Selective REM sleep deprivation during daytime: 
I. Time course of interventions and recovery sleep

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Selective REM sleep deprivation during daytime: II. Muscle atonia in NREM sleep

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Abstract

While repeated selective REM sleep deprivation by awakenings during nighttime has shown that the number of sleep interruptions required to prevent REM sleep increases within and across consecutive nights, the underlying regulatory processes remained unspecified. To assess the role of circadian and homeostatic factors in REM sleep regulation, REM sleep was selectively deprived in healthy young adult males during a daytime sleep episode (07 – 15 h) after a night without sleep. Circadian REM sleep propensity is known to be high in the early morning. The number of interventions required to prevent REM sleep increased from the first to the third 2-hour interval by a factor of two and then leveled off. Only a minor REM sleep rebound (11.6 %) occurred in the following undisturbed recovery night. It is concluded that the limited rise of interventions during selective daytime REM sleep deprivation may be due to the declining circadian REM sleep propensity, which may partly offset the homeostatic drive and the sleep-dependent disinhibition of REM sleep.

Key words: nonREM sleep; sleep homeostasis; sleep regulation; spectral analysis.

Abbreviations

FFT: fast Fourier transform; FIR: finite impulse response; NREM sleep: non-rapid eye movement sleep; rANOVA: ANOVA for repeated measures; RD: REM sleep deprivation; REM sleep: rapid eye movement sleep; SWA: power in the 0.75-4.5 Hz range of the sleep EEG; TST: total sleep time
Introduction

There is a long-standing interest in the mechanisms underlying rapid eye movement (REM) sleep regulation (see (17) for a recent review). Homeostatic and circadian factors can be discriminated. The homeostatic drive is evident from the REM sleep rebound that is induced by a selective REM sleep deprivation schedule (1, 12, 15, 18, 21), and from the increasing number of interventions required to prevent REM sleep (1, 12, 15, 16, 18, 21). There is also indication that a sleep-dependent disinhibition contributes to the rise in REM sleep during a sleep episode (5, 14). The circadian modulation of REM sleep is apparent from the variation of the REM sleep fraction of sleep within the NREM-REM sleep cycle, and the duration of the REM sleep episode (10, 22). Both exhibit their maximum in close proximity to the minimum of core body temperature, which under entrained conditions is in the early morning hours.

The homeostatic and circadian facets of REM sleep regulation interact, yet their respective influences are difficult to disentangle. This was in particular obvious in our recent 3-day selective REM sleep deprivation study (15). The dramatic rise in the number of interventions within the REM sleep deprivation nights could be attributed to the increasing homeostatic drive as the manifestation of REM sleep was prevented and also to the disinhibition of REM sleep as NREM sleep intensity declined in the course of sleep. Another possible contributing factor was the rise in circadian REM sleep propensity during a nighttime sleep episode. Since it was reasonable to assume that all these influences were present, we were faced with the problem of assessing their respective strength. An increasing homeostatic drive for REM sleep was not only evident from the rising number of interventions within the deprivation nights but also from their increase from night to night. However, the latter change was rather modest
and also the REM sleep rebound after the 3-day selective deprivation period was moderate.

In the present study we attempted to specify the role of homeostatic and circadian factors in REM sleep regulation. In contrast to our previous study (15) REM sleep was selectively deprived during a sleep episode scheduled during the daytime. This protocol was chosen because the circadian drive for REM sleep is large in the morning and then decreases, whereas the homeostatic and sleep-dependent drive of REM sleep change in the opposite direction. The main question was how the number of interventions required to prevent REM sleep would vary during daytime sleep and how it would affect the distribution of REM sleep in the recovery night.

Method

Subjects and Protocol

Twelve male subjects (mean 24±0.17 years), recruited from the student population, were paid for their participation in the study. Only 11 subjects were analyzed (see Statistics). The local ethical committee for research on human subjects approved the study protocol and written informed consent was obtained from the subjects prior to the study. The work fully conforms to the guiding principles for research involving animals and human beings (American Physiological Society). The current health status, medical history and subjective sleep quality were assessed by questionnaires. Only subjects who reported good health, no history of medical problems, no medication intake, and no sleep disturbances were included. A screening night served to exclude subjects with sleep apnea and nocturnal myoclonus. All subjects were nonsmokers, right-handed and
their habitual alcohol and caffeinated beverage intake was less than 6 glasses alcohol per week (mean 2.1) and less than 4 cups of caffeinated beverage per day (mean 1.3), respectively. On the three days preceding the study as well as during the study, the subjects were instructed to abstain from alcohol and caffeinated beverages, to keep a regular sleep-wake schedule (sleep from 23:00 to 7:00), and to refrain from napping. Compliance with these instructions was verified by continuous recording of wrist activity, and by determining breath-ethanol concentration when subjects came to the sleep laboratory.

The study protocol consisted of two sessions. Each session was composed of baseline sleep (23:00-7:00h; B1, B2), a wake episode of 24 h followed by daytime sleep (7:00-15:00h; E1, E2) and recovery sleep (23:00-7:00h; R1, R2). At least 4 days separated the two sessions. During the sleep deprivation night, the subjects were supervised by an experimenter to preclude involuntary napping or microsleeps. In all sleep episodes, subjects slept in the completely darkened bedrooms of the sleep laboratory. Subjects were repeatedly awakened during E1 to prevent REM sleep (RD condition). The second session served as a control condition. In E2 time in bed was restricted to match individually total sleep time (TST) of E1 by allowing a consolidated sleep episode. The fragmentation induced by repeated awakenings was not controlled for because disruptions of NREM sleep may have induced prolonged waking episodes.

During the selective REM sleep deprivation (E1), the subjects were awakened at the first sign(s) of REM sleep by switching on a dim red light (<5 lux) and entering the bedroom. The criteria for interventions were a desynchronized EEG without spindles or K-complexes and the concomitant reduction of the tonic electromyogram (EMG) amplitude for at least 20 s. A further criterion was the appearance of rapid eye
movements (REMs). However, the first appearance of REMs could not be used as a major criterion because the scoring rules of Rechtschaffen and Kales (20) necessitate the retroactive scoring of REM sleep on their occurrence. This would have precluded an effective REM sleep deprivation. After awakening from REM sleep, the subjects were kept awake for 2 min and were asked to complete a questionnaire and visual analog scales, and perform a letter cancellation task. A 2-min awakening period was selected on the basis of an earlier study (15). Dim red light was used to minimize light-induced effects on the circadian system.

Polygraphic Recordings

During sleep episodes the EEG (derivations: C3A2 and F3, P3, O1, F4, C4, P4, O2 and Cz referenced to C3), submental EMG, electrooculogram (EOG), and electrocardiogram (ECG) were recorded by a polygraphic amplifier (PSA24, Braintronics Inc., Almere, The Netherlands), digitized, and transmitted via fiber-optic cables to a personal computer. The EOG was recorded by a bipolar derivation. EEG, EMG and EOG signals were conditioned by the following analog filters: a high-pass filter (-3 dB at 0.16 Hz), a low-pass filter (-3 dB at 102 Hz, <-40 dB at 256 Hz), and a notch filter (50 Hz). Data were sampled with a frequency of 512 Hz, digitally filtered (EEG and EOG: low-pass FIR filter, -3 dB at 49 Hz; EMG: band-pass FIR filter, -3 dB points at 15.6 and 54 Hz), and stored on a PC with a resolution of 128 Hz. Vigilance states were visually scored for 20-s epochs according to standard criteria (20). Sleep onset was defined as first occurrence of stage 2. Power spectra of consecutive 20-s epochs (FFT routine, Hanning window, averages of five 4-s epochs) were computed for C3A2. The frequency resolution was 0.25 Hz and frequencies up to 25 Hz were analyzed. The two lowest
frequency bins (0.25 and 0.5 Hz) were excluded from the analysis because of numerous low-frequency artifacts (due to sweating etc.). Artifacts were identified by visual inspection and a semi-automatic procedure (i.e. power in the 0.75-4.5 Hz or 20-30 Hz band exceeding a threshold based on a moving average determined over 15 20-s epochs). Only 20-s epochs without artifacts were used for analysis.

**Simulations**

Simulations with the two-process model of sleep regulation (11) were performed based on the mean duration of sleep and waking in the control condition (the differences of time in bed between E1 and E2 were disregarded). The time constants (increase of S: 4.2 h; decrease of S: 18.2 h) corresponded to those used in Daan et al. (11).

**Statistics**

Three-, two- or one-way ANOVAs for repeated measures (rANOVA) with Huynh-Feldt correction were performed (for details see text). Post-hoc comparisons were performed with two-tailed paired t-tests. One subject was excluded from the analysis because both baseline recordings showed very low sleep efficiency (<75%). Thus 11 subjects contributed to the present analysis. Due to technical problems, one recording during selective REM sleep deprivation (E1) was lost. In three subjects the first baseline recording (B1) could not be used. Two had sleep efficiency below 80% and one reported a stomach-ache. These recordings were excluded or replaced with the corresponding data of B2 for performing rANOVAs. Post-hoc testing was performed with baseline mean (Bm) values. Some aspects were analyzed based on 2-h intervals. To
test for possible interference effects with sleep cycles, also analyses based on 90- and 100-min intervals were performed. However, because no principal difference in the time course was observed, only data of 2-h intervals are reported.

**Results**

**Number of interventions**

On average 19.4±2.4 (mean ± SE; N=11; range: 9-34) awakenings were required to prevent REM sleep. There was a considerable individual variation (Fig. 1A, left). The interventions per hour of sleep increased over the first three 2-h intervals (Fig. 1A, right) and then remained at the same level. The interventions were expressed relative to sleep because sleep efficiency decreased in the course of REM sleep deprivation (2-h interval 1-4: 86.25±1.08 %, 78.28±2.41 %, 63.64±3.90 %, 50.25±10.26 %, n=10, 1-way rANOVA factor '2-h interval', F3,27=9.51, p<0.0044). The attempts to enter REM sleep tended to occur in clusters (data not shown). Intervals of consolidated NREM sleep and epochs of extended waking separated the repeated attempts to enter REM sleep.

During uninterrupted daytime sleep (E2), REM sleep expressed as percentage of total sleep time appeared to increase from the first to the third 2-h interval (Fig. 1B, right). However, this increase was not significant.

**Sleep variables derived from visual scoring**

Table 1 summarizes the sleep variables for the different experimental conditions.
Experimental daytime sleep episodes. REM sleep was reduced to only 6.3 min (E1) compared to 73.5 min during undisturbed daytime sleep (E2). TST during the E2 control condition was restricted to match TST in E1. Slow wave sleep (SWS) did not differ significantly between the two daytime conditions. However, sleep efficiency was lower in E1 than in E2, while the amount of stage 1 and stage 2 was higher.

In comparison to baseline sleep, the daytime sleep episodes were characterized by shorter sleep latency. In the uninterrupted daytime sleep episode (E2) the amount of stage 2 was below baseline (BmE, truncated to match time in bed of E2), whereas the amount of stage 4 exceeded the baseline level. Other variables did not differ from baseline, in particular, the amount of REM sleep was similar between nighttime sleep (truncated) and uninterrupted daytime sleep.

Recovery nights. REM sleep was increased in the R1 recovery night following RD both relative to R2 and to the baseline night. There was less waking after sleep onset in R1 than in the baseline night. Stage 4 was below baseline in R2.

To investigate the time course of the REM sleep rebound, REM sleep was analyzed for consecutive 2-h intervals (Fig. 2). In baseline and recovery sleep, REM sleep increased over the course of the night (factor ‘2-h interval’). No significant differences between corresponding 2-h intervals were observed.

Slow-wave activity and simulation

The 24-h sleep deprivation increased SWA by 40.7% (mean of individual percentages) in E2 (undisturbed sleep) and by 33.1% in E1 (RD; Tab. 1) relative to baseline. This increase of SWA was significant in the first 2-h intervals (both conditions) and in the
second interval of E1 (Fig. 3B, left). The reduction of SWA in recovery sleep below baseline (R1: 3.9%; R2: 12.0%) was significant only for R2 (Tab. 1). SWA in the first 2-h interval of E2 was below baseline (Fig. 3B, right) and SWA in the third 2-h interval of R1 was significantly larger than in R2.

To test whether the changes in SWA were in accordance with the two-process model of sleep regulation (11), simulations were performed (Fig. 3A). After 24 h of continuous wakefulness (i.e. at the onset of the daytime sleep episode at 07.00 h), the level of Process S reached 122.7 % relative to the corresponding baseline value (100 %). At the onset of nighttime recovery sleep (23.00 h) S was at 89.0 % indicating a moderate reduction of sleep pressure when compared to baseline. A close correspondence between empirical SWA and the simulated level of S was observed (i.e. S was within the 95 % confidence interval for most 2-h intervals). S was below the empirical values in the third and fourth 2-h interval of R1 and in the fourth interval of R2.

**Discussion**

The selective REM sleep deprivation was effective in reducing REM sleep by more than 90 %. The time course of SWA was similar in the two experimental conditions and could be simulated on the basis of the two-process model (11). It was particularly interesting that the decline of SWA during daytime sleep was practically unaffected by the repeated awakenings required to prevent REM sleep. This observation is consistent with our previous study (15) and demonstrates that interference with REM sleep has little repercussions on the time course of SWA. It has been recently shown that a pharmacological elimination of REM sleep does not alter the time constant of Process S.
by which the exponential decline of SWA is described (19). Taken together, these observations demonstrate that the time course of SWA is little affected by the presence or absence of REM sleep.

**Processes affecting REM sleep**

In the previous 3-night REM sleep deprivation study, the number of interventions during the nighttime sleep episode showed a dramatic rise across the four consecutive 2-h intervals (Fig. 2 in (15)). This pattern was present in all three nights. Moreover, a modest night-to-night increase in the number of interventions was seen, representing a carry-over effect of the increase in REM sleep propensity. Two hypotheses were advanced to account for the results. The first hypothesis postulated a strong homeostatic drive and attributed the increasing number of interventions within the deprivation nights to its influence. However, since the ‘savings’ from night-to-night were modest, a functional substitution of waking for REM sleep was considered. The second hypothesis postulated a weak homeostatic drive as evidenced by the small night-to-night increase in the number of interventions and the moderate REM sleep rebound. According to this hypothesis, the rapid rise within the nights reflected the rise in circadian REM sleep propensity and the sleep-dependent disinhibition of REM sleep. The maximum of the REM sleep fraction of the NREM-REM sleep cycle is known to occur near the minimum of the circadian body temperature rhythm, which under entrained conditions is in the early morning hours (9).

In the present study the selective REM sleep deprivation began after 7 h in the morning, in temporal proximity to the circadian peak of REM sleep propensity. Similar to the previous nighttime deprivation schedule (15), the number of interventions was small in the first 2-h interval. The increased level of SWA as a consequence of prolonged
waking may have diminished the high circadian REM sleep propensity in this interval. The rise in the number of interventions from the first to the third interval was only about twofold and then leveled off. This contrasts with the 6-7-fold increase from the first to the fourth interval during the nighttime sleep episode (15). The increase in circadian REM sleep propensity during nighttime sleep may have enhanced the homeostatic and sleep-dependent REM sleep drive and thereby substantially contributed to the rapid rise in the number of interventions. In contrast, in the present study, the declining circadian REM sleep propensity during daytime sleep may have attenuated the rising trend due to homeostatic and sleep-dependent factors.

The percentage of REM sleep across 2-h intervals showed no significant trend (Fig. 1, lower part). This is in accordance with the results of a previous study in which sleep was scheduled to begin at 7 h in the morning (13). The prolongation of the waking episode preceding daytime sleep increased the propensity for NREM sleep (i.e. raised the level of Process S), which enhanced SWA (Tab. 1, Fig. 3). As has been mentioned above, the manifestation of REM sleep is inhibited when the propensity for NREM sleep is high. This accounts for the absence of a REM sleep rebound after 40 h of total sleep deprivation (6). However, a partial sleep deprivation schedule, which induces only a moderate rise in NREM sleep propensity, is followed by an increase in REM sleep (7, 8).

REM sleep deprivation gave rise to a modest but significant REM sleep rebound. As in the previous experiment (15) the increase in REM sleep was not limited to a particular phase of the sleep episode. A rise in REM sleep propensity is known to inhibit SWA (2). This may account for the observation that after selective REM sleep deprivation, SWA was enhanced in the third 2-h interval of the recovery night relative to the control
condition (Fig. 3). The decline of REM sleep propensity may have led to a disinhibition of SWA.

Benington and Heller (3, 4) proposed that it is the presence of nonREM sleep rather than the absence of REM sleep, which leads to an increase of REM sleep propensity. However, this hypothesis does not account for the difference in the time course of interventions during the present daytime REM sleep deprivation experiment and the previous nighttime experiment (15). The NREM sleep aspect was comparable in the two conditions.

In conclusion, the data from the present and previous experiment (15) provide evidence for the interaction of homeostatic and circadian components in REM sleep regulation. However, it remains difficult to quantify their contribution to REM sleep propensity. This is due to the differences of the two main markers (i.e. number of interventions during deprivation and REM sleep during recovery sleep), and the influence of NREM sleep propensity. A basic problem emerging from this study is whether the concept of REM sleep as a unitary state is a valid basis for investigating its regulatory aspects. The following companion paper addresses this question (23).

**Acknowledgements**

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**Figure legends**

**Figure 1:** A: Number of interventions required in selective REM sleep deprivation episodes (E1). Values are expressed as number of interventions per hour of sleep and plotted for consecutive 2-h intervals. Left: individual data; right: each bar represents mean values (+ SE; n=9; only the subjects with awakenings in the last 2-h interval were included). Statistics: 1-way rANOVA factor ‘2-h interval’ significant. * p<0.05 (2-tailed paired t-test) compared with following 2-h interval. B: Amount of REM sleep during the control experiment (E2). REM sleep is expressed as percentage of TST within the 2-h interval and plotted for the first three consecutive 2-h intervals. Due to matching TST in E2 with E1 average time in bed was only approximately 6 h. Left: individual data; right: mean values (+ SE; n=9). Statistics: 1-way rANOVA factor ‘2-h interval’ not significant.

**Figure 2:** Amount of REM sleep in consecutive 2-h intervals during baseline and recovery sleep (n=11, mean + SE). Bm (open bars): baseline mean; R1 (solid bars): recovery sleep after selective REM sleep deprivation; R2 (hatched bars): recovery sleep in control condition. Three-way rANOVA (factors ‘2-h interval’, ‘session’ (1, 2) and ‘condition’ (B, R)) revealed a significant effect for factor ‘2-h interval’ and interaction ‘condition’ x ‘session’. Two-way rANOVAs (factors ‘2-h interval’ and ‘condition’ (R1, R2), (Bm, R1), or (Bm, R2)) revealed a significant effect for factor 2-h interval in all tests performed and a significant interaction ‘2-h interval’ x ‘condition’ for the comparison of R1 and R2.
**Figure 3:** A: Simulation of homeostatic Process S (solid line) and time course of empirical slow-wave activity (SWA) in consecutive 2-h intervals (open circles: control condition; filled circles: RD condition). S was simulated based on the average timing of the control condition. The level of S at sleep onset during baseline was set to 100%. SWA values were standardized with mean SWA of baseline in the first 2-h interval. This value was assigned to the level of Process S after 1 h of sleep (77.1%). SWA data were plotted (mean ± SE) at interval midpoints. Bm: baseline mean; E1: daytime sleep with selective REM sleep deprivation; E2: control condition with total sleep time corresponding to E1; R1, R2: recovery nights. * S outside the 95% confidence interval of SWA data for R1; ° S outside the 95% confidence interval for R2. Dotted vertical line in experimental sleep denotes average duration of sleep in E2. No reliable value of SWA could be determined in the forth 2-h interval of E1 since subjects did not sleep much. B: Changes of SWA in daytime (left) and recovery sleep (right). Deviation of SWA from the corresponding 2-h interval of baseline mean (Bm) are plotted (mean ± SE). Solid bars: session 1, RD; open bars: session 2, control. Statistics: 3-way rANOVA (factors ‘condition’ ((B, E) or (B, R)), ‘session’ (1, 2), and ‘2-h interval’) on log-transformed SWA values. Significant effect of factors ‘condition’ and ‘interval’ during experimental sleep; significant effect of factor ‘interval’ and interactions ‘condition’ x ‘session’ and ‘condition’ x ‘interval’. * p<0.05 (2-tailed paired t-test) compared to Bm; ° p<0.05 R1 vs. R2.
Tables

Table 1: Sleep variables derived from visual scoring.

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<th>BmR (baseline)</th>
<th>R (recovery sleep)</th>
<th>BmE (truncated baseline)</th>
<th>E (experimental sleep)</th>
<th>Time in bed</th>
<th>Latency to stage 2</th>
<th>Total sleep time</th>
<th>Sleep efficiency (%)</th>
<th>REM sleep latency</th>
<th>Waking after sleep onset</th>
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<th>Stage 2</th>
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<td>8.09±0.51</td>
<td>6.42±0.83</td>
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<tr>
<td>SWA (µV²)</td>
<td>413.44±59.01</td>
<td>385.28±51.88</td>
<td>476.19±68.68</td>
<td>538.32±94.19°</td>
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<td>346.72±40.22°</td>
<td>572.60±82.40°</td>
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Variables are in min ± SE except for sleep efficiency that is expressed as total sleep time as a percentage of time in bed, and slow-wave activity (SWA) that is expressed as power (µV²) in the 0.75-4.5 Hz frequency range. Session 1 is the selective REM sleep deprivation condition (deprivation in E1), and session 2 the control condition (total sleep time in E2 matched with E1). BmR: Baseline mean (8 h time in bed); BmE: Baseline mean (truncated to match time in bed of E2). REM sleep latency is the time from sleep onset (first occurrence of stage 2) to the first occurrence of REM sleep. N=11 for the baseline and recovery sleep, n=10 for the experimental sleep (n=11 for REM sleep latency in E1). The first intervention or the first appearance of REM sleep was taken to calculate REM sleep latency in E1. Statistics: post-hoc 2-tailed paired t-tests (p<0.05)
performed when the preceding 2-way rANOVA with factor 'condition' (B, E) or (B, R) and factor 'session' (1, 2) or their interaction 'condition' x 'session' revealed significance.

* difference to control (session 2); ° difference to baseline mean. E2 was compared to truncated baseline data, i.e. the analysis interval was matched with time in bed of E2.
Fig. 1

A

# interventions (per hour of sleep within the 2h interval)

B

REMS (%) (per TST)

2-h interval
Fig. 2

REM sleep (min)

2-h interval

- Bm
- R1
- R2
Fig. 3

A

![Graph showing time of day vs. Process S/ SWA (%)]

Time of day (h)

Process S / SWA (%)

B

![Bar graphs showing SWA (%) deviation from Bm for E1 and E2, and R1 and R2]