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Effects of Acute Sleep Deprivation on the Sequential Rate of Torque Development throughout the Force-Time Curve

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Abstract

Objective The impact of sleep deprivation on the physiological determinants of explosive torque production remains poorly understood. We aimed at determining the acute effects of 24 hours of sleep deprivation on the sequential rate of torque development (RTD) obtained during plantar flexion through maximum voluntary isometric contraction (MVIC).

Materials and Methods The study included 14 healthy-young adults (8 men and 6 women). The participants visited the laboratory on 2 different occasions: without and with 24 hours of sleep deprivation. In each session, the subjects were tested for RTD of the plantar flexors with concomitant recordings of the electromyographic (EMG) amplitude of the soleus over the following time intervals: 0 to 30, 30 to 50, 50 to 100, and 100 to 150 ms.

Results Sleep deprivation did not affect peak RTD (without sleep deprivation: $283.3 \pm 111.6\ N.m.s^{-1}$ versus with sleep deprivation: $294.9 \pm 99.2\ N.m.s^{-1}$; $p > 0.05$) of plantar flexion. The sequential values of RTD, as well as the normalized amplitude of the soleus EMG, remained similar between both conditions ($p > 0.05$).

Discussion In conclusion, we found that 24 hours of sleep deprivation do not affect muscle activation, nor explosive torque production throughout the torque-time curve. Thus, exercise performance and daily functionality in tasks involving rapid torque development might remain well preserved after 24 hours of acute sleep deprivation.

Keywords
- fatigue
- sleep deprivation
- torque-time curve
- electromyography

Introduction

Disturbed sleep is detrimental for individual health, well-being, and work productivity.[1] Sleep disturbance may be secondary to sleep deprivation, involving a sustained state of wakefulness with no effective sleep (such as for one or more whole nights), or sleep restriction, which is typically associated with a chronically-reduced sleep duration (such as when humans fall asleep later or wake earlier than normal).[1,2] In athletes, sleep can frequently be disturbed in the 24 hours that precede decisive competitions or qualifiers, and this can impair motivation,[3] which is essential for exercise performance. In addition, travelling to a distant place to compete is also quite frequent in sports,[4] which can

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impact sleep quality or quantity due to unfamiliar environments. Thus, examining the impact of 24 hours of sleep deprivation in motor performance is relevant in the context of sports.

Recent research[5] revealed that motor control is affected by 24 hours of sleep deprivation, acting as an additional determinant of antagonist/agonist coactivation during maximum voluntary isometric contractions (MVICs) towards increased joint stiffness. Such pattern of change, particularly when occurring in flexor-extensor muscle pairs, has been widely associated with slower movement.[6] Whether this effect is accompanied by a sleep-related reduction in explosive torque production (that is, rate of torque development – RTD) has not yet been determined.

The RTD represents the rate of rise of the torque-time curve (Δ torque/Δ time), and it is important in circumstances when torque-production times are short (100 ms to 300 ms, for example), such as when reversing a fall or in several athletic events (like jumps, throws, sprints).[7] In specific, peak RTD represents the highest RTD value of the torque-time curve, measured over epochs of 20 ms.[8] There is general agreement that RTD depends both on neural as well as on muscular factors,[9] and it has been suggested that the predominance of these specific mechanisms changes over time during the course of each contraction.[10,11] During the first 50 ms of contraction, the RTD is primarily influenced by the capacity of the central nervous system to induce maximal muscle activation.[11] From then on ($>$ 50 ms), the RTD is more strongly related with changes in intrinsic contractile properties and maximum muscle strength.[10]

Alterations in the RTD in response to experimental interventions are not only caused by changes in muscle morphology and architecture, but also by adaptations in the central nervous system (primarily due to the motor unit discharge rate).[12,13] The measurement of surface electromyographic (EMG) activity, which reflects the activation of a large area of muscle underlying the recording electrodes, has provided evidence for parallel increases in RTD and EMG amplitude after regimented resistance training.[12,13] However, there are several methodological issues associated with EMG measurements during voluntary muscle contractions that are likely to compromise both between- and within-person comparisons (such as electrode position and thickness of the subcutaneous adipose tissue).[14,15] To overcome these limitations, EMG epochs are often normalized using evoked responses, namely the motor (M-) wave.[16] The M-wave represents the compound muscle action potential resulting from the direct and synchronous depolarization of motor axons innervating the muscles.[17] This response can be easily elicited via percutaneous electrical nerve stimulation at the periphery, and it is highly reliable, as demonstrated in past research.[18]

Previous research[19,20] indicates that 60 hours of sleep deprivation do not affect the peak RTD of the knee extensors or forearm flexors. More recently, it was also demonstrated that two nights of sleep deprivation reduced knee-extension MVIC, but not the peak RTD.[21] Thus, there is compelling evidence that the peak RTD remains virtually unaltered in up to 60 hours of sleep deprivation. Despite the relevance of these findings, it is important to note that the peak RTD does not discriminate between the different mechanisms underlying explosive torque production. Thus, based on these concepts, a more detailed analysis of the rising torque–time curve might enable a better characterization of how sleep deprivation affects explosive motor actions. Ultimately, this approach may unravel different interactions between acute sleep deprivation and known predictors of RTD (such as neural versus muscular impact).

Therefore, we sought to investigate the effects of 24 hours of sleep deprivation on the sequential periods of RTD for plantar flexion MVIC and the corresponding normalized EMG amplitude of the soleus muscle. The soleus muscle is particularly well suited for this specific purpose, because it is a monoarticular muscle that enables a complete dissociation between the latency of its M-wave (5 ms to 8 ms) and H-reflex (30 ms to 45 ms),[16] which is methodologically critical in identifying the motor response, assessed by electrical stimulation of the tibial nerve, that is essential for normalizing the sequential periods of EMG recordings. Since sleep deprivation has been purported to exert a negative impact on muscle strength via disturbed neural function,[2] we hypothesized that RTD and EMG amplitude would be diminished in the early phase of MVIC, but not in the last period of the rising torque-time curve.

Materials and Methods

Participants

Ethical Considerations

The experimental protocol was approved by the institutional Ethics Committee (CEFMH no. 13/2019) and is in accordance with the Declaration of Helsinki. Prior to study entry, the risks and procedures were explained in detail, and written informed consent was obtained from all participants.

Selection Strategies

According to a recent study[21] with a crossover design, the MVIC is equivalent to 245.5 ± 51.4 Nm and 307.7 ± 75.3 Nm with and without sleep deprivation respectively. If the difference between these values represents the true difference between conditions, a sample size of 14 participants was determined to achieve 95% of power to correctly reject the null hypothesis. The MVIC values were considered for sample size calculations, due to an absence of effects relative to the impact of sleep deprivation on the peak RTD in two previous studies.[20,21] Thus, 14 young and healthy volunteers (8 men, 6 women; mean age: 21.8 ± 2.5 years) were included in the study. Recruitment was performed in local communities and the institution's surroundings via word of mouth.

Inclusion and Exclusion Criteria

Participants taking no medications, with normal blood pressure ($\leq$ 120/80 mmHg)[22] were included. The exclusion criteria were active smoking status, known metabolic disease, cardiovascular disease, respiratory disorders, and orthopedic issues limiting exercise performance (assessed by a health-screening questionnaire). At baseline, the participants filled

out a questionnaire on quality of sleep, the Portuguese version of the Pittsburg Sleep Quality Index (PSQI-PT).[23] Poor sleep quality was defined as an exclusion criterion by a cut-off value of ≥ 5 (on a scale of 0 to 21). Moreover, the participants were asked to follow normal sleep-wake patterns and to record sleep time on the three days preceding each testing session (except during the night of sleep deprivation), which was reported upon arrival at the laboratory. This was performed to exclude all participants exhibiting persistent sleep restriction (< 7 hours of sleep duration/day) that could interfere with the experimentally-induced effects of 24 hours of sleep deprivation.

Study Design

The present study has a crossover design, with measurements taken with and without 24 hours of sleep deprivation and the participants acting as their own controls. The participants were familiarized with the experimental protocol before the first testing day. Each participant was required to visit the laboratory twice, with an interval of 5 to 7 days between testing sessions. In a randomized and counterbalanced order, each session was preceded by either a night of normal sleep (without sleep deprivation) or a night of total sleep deprivation. The participants were asked to arrive at the laboratory early in the morning, with a fasting period of 4 hours. All testing sessions were conducted between 7am and 11am, to avoid daily variations in muscle strength related to human circadian rhythms.[24] Each participant was requested to avoid ingestion of xanthine derivatives 8 to 12 hours before the experimental sessions and physical exercise for at least 24 hours before testing. On each session, the participants were tested for plantar flexion RTD (isometric testing), with surface EMG recordings of the soleus muscle.

Sleep Conditions

In the experimental session without sleep deprivation, the participants were instructed to sleep adequately at home, for 7 to 9 hours, and to record the precise time they got into and out of bed. After waking up, they reported to the laboratory for testing. In the sleep deprivation condition, volunteers were advised to remain at home, where they were allowed to perform sedentary and light activities, limiting the differences in physical activity and mental stress between conditions.[25,26] The participants were asked to return to the laboratory early in the morning, in a sleep-deprived state. They were provided with a GT3X+ accelerometer (Actigraph, LLC, Pensacola, FL, United States), as used in previous research on sleep restriction and physical performance,[27] in order to confirm the sleep deprivation status of each participant. This device has been shown to be valid and reliable to detect sleep–wake patterns.[28,29] Further methodology details on activity-based monitoring can be consulted on previous research.[5]

Instrumentation

Characterization of the Participants

Anthropometric measurements (height and body mass) were taken at baseline using a stadiometer and a standing digital scale (BF350, Tanita Corporation, Tokyo, Japan). Blood pressure was also measured at baseline in duplicate (Tango, SunTech Medical, Inc., Morrisville, NC, United States).

Torque

All assessments were made unilaterally on the participants' dominant lower limb, using a System 3 Pro isokinetic dynamometer (Biodex Medical Systems, Inc., Shirley, NY, United States). The measurements were taken in the seated position, with the participants maintaining their hips and knee flexed at 120° and ankle at 110° of plantar flexion.[30] The flexed position at the knee joint was chosen to reduce the mechanical contribution of the gastrocnemii to the plantar flexor contraction.[31] The axis of rotation of the dynamometer was aligned with the anatomical ankle flexion–extension axis. The dynamometer torque signal was sampled at an analog-to-digital conversion rate of 1,000 Hz by using an external analog-to-digital converter (USB-6251, Emerson, St. Louis, MO, United States). Then, the signal was smoothed offline using a digital fourth-order, zero-lag Butterworth filter with a low-pass cutoff frequency of 10 Hz.

Surface Electromyography

In both testing sessions, the soleus surface EMG electrode (DE 2.x series EMG sensors, Delsys, Inc., Natick, MA, United States) was placed in accordance with the recommendations for the noninvasive assessment of muscle electromyographic signals.[32] To decrease the impedance of the interface between the skin and the electrode, the skin was prepared by removing hair through abrasion and then cleaning with alcohol. A two-slot adhesive interface was used to firmly stick each EMG sensor to the skin. A strap electrode soaked in water was used as ground, positioned at the ankle distal to the recording electrode. Once the appropriate electrode placements were confirmed, these locations were marked with indelible ink to ensure consistency for future test sessions. Surface EMG signals were band-pass filtered (10 Hz to 2,000 Hz) and amplified to a total gain of 1,000 using a Bagnoli-8 amplifier (Delsys, Inc.). Then, a 16-bit analog-to-digital converter (USB-6251, Emerson) was used to sample the signal at 10 kHz. The EMG data were recorded in synchrony with the torque signal originating from the System 3 Pro isokinetic dynamometer (Biodex Medical Systems, Inc.) using the Mr. Kick software (Knud Larsen, Center for Sensory-Motor Interaction , Aalborg University, Aalborg, Denmark).

Percutaneous Electrical Stimulation

The posterior tibial nerve was stimulated with a constant current isolated stimulator (STMISOLA, Biopac Systems, Inc., Goleta, CA, United States). Each participant was initially familiarized with a range of electrical stimuli (1 mA to 40 mA) over a period of ~ 5 minutes. A self-adhesive cathode (with 8 mm in diameter, silver/silver chloride [Ag/AgCl]) was placed in the popliteal fossa, and the anode (5×10, Compex Médical SA, Écublens, Switzerland) was placed proximal to the patella. The final placement of the cathode was identified using a handheld ball-milled cathode (with 0.5 cm in

diameter). Once the position eliciting the greatest response with the minimum stimulus intensity was determined, the stimulation electrode was firmly fixed to this site with rigid straps and taping.[18]

Experimental Procedures

M-wave Recruitment Curve

The motor response was elicited while each participant maintained a low-level tonic contraction of the plantar flexors (10% of MVIC) – all participants were provided with online visual feedback of the torque exerted, which was displayed on a computer monitor. Initially, by delivering three 1-ms rectangular pulses at each intensity level, the current intensity was increased by 5 mA from 0 until there was no further increase in the peak twitch torque, nor in the concomitant peak-to-peak M-wave amplitude. The current intensity was defined as that of the upper current. Then, to construct the M-wave recruitment curve, the upper intensity was divided into 22 segments that were equally separated on a logarithmic scale. For each current intensity, 16 stimuli were delivered at 3-s intervals with 2 m of rest every 88 stimulations.[33]

Rate of Torque Development

The RTD for the planta -flexion exercise was determined based on three isometric contractions, and the trial with the highest peak RTD was used for analysis.[10] The participants were instructed to exert their maximum force as "fast and hard as possible", and were then allowed to rest for 60 s between each trial.[7] According to the available literature, this verbal instruction is well suited to maximize the RTD during voluntary contractions.[9] The torque level was displayed on a sensitive scale to provide biofeedback, and the participants were instructed to avoid countermovement or pre-tension (change in baseline torque during the 100 ms prior to contraction onset). Plantar flexions were repeated whenever pre-tension or countermovement were identified. Each testing session began with a brief warm-up protocol consisting of 5 to 10 submaximal isometric plantar flexions (at 60% to 70% of the maximum effort perceived by the participant).

Data Analysis

Rate of Torque Development

The signal onsets of all voluntary contractions were visually identified following previous recommendations.[29] The RTD (Δ torque/Δ time) was quantified as the average slope of the torque-time curve for 4 sequential time periods (0 ms to 30 ms; 30 ms to 50 ms; 50 ms to 100 ms; and 100 ms to 150 ms). Finally, we also computed the peak RTD as the steepest part of the curve using a moving sampling window of 20 ms.[8]

Electromyography

Average rectified values (ARVs) were extracted from the soleus EMG recordings. The ARVs were calculated over the following time windows: 0 ms to 50 ms, 50 ms to 100 ms, and 100 ms to 150 ms. The peak-to-peak amplitude of the M-wave was computed offline from unrectified EMG epochs. To reduce the variability among participants, the soleus ARV was normalized to the maximum motor wave (Mmax, taken as the single largest M-wave recorded during the construction of the recruitment wave).[10,35]

Statistical Analysis

All data were tested for normality using the Shapiro-Wilk test. Paired-samples *t*-tests were used to explore differences between conditions in sleep-wake patterns (with versus without sleep deprivation) and the peak RTD. Two-way analysis of variance (ANOVA) with repeated measures was used to evaluate the effects of sleep deprivation on the sequential RTD obtained throughout the rising torque-time curve (Condition [2]: with sleep deprivation versus without sleep deprivation x Time [4]: RTD 0–30 ms versus RTD 30–50 ms versus RTD 50–100 ms versus RTD 100–150 ms). A similar approach was used to explore the impact of sleep deprivation on the sequential soleus EMG obtained throughout the rising torque-time curve (Condition [2]: with sleep deprivation versus without sleep deprivation x Time [3]: ARV 0–50 ms versus ARV 50–100 ms versus ARV 100–150 ms). The Greenhouse-Geisser corrected values were used when the assumption of sphericity was violated. Adjustment for multiple comparisons was made with the Bonferroni correction. The partial eta-squared values (η^2; proportion of total variance that is attributable to an effect) were reported for all significant and non-significant main effect analyses (small effect: 0.010–0.060; medium effect: 0.060–0.140; large effect: >0.140).[36] All data are reported as mean $\pm$ standard deviation (SD) values. Statistical significance was set at $p<0.05$. Data analysis was performed using the IBM SPSS Statistics for Windows (IBM Corp., Armonk, NY, United States) software, version 25.0.

Results

None of the 14 participants (mean age: 21.8 ± 2.5 years; mean height: 168.7 ± 7.3 cm; mean weight: 63.0 ± 9.6 kg; and mean body mass index [BMI]: 22.1 ± 2.2 kg/m^2) showed any signs of persistent sleep restriction on the 3 days that preceded each experimental condition (sleep duration >7.5 h/day). Moreover, no participant was excluded at baseline due to poor sleep quality (mean PSQI-PT score of 3.4 ± 1.3). Sleep efficiency and the number of physically-active episodes during the nights that preceded both experimental sessions were both significantly different under the two conditions ($p<0.05$), which enabled us to confirm sleep deprivation in each participant. These data were published in Gonçalves et al.[5]

The peak RTD obtained during plantar flexion MVIC was similar regarding the two conditions (without sleep deprivation: 283.3 ± 111.6 N.m.s^{-1}; with sleep deprivation: 294.9 ± 99.2 N.m.s^{-1}; $t=-0.67$; $p=0.512$; $\eta^2=0.034$) (►**Figure 1**). The RTD and soleus ARV data throughout the rising force-time curve with and without sleep

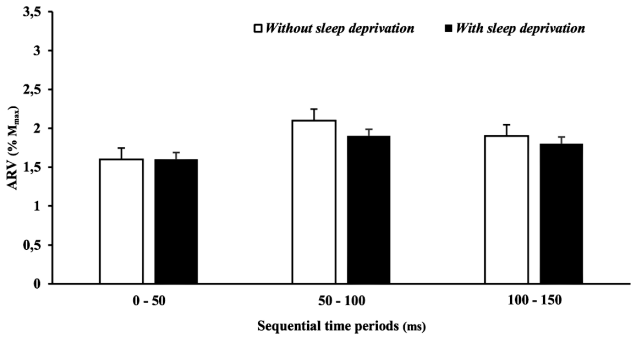

Fig. 1 Peak RTD obtained with and without sleep deprivation. The dashed lines represent individual participants and the solid black line represents the mean value of all cases.

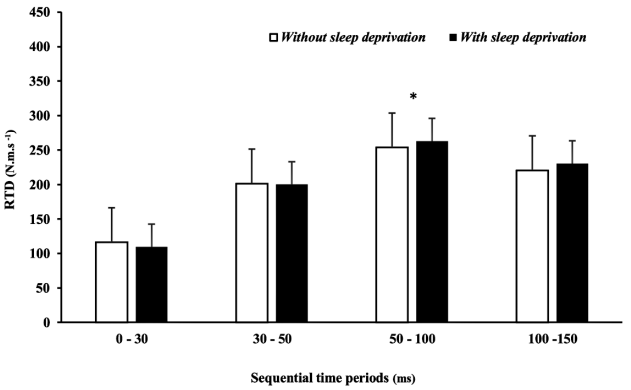

Fig. 2 Absolute RTD obtained with and without sleep deprivation at four different epochs of torque production during MVICs. Note: *Significantly different from the 0-30 ms and 30- 50 ms epochs ($p < 0.0001$).

deprivation are depicted in ►**Table 1**. The sequential RTD revealed no significant main effects for condition ($F = 0.09$; $p = 0.762$; $\eta^2 = 0.009$) or condition-by-time interaction ($F = 0.58$; $p = 0.506$; $\eta^2 = 0.050$). In contrast, there was a significant time main effect for the sequential RTD ($F = 31.8$; $p < 0.0001$; $\eta^2 = 0.743$). Post-hoc tests revealed that the steepest portion of the torque-time curve was attained during the first 50 ms of contraction. The RTD increased progressively from the 0-30 ms to the 30-50 ms interval (RTD 0-30 ms versus RTD 30-50ms versus RTD 50-100 ms; $p < 0.0001$). From then on, the TD did not exhibit further changes (RTD 50-100 ms versus RTD 100-150 ms; $p = 0.267$) (►**Figure 2**).

The soleus ARV obtained at selected time windows throughout the rising torque-time curve was similar under the two conditions (condition main effect: $F = 0.09$; $p = 0.774$; $\eta^2 = 0.008$) and remained unchanged over time (time main effect: $F = 2.1$; $p = 0.147$; $\eta^2 = 0.160$) (►**Figure 3**). Finally, there were no condition-by-time interactions for the soleus ARV ($F = 0.05$; $p = 0.951$; $\eta^2 = 0.005$).

Discussion

The present study aimed to investigate the effects of 24 hours of sleep deprivation on the sequential periods of RTD obtained throughout the torque-time curve in response to plantar flexion MVIC and the corresponding normalized EMG amplitude of the soleus muscle. We found that neither the RTD, nor the soleus sequential EMG were altered after 24 hours of sleep deprivation. These results agree with those of past reports[18,19,21] demonstrating that the peak RTD and EMG amplitude remain well preserved after 48-60 hours of sleep deprivation. However, the novelty of the present study is that, in contrast with what was hypothesized, sleep deprivation had virtually no impact on the RTD obtained either in the early or the last stage of MVIC.

There is general agreement that the RTD is highly related to physical performance in sports and functionality in tasks of daily living.[9] The RTD is rapidly and profoundly affected by

Table 1 Absolute RTD and normalized soleus ARV obtained with and without sleep deprivation during MVICs.

Variables	Without sleep deprivation: mean ± SD	With sleep deprivation: mean ± SD	*p*-value	Eta-squared values
RTD				
RTD $_{0-30\,ms}$ (in N.m.s^{-1})	116.2 ± 16.0	109.4 ± 13.9	0.462	0.050
RTD $_{30-50\,ms}$ (in N.m.s^{-1})	201.4 ± 26.9	199.9 ± 25.8	0.894	0.002
RTD $_{50-100\,ms}$ (in N.m.s^{-1})	253.7 ± 31.9	262.9 ± 32.2	0.525	0.038
RTD $_{100-150\,ms}$ (in N.m.s^{-1})	220.6 ± 29.6	230.4 ± 27.4	0.544	0.034
Peak RTD (N.m.s^{-1})	283.3 ± 111.6	294.9 ± 99.2	0.512	0.034
Soleus EMG				
ARV $_{0-50\,ms}$ (% Mmax)	1.6 ± 0.3	1.6 ± 0.3	0.946	0.001
ARV $_{50-100\,ms}$ (% Mmax)	2.1 ± 0.3	1.9 ± 0.4	0.703	0.014
ARV $_{100-150\,ms}$ (% Mmax)	1.9 ± 0.2	1.8 ± 0.2	0.809	0.006

Abbreviations: ARV, average rectified value; EMG, electromyography; MVIC, maximum voluntary isometric contraction; RTD, rate of torque development; SD, standard deviation.

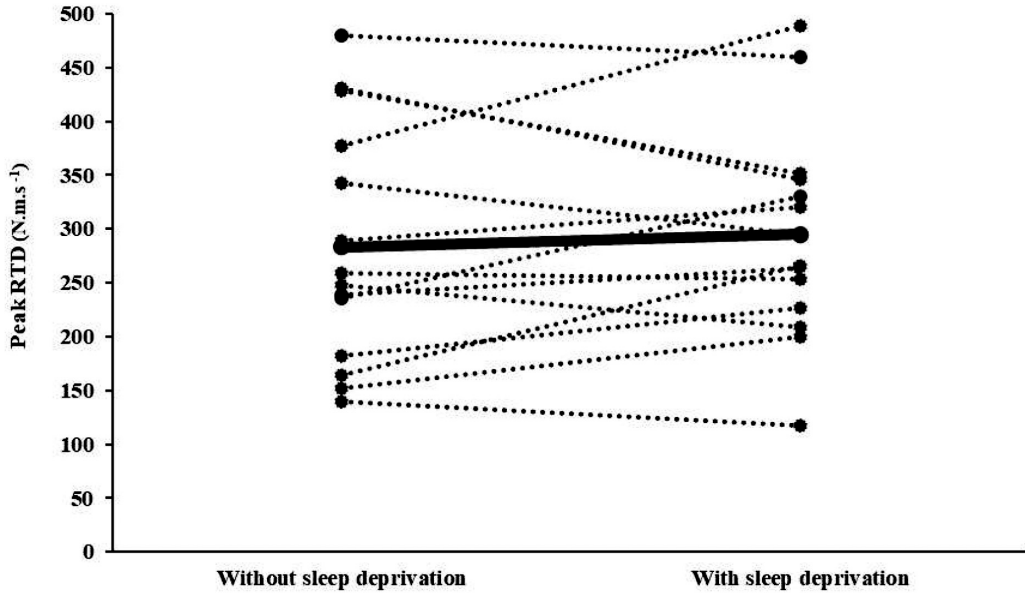

Fig. 3 Normalized soleus ARV of EMG signal obtained with and without sleep deprivation at three different epochs of torque production during MVICs.

fatigue; this concept is corroborated by previous studies[35] showing that, in a fatigued state, the magnitude of the decrement in explosive torque production is even greater than that observed on MVIC. Even though there is undisputed evidence that fatigue is insidiously manifested in sleep-deprived persons,[37] previous research has reported no interaction between sleep deprivation and the peak RTD. The peak RTD provides a poor resolution of torque development during the initial stages of MVIC and may not capture the first 50 ms of contraction – an epoch that is strongly associated with the ability of the central nervous system to induce maximum muscle activation during MVIC.[10,11] Furthermore, previous research[11,35,36] has also shown that while the early stage of explosive torque development (0–50 ms) is highly susceptible to fatigue, this is not the case for the RTD obtained at subsequent time points ($>$ 50 ms). Thus, we hypothesized that the sequential analysis of torque development throughout the rising portion of the torque–time curve might provide a better insight into the impact of sleep deprivation on neuromuscular function. In contrast to what was hypothesized, the findings of the present study indicate that 24 hours of sleep deprivation do not elicit any changes in sequential RTD values (including the first 50 ms of torque production).

Experimentally-induced total sleep deprivation is a defined period of at least 24 hours of extended wakefulness, which directly causes subjective feelings of tiredness and exhaustion.[37] Even relatively-moderate sleep restriction (sleep period of up to 6 hours per night) can seriously disturb neurobehavioral function (causing deficits in cognitive performance, for example) in healthy adults, which is equivalent to the impairment verified after two nights of sleep deprivation.[39] Moreover, it has been shown that 24 hours of sleep deprivation leads to substantial impairments in vigor and mood.[21] Despite all of these factors, according to the data of the present study, the neurophysiological basis of explosive torque production remains well preserved in a state of sleep deprivation Although neither athletic nor older populations were directly measured, these findings are relevant because they provide preliminary evidence that, even after 24 hours of sleep deprivation, explosive force may be well preserved in response to athletic motor tasks (jumps, throws, sprints, for example) involving limited time for torque production (such as 100-300 ms;. In addition, from a functional perspective, these findings also show that one night of sleep deprivation is not likely to disturb the ability to rapidly regain balance during sudden postural perturbations (such as during a fall).[7]

As with the RTD, the soleus EMG was also measured at different epochs of contraction. This was done to further examine the hypothesis of reduced muscle activation in a state of sleep deprivation. There is general agreement that the ability to achieve high levels of neural drive at the onset of muscle contraction is intimately related with the magnitude of torque production during the first milliseconds of MVIC.[10] In fact, previous reports showed a strong relationship between the EMG amplitude of agonist muscles (such as the rectus femoris, vastus lateralis, vastus medialis) and measures of early-stage explosive voluntary force production (impulse 0–50 ms),[12,40] with muscle determinants explaining no more than 18% to 30% of the variance in the RTD for this specific time interval.[41] It has also been shown that neuromuscular fatigue is accompanied by a decline ($\sim$ 30%) in EMG amplitude during the first 50 ms of explosive contractions,[35] and this is likely secondary to altered motor-unit recruitment and firing frequency during the early phase of muscle contraction.[11]

The effects of sleep deprivation on mental or attentional fatigue are relative unequivocal,[1] and they may directly affect behavior. Sleep deprivation is associated with a greater perception of task difficulty.[37] In addition, it decreases

logical reasoning, coding, decision-making, alertness and filtering efficiency,[42,43] all of which might lead to impaired neural drive to the active muscles (such as central fatigue). Despite this, 24 hours of sleep deprivation did not elicit significant changes in the soleus EMG in up to 150 ms of plantar flexion MVIC. These results are well aligned with those of previous reports showing that 24 hours of sleep deprivation exerted an innocuous effect on motor-evoked potentials,[25] V-wave normalized amplitude,[5] and central activation ratio.[21]

Limitations

The present study has at least two important limitations. First, actigraphy was not used to determine the sleep quality and total time during the three nights that preceded each experimental session. Instead, to ensure that none of the participants arrived at the laboratory in a sleep-restricted state, they were all asked to record and report their total sleep time in those specific days. Second, the EMG was obtained only for the soleus muscle. This specific muscle is well known for its limited ability to develop torque rapidly during maximal contractions,[7] resulting from its specific type-I fiber composition, with slow contractile velocity.[44]

Conclusion

We have provided preliminary evidence that 24 hours of sleep deprivation do not affect the rate of rise of the torque–time curve, nor the soleus EMG in response to MVIC. Thus, is likely that exercise performance or daily functionality in tasks involving explosive torque production remain well preserved after one night of sleep deprivation. Nevertheless, it cannot be excluded that some components of human performance may still be affected[1] when individual motivation[3] and perceived exertion,[25,45] resulting from the disturbance of the circadian rhythms caused by sleep deprivation, are compromised.

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Conflict of Interests

The authors have no conflict of interests to declare.

References

1 Fullagar HHK, Skorski S, Duffield R, Hammes D, Coutts AJ, Meyer T. Sleep and athletic performance: the effects of sleep loss on exercise performance, and physiological and cognitive responses to exercise. Sports Med 2015;45(02):161–186
2 Knowles OE, Drinkwater EJ, Urwin CS, Lamon S, Aisbett B. Inadequate sleep and muscle strength: Implications for resistance training. J Sci Med Sport 2018;21(09):959–968
3 Brotherton EJ, Moseley SE, Langan-Evans C, et al. Effects of two nights partial sleep deprivation on an evening submaximal weightlifting performance; are 1 h powernaps useful on the day of competition? Chronobiol Int 2019;36(03):407–426
4 Watson AM. Sleep and Athletic Performance. Curr Sports Med Rep 2017;16(06):413–418
5 Gonçalves AD, Teodosio C, Pezarat-Correia P, Vila-Chã C, Mendonca GV. Effects of acute sleep deprivation on H reflex and V wave. J Sleep Res 2021;30(03):e13118
6 Latash ML. Muscle coactivation: definitions, mechanisms, and functions. J Neurophysiol 2018;120(01):88–104
7 Johnson ST, Kipp K, Norcross MF, Hoffman MA. Spinal and supraspinal motor control predictors of rate of torque development. Scand J Med Sci Sports 2015;25(05):623–629
8 Haff GG, Ruben RP, Lider J, Twine C, Cormie P. A comparison of methods for determining the rate of force development during isometric midthigh clean pulls. J Strength Cond Res 2015;29(02):386–395
9 Maffiuletti NA, Aagaard P, Blazevich AJ, Folland J, Tillin N, Duchateau J. Rate of force development: physiological and methodological considerations. Eur J Appl Physiol 2016;116(06):1091–1116
10 Folland JP, Buckthorpe MW, Hannah R. Human capacity for explosive force production: neural and contractile determinants. Scand J Med Sci Sports 2014;24(06):894–906
11 Morel B, Rouffet DM, Saboul D, Rota S, Clémençon M, Hautier CA. Peak torque and rate of torque development influence on repeated maximal exercise performance: contractile and neural contributions. PLoS One 2015;10(04):e0119719
12 Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P. Increased rate of force development and neural drive of human skeletal muscle following resistance training. J Appl Physiol 2002;93(04):1318–1326
13 Van Cutsem M, Duchateau J, Hainaut K. Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. J Physiol 1998;513(Pt 1):295–305
14 Aagaard P. Training-induced changes in neural function. Exerc Sport Sci Rev 2003;31(02):61–67
15 Lanza MB, Balshaw TG, Massey GJ, Folland JP. Does normalization of voluntary EMG amplitude to M_{MAX} account for the influence of electrode location and adiposity? Scand J Med Sci Sports 2018;28(12):2558–2566
16 Tucker KJ, Tuncer M, Türker KS. A review of the H-reflex and M-wave in the human triceps surae. Hum Mov Sci 2005;24(5-6):667–688
17 Rozand V, Grospetre S, Stapley P, Lepers R. Assessment of Neuromuscular Function Stimulation UPEN. J Vis Exp 2015;103:1–11
18 Mendonca GV, Teodósio C, Mouro M, et al. Improving the reliability of V-wave responses in the soleus muscle. J Clin Neurophysiol 2019;36(02):97–103
19 Symons JD, Bell DG, Pope J, VanHelder T, Myles WS. Electromechanical response times and muscle strength after sleep deprivation. Can J Sport Sci 1988;13(04):225–230
20 Vaara JP, Oksanen H, Kyröläinen H, Virmavirta M, Koski H, Finni T. 60-Hour Sleep Deprivation Affects Submaximal but Not Maximal Physical Performance. Front Physiol 2018;9:1437
21 Skurvydas A, Kazlauskaite D, Zlibinaite L, et al. Effects of two nights of sleep deprivation on executive function and central and peripheral fatigue during maximal voluntary contraction lasting 60s. Physiol Behav 2021;229:113226
22 Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Hypertension 2018;71(06):e13–e115
23 Del Rio João KA, Becker NB, de Neves Jesus S, Isabel Santos Martins R. Validation of the Portuguese version of the Pittsburgh Sleep Quality Index (PSQI-PT). Psychiatry Res 2017;247:225–229
24 Araujo LG, Waterhouse J, Edwards B, Santos EHR, Tufik S, Tlio De Mello M. Twenty- four-hour rhythms of muscle strength with a

consideration of some methodological problems. Biol Rhythm Res 2011;42(06):473–490
25 Temesi J, Arnal PJ, Davranche K, et al. Does central fatigue explain reduced cycling after complete sleep deprivation? Med Sci Sports Exerc 2013;45(12):2243–2253
26 Arnal PJ, Lapole T, Erblang M, et al. Sleep Extension before Sleep Loss: Effects on Performance and Neuromuscular Function. Med Sci Sports Exerc 2016;48(08):1595–1603
27 Chase JD, Roberson PA, Saunders MJ, Hargens TA, Womack CJ, Luden ND. One night of sleep restriction following heavy exercise impairs 3-km cycling time-trial performance in the morning. Appl Physiol Nutr Metab 2017;42(09):909–915
28 Cellini N, Buman MP, McDevitt EA, Ricker AA, Mednick SC. Direct comparison of two actigraphy devices with polysomnographically recorded naps in healthy young adults. Chronobiol Int 2013; 30(05):691–698
29 Cole RJ, Kripke DF, Gruen W, Mullaney DJ, Gillin JC. Automatic sleep/wake identification from wrist activity. Sleep 1992;15(05): 461–469
30 Pierrot-Deseilligny E, Burke D. The circuitry of the human spinal cord: Its role in motor control and movement disorders. In:Cambridge: Cambridge University Press;; 2009
31 Cresswell AG, Löscher WN, Thorstensson A. Influence of gastrocnemius muscle length on triceps surae torque development and electromyographic activity in man. Exp Brain Res 1995;105(02):283–290
32 Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. J Electromyogr Kinesiol 2000;10(05):361–374
33 Vila-Chã C, Falla D, Correia MV, Farina D. Changes in H reflex and V wave following short-term endurance and strength training. J Appl Physiol 2012;112(01):54–63
34 Tillin NA, Jimenez-Reyes P, Pain MTG, Folland JP. Neuromuscular performance of explosive power athletes versus untrained individuals. Med Sci Sports Exerc 2010;42(04):781–790
35 Buckthorpe M, Pain MTG, Folland JP. Central fatigue contributes to the greater reductions in explosive than maximal strength with high-intensity fatigue. Exp Physiol 2014;99(07):964–973
36 Cohen J. Statistical power analysis for the behavioral sciences. In: New York: Routledge Academic;; 1988
37 Engle-Friedman M, Mathew GM, Martinova A, Armstrong F, Konstantinov V. The role of sleep deprivation and fatigue in the perception of task difficulty and use of heuristics. Sleep Sci 2018; 11(02):74–84
38 Jenkins NDM, Housh TJ, Traylor DA, et al. The rate of torque development: a unique, non-invasive indicator of eccentric-induced muscle damage? Int J Sports Med 2014;35(14): 1190–1195
39 Van Dongen HPA, Maislin G, Mullington JM, Dinges DF. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. Sleep 2003;26(02): 117–126
40 de Ruiter CJ, Van Leeuwen D, Heijblom A, Bobbert MF, de Haan A. Fast unilateral isometric knee extension torque development and bilateral jump height. Med Sci Sports Exerc 2006;38(10): 1843–1852
41 Andersen LL, Aagaard P. Influence of maximal muscle strength and intrinsic muscle contractile properties on contractile rate of force development. Eur J Appl Physiol 2006;96 (01):46–52
42 Drummond SPA, Anderson DE, Straus LD, Vogel EK, Perez VB. The effects of two types of sleep deprivation on visual working memory capacity and filtering efficiency. PLoS One 2012;7(04): e35653
43 Harrison Y, Horne JA. One night of sleep loss impairs innovative thinking and flexible decision making. Organ Behav Hum Decis Process 1999;78(02):128–145
44 Johnson MA, Polgar J, Weightman D, Appleton D. Data on the distribution of fibre types in thirty-six human muscles. An autopsy study. J Neurol Sci 1973;18(01):111–129
45 Roberts SSH, Teo WP, Aisbett B, Warmington SA. Effects of total sleep deprivation on endurance cycling performance and heart rate indices used for monitoring athlete readiness. J Sports Sci 2019;37(23):2691–2701

