supramaximality. Stimulus intensity was determined each day (51 ± 9 and 52 ± 9 mA for CO and 49 ± 10 and 48 ± 11 mA for SD for days 1 and 2, respectively).

TMS. Single-pulse TMS was used to evoke MEP in the right quadriceps muscles. The motor cortex was stimulated by a magnetic stimulator (Magstim 200^2; The Magstim Company Ltd., Whitland, UK) with a 110-mm double-cone coil (maximum output of 1.4 T). Single stimuli were applied to the contralateral motor cortex producing an induced postero-anterior current. Subjects wore a cervical collar during all TMS measures to stabilize the head and neck. Every centimeter from 1 cm anterior to 3 cm posterior of the vertex was demarcated along the nasal–inion line and to 2 cm over the left cortex. Optimal coil position was determined by assessing MEP responses evoked during brief isometric knee extension at 10% maximal voluntary contraction force (MVC) and 50% maximal stimulator output. The optimal coil position corresponded to the site producing the largest MEP amplitudes in VL, RF, and VM with minimal biceps femoris MEP amplitude. Optimal coil position was marked on a cloth cap secured to the scalp, and it was determined each day since the wearing of an immovable head covering over the course of 2 d was impractical. Stimulus intensity was determined by stimulus–response curve from responses during brief isometric knee extension at 20% MVC. Four consecutive contractions were performed at 15-s intervals at each of the following randomly ordered stimulus intensities: 20%, 30%, 40%, 50%, 60%, 70%, and 80% maximal stimulator output. Optimal stimulus intensity was defined as the minimum stimulus intensity evoking maximal MEP amplitude in all measured quadriceps muscles. A suboptimal stimulus intensity was also determined from the stimulus–response curve at 20% MVC. This intensity corresponded to a stimulus intensity evoking MEP amplitudes approximately half their maximum for VL, RF, and VM.

Neuromuscular testing. Neuromuscular measures (force and electromyography) were assessed at four time points during each condition (day 1 (D1), day 2 precycling (PRE), post–40-min submaximal cycling (POST40), and postcycling task failure (POST TF)) (Fig. 1A). After determining the optimal site and intensity for TMS and PNS each day, maximal force was determined from four MVC separated by 30 s. In the latter two MVC, PNS (100-Hz doublet) was delivered at peak force and immediately after in the relaxed state (100- and 10-Hz doublets). Three series of five contractions were performed with real-time visual feedback, consisting of four during which TMS was delivered (100%, 75%, and 50% MVC at optimal stimulus intensity (45) and 50% MVC at suboptimal stimulus intensity) and another MVC with PNS (single stimulus delivered at peak force and again in the relaxed muscle in the potentiated state). Contractions began at 15-s intervals and sets were separated by 30 s. Subjects were instructed to maintain or return to the prestimulus force level after TMS. At POST40 and POST TF, measures began exactly 2 min 30 s after the cessation of cycling. Only two MVC, the latter with PNS doublets, and two series of five contractions (100%, 75%, and 50% MVC at optimal stimulus intensity, 50% MVC at suboptimal stimulus intensity and MVC with single PNS stimuli) were performed because of the time-sensitive nature of the measurement with fatigue (Fig. 1B).

Cognitive task. Subjects were required to complete four blocks of the Simon task (i.e., a classic paradigm used to assess the ability to focus attention while ignoring irrelevant information; for a review, see ref. 40) at four time points during each condition (day 1 (D1), day 2 precycling (PRE), from 20 to 40 min of the 40-min submaximal cycling bout ($CYCL_{20–40}$), and POST TF) (Fig. 1A). Each block consisted of 96 trials, and blocks were performed at precisely 5-min intervals, giving subjects between 60 and 90 s of "cognitive rest." The cognitive task was performed while seated on the cycle ergometer facing a computer screen at a distance of

1.0 m. A response button was fixed to each of the handlebars (right and left) of the ergometer. A fixation point (white circle) was in the center of the screen and remained present throughout the trials. Subjects were instructed to respond as quickly and accurately as possible by pressing the appropriate response button according to the color of circle presented either to the left or to the right of the fixation point at a visual angle of 8.6°. Subjects were instructed to respond according to the color of the stimulus while ignoring its spatial location. The mapping of stimulus color to response button was counterbalanced across subjects. The task was composed of two equally probable trial types: congruent trials where the spatial location of the stimulus corresponded to the task-relevant aspect of the stimulus (e.g., left stimulus/left response) and incongruent trials where the spatial location of the stimulus corresponded to the opposite spatial location of the response (e.g., left stimulus/right response). As soon as a response button was pressed, or after 1500 ms in the absence of a response, the stimulus was removed and the next trial presented.

Exercise protocol. On day 2, subjects performed a two-part cycling test at self-selected pedal frequency. The first part consisted of 40 min of submaximal exercise as a 5-min warm-up at 50% MAP and 35 min at 65% MAP (i.e., 210 ± 20 W). RPE was assessed by 100-mm visual analog scale (31) every 5 min from 10 min and HR was recorded throughout. Beginning at 20 min of part 1, subjects performed the cognitive task while cycling. The second part, i.e., the timed exercise to task failure (TTF), commenced with 5 min at 65% MAP, increasing stepwise by 5% MAP every 5 min until task failure. RPE was assessed every 5 min and at task failure and HR was recorded throughout. Subjects were required to remain seated throughout the cycling test, and an investigator blinded to the exercise time provided standardized encouragement in both conditions.

Data Analysis

Activity. Mean activity in arbitrary units per minute was determined from 0800 on day 1 to 1430 on day 2. Subanalyses on the normal sleep period from (2300 to 0800) and the nonsleep period (day 1 from 0800 to 2300 and day 2 from 0800 to 1430) were also conducted.

PNS. Voluntary activation was assessed peripherally (VAp) by twitch interpolation using the superimposed and potentiated twitch amplitudes elicited by PNS 100-Hz doublets during and after MVC and calculated from the following equation: [1 − (PNS 100-Hz superimposed twitch/Db100)] × 100. The evolution of low- and high-frequency fatigue was evaluated from the change in the ratio of low-frequency (Db10, 10 Hz) doublet to high-frequency (Db100, 100 Hz) doublet (48).

TMS. Peak-to-peak amplitude of MEP and M-waves were measured, and MEP amplitude was normalized to maximal M-wave amplitude during MVC (Msup) and Mmax measured at the same time point. In one subject, MEP normalization by Msup was not performed because of difficulties in eliciting Msup. All analyses involving Msup or values normalized with Msup were thus performed on 11 subjects. Cortical voluntary activation (VAc) during maximal effort was measured by modified twitch interpolation. Corticospinal excitability increases substantially during the transition from relaxed to contracted muscle states (47), thus underestimating TMS in the relaxed muscle. Instead, the potentiated twitch amplitude elicited by TMS in relaxed muscle was estimated. At each time point, a linear regression was performed on the relation between superimposed twitch evoked when TMS was delivered at 100%, 75%, and 50% MVC and voluntary force (45). This relation was extrapolated, and the *y*-intercept was interpreted as the estimated resting twitch amplitude. VAc was assessed with the following equation: [1 − (TMS superimposed twitch/estimated resting twitch)] × 100. The reliability of this method has recently been validated in the knee extensors (13). The duration of the CSP was determined visually and defined as the duration from the cortical stimulus to the return of continuous voluntary electromyography (39).

Cognitive task. RT <100 ms were considered anticipated responses and were thus excluded from further analyses. The rates of errors and omissions (RT >1500 ms) were both calculated as a percentage of the total number of trials. Mean RT for correct trials was calculated for each condition (SD and CO), time (D1, PRE, $CYCL_{20–40}$, and POST TF), block (1, 2, 3, and 4), and congruency (congruent and incongruent).

Statistics

Exercise and neuromuscular responses. All data were assessed for normality before statistical analysis was performed. Two-way repeated-measures ANOVA (condition × time) was used to evaluate the differences between D1 and PRE in CO and SD. Then, two-way repeated-measures ANOVA (condition × time) was used to assess the changes on day 2 for all neuromuscular measures. Two-way repeated-measures ANOVA (condition × time) was conducted on RPE and HR in parts 1 and 2 of the cycling protocol. Comparison of CSP between days was not conducted because optimal stimulus intensity was determined each day and changes to stimulus intensity influence CSP duration independent of other factors. When ANOVA revealed significant interactions, the Newman–Keuls *post hoc* test was used to identify differences. VAc was assessed by two-way nonparametric repeated-measures ANOVA because these data were not normally distributed. Student's paired *t*-tests were used to evaluate differences in TTF performance, activity, and sleep patterns. Data are presented as mean ± SD.

Cognitive task. The arcsine transformations of mean RT and error rate were both evaluated by ANOVA with condition (SD and CO), time point (D1, PRE, $CYCL_{20–40}$, and POST TF), block (1, 2, 3, and 4), and congruency as within-subject factors. To correct for violation of sphericity assumptions, a Greenhouse–Geisser degree of freedom correction was applied. *Post hoc* Newman–Keuls analyses were conducted on all significant interactions. Arcsine transformations of

omission rate were assessed by nonparametric Wilcoxon signed-rank test. Data are presented as mean ± SE of the mean.

Statistical significance was set at $P < 0.05$ for all statistical analyses.

RESULTS

Sleep Patterns and Sleepiness

Normal sleep patterns were characterized by scores of 3 ± 1 on the Pittsburgh Sleep Quality Index, 56 ± 8 on the Horne–Ostberg Morningness–Eveningness questionnaire, and 6 ± 2 on the Epworth Sleepiness Scale. There were no differences between conditions in the time subjects slept (CO, 2335, vs SD, 2335; $P = 1.00$) or woke up (CO, 8:04 a.m., vs SD, 8:02 a.m.; $P = 0.88$) or in the number of hours they slept (CO, 8 h 29 min ± 53 min, vs SD, 8 h 27 min ± 48 min; $P = 0.88$) the three nights before the experimental protocols. Subjects were more active in SD than CO (CO, 71 ± 15 arbitrary units per minute, vs SD, 89 ± 25 arbitrary units per minute; $P = 0.028$). This was exclusively due to a difference in activity during the normal sleep period (CO, 19 ± 27 arbitrary units per minute, vs SD, 45 ± 15 arbitrary units per minute; $P = 0.002$).

There was no difference between conditions on day 1 on the Stanford Sleepiness Scale ($P = 1.00$). Sleepiness increased from day 1 to 2 in SD only (CO, 1.7 ± 0.5 and 1.8 ± 0.6, vs SD, 1.7 ± 0.7 and 4.0 ± 1.2, for days 1 and 2, respectively; $P < 0.001$). Subjective sleepiness was greater at all time points on day 2 in SD than CO ($P < 0.001$).

Performance, RPE, and HR during Exercise

Cycling time to task failure was significantly shorter in SD than CO (Fig. 2A). RPE was significantly greater in SD than CO and increased ($P < 0.001$) during 40 min of submaximal exercise. There was no difference in RPE during TTF between conditions ($P = 0.15$) as RPE increased to task failure ($P < 0.001$) (Fig. 2B). There was also no difference in HR between SD and CO during 40-min submaximal cycling (mean HR: CO, 159 ± 14 beats per minute, vs SD, 157 ± 15 beats per minute; $P = 0.12$). During TTF, HR was higher in CO than SD at all time points (HR at task failure: CO, 180 ± 12 beats per minute, vs SD, 173 ± 14 beats per minute; $P < 0.001$).

Neuromuscular Responses

Maximal voluntary and evoked forces. There were no differences in MVC between conditions or days ($P > 0.05$). MVC decreased with exercise from PRE to POST40 ($P = 0.011$) and then no further to POST TF ($P = 0.09$). Similarly, Db100, Db10/Db100, and potentiated twitch and estimated resting twitch amplitudes were similar between D1 and PRE and between conditions ($P > 0.05$), and all decreased with exercise (Table 1).

M-waves. Decreased VL and RF Mmax and RF Msup were observed from D1 to PRE ($P < 0.01$). No differences in VM Mmax or VL or VM Msup were observed between days ($P > 0.05$). Both Mmax and Msup decreased with exercise in both conditions and all muscles ($P < 0.01$) (Table 1).

TMS stimulus intensity. There was no difference between conditions ($P = 0.71$) or days ($P = 0.68$) for optimal stimulus intensity. Mean optimal stimulus intensity was 65% ± 8% and 62% ± 9% for CO and 62% ± 9% and 63% ± 12% for SD for days 1 and 2, respectively. There was also no difference between conditions ($P = 0.46$) or days ($P = 0.59$) for submaximal stimulus intensity. Mean submaximal stimulus intensity was 35% ± 7% and 35% ± 8% for CO and 36% ± 8% and 36% ± 8% for SD for days 1 and 2, respectively.

Voluntary activation. There were no differences between conditions for either VAc ($P = 0.34$) or VAp ($P = 0.31$). There was a trend for VAc to decrease with exercise; however, this did not achieve statistical significance ($P = 0.059$) (Fig. 3A). Peripheral voluntary activation decreased with exercise ($P = 0.003$) and was lower at POST TF than both PRE ($P = 0.003$) and POST40 ($P = 0.014$) (Fig. 3B).

MEP (at optimal stimulus intensity). No differences in MEP/Mmax or MEP/Msup were observed between days or conditions for any muscle or contraction intensity ($P > 0.05$).

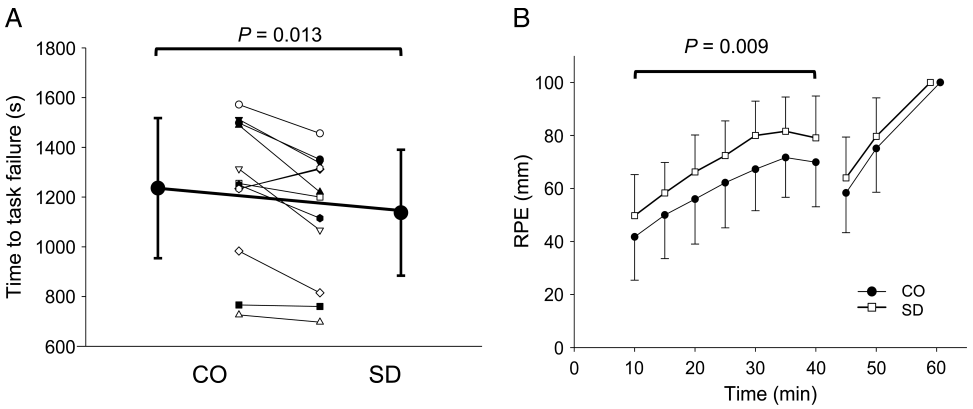

FIGURE 2—Effect of SD and CO conditions on mean and individual cycling time to task failure (A) and RPE during the cycling protocol (B). There was higher RPE in SD than CO ($P = 0.009$) during the first 40 min. Values are presented as mean ± SD.

TABLE 1. Neuromuscular parameter evolution with time in SD and CO conditions at D1, PRE, POST40, and POST TF (n = 12 unless otherwise indicated).

		D1	PRE	POST40	POST TF
MVC (N)	CO	599 ± 121	610 ± 100	544 ± 97***	515 ± 85***
	SD	589 ± 95	577 ± 94	510 ± 92***	494 ± 71***
Potentiated twitch (N)	CO	159 ± 34	160 ± 35	123 ± 30***	115 ± 26***,****
	SD	158 ± 30	160 ± 30	125 ± 26***	117 ± 27***,****
Db100 (N)	CO	268 ± 50	271 ± 45	218 ± 47***	206 ± 48***
	SD	268 ± 44	266 ± 46	222 ± 51***	211 ± 49***
Db10/Db100	CO	1.01 ± 0.08	1.01 ± 0.06	0.75 ± 0.12***	0.74 ± 0.11***
	SD	1.04 ± 0.06	1.01 ± 0.08	0.75 ± 0.09***	0.73 ± 0.09***
Estimated resting twitch (N)	CO	101 ± 45	102 ± 45	69 ± 42***	52 ± 27***,****
	SD	103 ± 35	98 ± 34	78 ± 41***	59 ± 33***,****
Mmax (mV)					
VL	CO	17.1 ± 3.3**	15.7 ± 3.5	14.4 ± 4.3*	12.5 ± 4.6***,******
	SD	16.4 ± 3.1**	15.5 ± 2.5	14.6 ± 2.7*	12.4 ± 4.5***,******
RF	CO	7.7 ± 2.5***	6.9 ± 2.3	6.4 ± 2.1*	4.9 ± 1.8***,******
	SD	8.5 ± 2.6***	8.0 ± 2.5	7.1 ± 2.5*	5.8 ± 2.8***,******
VM	CO	13.5 ± 4.4	13.1 ± 4.4	12.5 ± 5.3	10.1 ± 4.5**,*****
	SD	12.1 ± 4.2	11.5 ± 3.8	9.9 ± 3.0	7.7 ± 3.9**,*****
Msup (mV) (n = 11)					
VL	CO	15.2 ± 3.9	14.4 ± 3.9	12.9 ± 4.3*	11.1 ± 4.1***,*****
	SD	14.3 ± 3.6	14.2 ± 4.0	13.3 ± 3.1*	12.0 ± 5.4***,*****
RF	CO	8.2 ± 3.2**	7.2 ± 2.7	6.7 ± 2.5	5.5 ± 2.2***,*****
	SD	9.0 ± 3.3**	8.6 ± 3.0	7.6 ± 2.5	6.2 ± 3.0***,*****
VM	CO	10.3 ± 3.8	9.6 ± 3.8	9.3 ± 3.7	8.0 ± 4.7*,*****
	SD	10.3 ± 2.9	10.1 ± 2.3	9.5 ± 2.1	7.6 ± 3.2*,*****

There were no differences between CO and SD ($P > 0.05$).
*Time point significantly different from PRE ($P < 0.05$).
**Time point significantly different from PRE ($P < 0.01$).
***Time point significantly different from PRE ($P < 0.001$).
****Time point significantly different from POST40 ($P < 0.05$).
*****Time point significantly different from POST40 ($P < 0.01$).
******Time point significantly different from POST40 ($P < 0.001$).

Increased VL MEP/Mmax and MEP/Msup with exercise at all contraction intensities were observed ($P < 0.05$). VM MEP/Mmax at 100% and 75% MVC and MEP/Msup at 100% MVC increased with exercise ($P < 0.05$). The increase in VM MEP/Mmax at 50% MVC approached statistical significance ($P = 0.050$). There were no changes in RF MEP/Mmax or MEP/Msup ($P > 0.05$) with exercise (see Figure, Supplemental Digital Content 1, http://links.lww.com/MSS/A310, which illustrates the effect of SD and CO conditions and exercise on MEP).

MEP (at suboptimal stimulus intensity). Both VL MEP/Mmax ($P = 0.011$) and MEP/Msup ($P = 0.026$) increased with exercise. There were no changes in RF or VM MEP/Mmax or MEP/Msup ($P > 0.05$) with exercise and no differences between conditions or days for any muscle ($P > 0.05$) (see Figure, Supplemental Digital Content 1, http://links.lww.com/MSS/A310, which illustrates the effect of SD and CO conditions and exercise on MEP).

CSP. Analysis of CSP was performed on 11 subjects because one subject did not return to precontraction force levels after the delivery of TMS, thus making CSP determination impossible. There were no differences in CSP between conditions for any muscle or contraction intensity ($P > 0.05$). CSP was shorter at both POST40 and POST TF

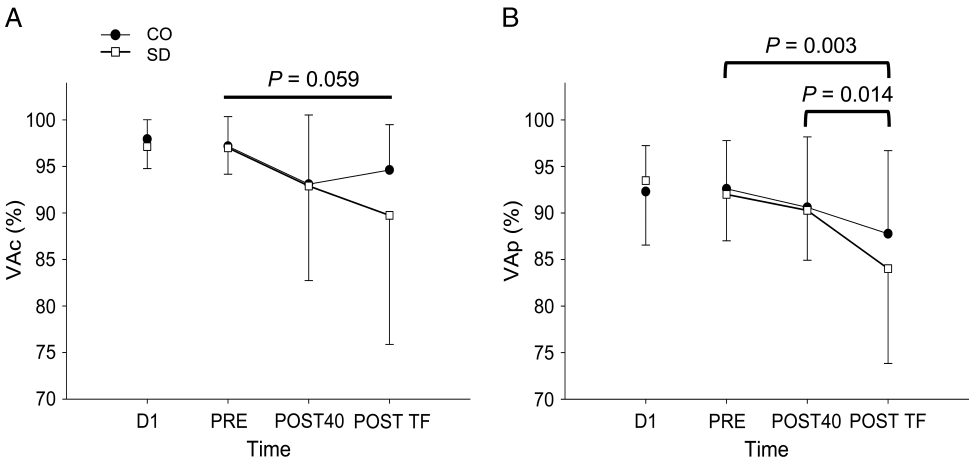

FIGURE 3—Effect of SD and CO conditions and exercise on VAc (A) and VAp (B). Values are presented as mean ± SD.

than at PRE for all muscles and contraction intensities ($P < 0.01$) (see Figure, Supplemental Digital Content 2, http://links.lww.com/MSS/A311, which illustrates the effect of SD and CO conditions and exercise on CSP).

Cognitive Task

RT. Results showed the main effects of condition ($P = 0.011$), trial congruency ($P = 0.019$), time ($P = 0.023$), block ($P = 0.024$), and an interaction between condition and time ($P = 0.035$). RT was longer for incongruent trials (406 ± 11 ms) than congruent trials (377 ± 10 ms). The interaction between condition and time indicated that RT lengthened in SD in PRE (375 ± 9 ms, $P = 0.007$) and POST TF (371 ± 16 ms, $P = 0.002$) compared with CO (349 ± 8 and 337 ± 10 ms for PRE and POST TF, respectively). Conversely, during $CYCL_{20–40}$, RT in SD (347 ± 11 ms) did not differ from RT observed in CO (CO, 333 ± 9 vs 347 ± 11 ms; $P = 0.20$) (Fig. 4A). No other interactions were observed.

Decision errors and omissions. A classic congruency effect was observed with the prevalence of errors in incongruent trials (6.19% ± 0.7%) greater than that in congruent trials (3.04% ± 0.4%, $P < 0.001$). There were no other main effects or interactions. Wilcoxon signed-rank test showed that the omission rate was greater in SD during PRE (0.82%, $P = 0.012$) and POST TF (1.68%, $P = 0.002$) than that in CO (0.02% and 0% for PRE and POST TF, respectively). Conversely, no omissions were observed in either SD or CO during $CYCL_{20–40}$ (Fig. 4B).

DISCUSSION

The principal findings of this study are that one night of SD resulted in decreased cycling time to task failure, increased RPE during cycling, and both longer RT and higher omitted response rates at rest without evidence of decreased cognitive control efficiency compared with CO. Despite increased RPE in SD, submaximal cycling exercise restored information processing efficiency to baseline levels. Furthermore, changes within the muscle or to voluntary activation measured after task failure cannot explain the decrement in exercise performance with SD. The hypothesis that increased central fatigue might elucidate performance deterioration was refuted because neuromuscular function was not affected by SD.

Cycling performance. The diminished cycling performance in SD may be explained by differences in RPE and sleepiness. Motivation and the decision to stop exercise involve complex cognitive functions. Sleepiness, as assessed by the Stanford Sleepiness Scale, was greater in SD than CO and during exercise when sleepiness increased in CO and was unchanged by SD. These, coupled with prior research indicating that combined intermittent exercise and SD cause individuals to be more susceptible to negative mood states than SD alone (38), suggest that increased sleepiness during exercise and mood disturbances may have contributed to reduced exercise performance in SD.

RPE and HR. During the 40 min of submaximal cycling, RPE was significantly greater with SD. Despite this difference, there was no difference in RPE during TTF between conditions. All subjects, however, had maximal RPE at task failure, although this occurred 59 s later in CO (mean performance time decrement of 7.5% in SD). This result concurs with the findings of Marcora et al. (22), who compared TTF after both a 90-min mentally fatiguing task and a 90-min mentally neutral task. In this study, RPE was higher in the mentally fatiguing condition except at task failure, which occurred earlier after the mentally fatiguing task. Sleep loss has previously been shown to have dramatic effects on emotional processing, judgment, and self-esteem, and subjects were more likely to report increased feelings of worthlessness, inadequacy, powerlessness, and failure (16). Emotional modifications may explain the difference in a self-reported measure like RPE and require further investigation. SD also reduced exercise HR only during TTF in the present study. The finding that SD results in decreased exercise HR is equivocal (23,25,26,33,37), suggesting that exercise duration and/or intensity may be important factors influencing

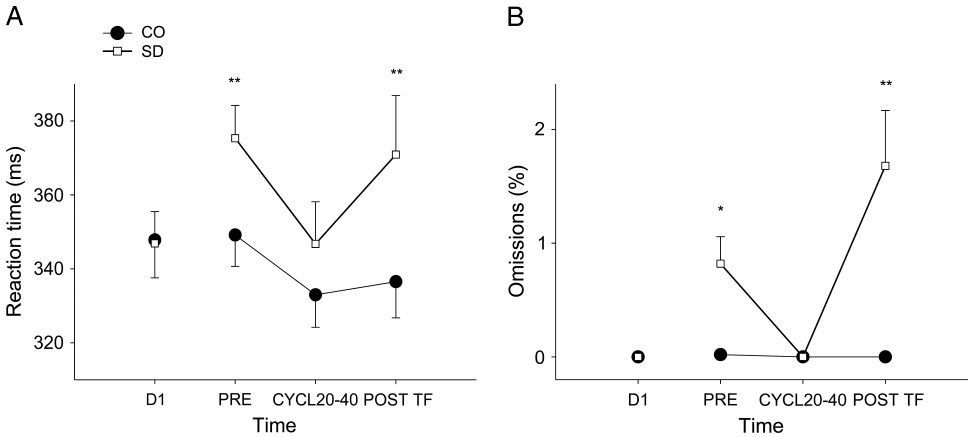

FIGURE 4—Effect of SD and CO conditions and exercise on RT (A) and omission rate (B). Values are presented as mean ± SE of the mean. Results in SD are significantly different from CO. *$P < 0.05$ **$P < 0.01$.

the effect of SD on HR. Scott and McNaughton (37) discussed several proposed mechanisms to explain lower HR during exercise in SD, including plasma volume expansion and decreased respiratory controller sensitivity, and their potential problems or the data required to support them. Interestingly, in conjunction with the increased RPE during TTF after a mentally fatiguing task, Marcora et al. (22) observed lower HR only at task failure and attributed this difference to task failure occurring earlier. Further investigation is required to identify the mechanisms and conditions underlying decreased exercise HR with SD.

Neuromuscular function. Our hypothesis that a greater reduction in the neural recruitment of motor units, central fatigue, might partially explain diminished cycling performance with SD was refuted. Maximal voluntary force and electrically evoked M-wave and force decreased with exercise, agreeing with previous studies of aerobic exercise (29). There was evidence of decreased voluntary activation, including VAc showing a strong tendency to decrease with exercise ($P = 0.059$). Isometric MVC has been shown to begin to recover immediately after a fatiguing task (11). Peripheral voluntary activation was evaluated before VAc at each evaluation, and the additional recovery time may have been sufficient to create this discrepancy and render VAc evaluation insufficiently sensitive to real changes in some subjects. However, measures of central fatigue recover more slowly than peripheral responses (unpublished data and Ref. (11)), suggesting that the effect of PNS and TMS testing order was likely minimal. Previous studies evaluating TMS measures in SD generally observed results in SD and CO to be similar (6,10,19,36). Only MEP amplitude during muscular contraction was a common measure with any of these studies. Scalise et al. (36) observed no change in absolute MEP amplitude after at least 24 h of SD in opponens pollicis, mirroring our observation that MEP amplitude is unaffected by SD. VL MEP amplitude and VM MEP amplitude at some contraction intensities increased with exercise, consistent with findings in fatiguing submaximal and maximal isometric contraction protocols (14). Conversely, RF MEP amplitude and VM MEP amplitude at some contraction intensities did not change with exercise, consistent with other cycling protocols (12,17,39), including two of comparable duration. The discrepancy between these studies (17,39) and the present study may be due to their use of lower TMS intensities (30%–60% maximal stimulator output vs mean stimulus intensity >60% maximal stimulator output in all sessions in the present study). These results also suggest that different muscles of the quadriceps may not demonstrate a homogeneous response to exercise, although the rapid recovery of MEP to baseline levels postexercise (44) may mask exercise-induced changes in RF and VM. Changes in MEP amplitude during exercise did not differ between SD and CO, indicating that corticospinal excitability was unaffected by SD, both at rest and after fatiguing exercise.

The amplitude of MEP at 50% MVC was evaluated by TMS delivered at two stimulus intensities, one to evoke maximal MEP amplitudes and the other to evoke half-maximal MEP amplitudes, both determined from the stimulus–response curve at 20% MVC. For all muscles, the same changes were observed at both TMS stimulus intensities. The changes in MEP amplitude observed in this study were independent of TMS stimulus intensity. If submaximal MEP responses are not measured, real changes in cortical excitability may be overlooked if the stimulus–response curve shifts to the left or right and maximal MEP amplitude remains unaffected. This however was not the case in the present study.

The finding that CSP decreased with exercise is novel. This contrasts the increased CSP observed in sustained submaximal and maximal isometric contractions (14) and its lack of change after other cycling protocols (12,17,39). The difference between cycling protocols of similar duration (17,39) may be due, at least in part, to the aforementioned difference in TMS intensities used. After exercise cessation, CSP has been observed to rapidly return to baseline values (43), suggesting that the magnitude of decrease may be underestimated. The primary inhibitory cerebral neurotransmitter is GABA, which is derived from glutamate. CSP is predominantly mediated by $GABA_B$ receptors (27); thus, decreased $GABA_B$ concentration would reduce cortical inhibition and CSP duration. After 3 h of cycling at 60% $\dot{V}O_{2max}$, cerebral ammonia uptake and its accumulation in cerebral spinal fluid were observed (32). Previously, maximal incremental cycling to task failure (approximately 12 min) showed cerebral ammonia uptake without cerebral spinal fluid accumulation (7). Proposed by Nybo et al. (32) and supported by previous research in rats (15), a minimum duration and exercise intensity is necessary to exceed the ammonia removal capacity of the brain. Accumulation of ammonia in cerebral spinal fluid could cause decreased cortical glutamate concentration since ammonia is condensed with glutamate to produce glutamine during ammonia removal. Consequently, GABA concentration would decrease, resulting in decreased cortical inhibition. Whether this mechanism may explain the observed reduction of intracortical inhibition during prolonged exercise requires further investigation. The lack of difference between CSP shortening in CO and SD indicates that any mechanism contributing to shorter CSP during exercise is unaffected by SD.

Cognitive performance, SD, and exercise. This study reproduced cognitive deficits widely reported after one night of SD, notably slowed response speed, and increased the number of omitted responses (e.g., Ref. [46]). No evidence of decreased response inhibition was observed in SD as demonstrated by the lack of primary interaction between congruency and condition or second-order interaction with the addition of time points (D1, PRE, $CYCL_{20–40}$, and POST TF). Using three short Stroop tasks (Color-Word, Emotional, and Specific), Sagaspe et al. (35) similarly observed that 36 h of SD did not affect cognitive control. Cognitive control was also unaffected by exercise because there was no interaction between congruency and time points. In conjunction with the lack of significant interactions involving mean RT or decision error, these results suggest that neither SD or exercise nor

their interaction influenced cognitive control. The present study is consistent with Killgore (16) and suggests that cognitive processes are differentially sensitive to SD because some cognitive functions were impaired (e.g., slowing of response speed), whereas others were unaffected (e.g., selective response inhibition).

Shorter RT during exercise was not associated with increased decision error, indicating that the response strategy (i.e., speed–accuracy trade-off) did not change and that exercise specifically caused increased performance. In accordance with our hypothesis, this positive effect of acute submaximal exercise also counteracted the negative effects of SD and restored information processing efficiency (i.e., faster RT and fewer omissions) to baseline levels. This benefit could have been due to greater exercise-induced nervous system activation (e.g., increased HR [8,9], increased plasma catecholamines [5]), which could have temporarily negated the decreased alertness and attentional capacities caused by SD. This gain may have endured for a short duration; however, it was no longer observed at POST TF, reinforcing the established transient postexercise benefits of exercise on cognitive performance (3). The exact mechanism(s) for transient improvements in cognitive performance during exercise remains to be elucidated.

Limitations. Without the availability of electroencephalography, the effects of possible microsleeps are unknown despite constant subject supervision. Effects of subjects being exposed to low levels of light and being more active in SD may also have influenced the results. The performance measure of TTF was chosen despite its limited application to real-world exercise performance, greater variability, and important motivational component. The primary goal was to exhaust the subject, and if a time trial was used, the associated pacing strategies may have complicated interpretation of the results. Neuromuscular assessment was not conducted on the same apparatus as cycling; thus, there was a delay from exercise termination to neuromuscular evaluation, meaning that changes in neuromuscular measures immediately postexercise would not have been identified. Measurement of electromyography was not conducted during the exercise bouts, thus preventing neuromuscular evaluation of the effects of SD during exercise. Further studies are required to investigate combined PNS and TMS measures during exercise with SD.

CONCLUSION

In summary, one night of complete SD resulted in decreased cycling time to task failure compared with a control condition. Self-reported measures, including RPE, were altered in SD, confirming the importance of emotional processing in SD-induced performance deficits. Cognitive processes appear to be differentially sensitive to SD because only some cognitive functions were impaired. Furthermore, the compensatory effect of acute submaximal exercise on cognitive deficits induced by sleep loss was demonstrated. Neuromuscular function 3–4 min after cycling cessation was similar between CO and SD, indicating that changes in the muscle and to the motor nervous system likely cannot explain any of the decrement in exercise performance with SD. Thus, the hypothesis that increased central fatigue after one night of complete SD contributes to decreased exercise performance is unsupported.

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