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Effects of SWS deprivation on subsequent EEG power density and spontaneous sleep duration¹

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Summary In order to test predictions of the 2-process model of sleep regulation, the effects of slow wave sleep (SWS) deprivation by acoustic stimulation during the first part of the sleep period on EEG power density and sleep duration were investigated in 2 experiments. In the first experiment, 8 subjects were deprived of SWS during the first 5 h of a baseline nocturnal sleep period without awakening. Compared to the same interval of undisturbed sleep, power densities in the delta frequencies were attenuated. In the hour following SWS deprivation, power densities in the delta and theta frequencies were considerably enhanced in comparison with the same interval of undisturbed sleep. No change in sleep duration was observed. In the second experiment, 8 subjects were sleep deprived for 1 night and recovery sleep was initiated at 11 a.m. on 2 occasions. In 1 condition subjects were deprived of SWS during the first 3 h of recovery sleep. In the other condition recovery sleep was not experimentally disturbed. During undisturbed recovery sleep, power densities in the delta and theta frequencies were higher than during baseline sleep. During SWS deprivation, power densities in this frequency range were lower than during undisturbed recovery sleep. In the hour following SWS deprivation, power densities were enhanced relative to the same interval of undisturbed recovery sleep. Again, SWS deprivation did not cause an increase of sleep duration. The observed changes in EEG power density support the hypothesis that this EEG parameter reflects the homeostatic process S. The absence of an increase in sleep duration after SWS deprivation, however, contradicts the hypothesized causal role of this process in the regulation of sleep length.

Key words: Spectral analysis; Human; Sleep; SWS deprivation; EEG; Two-process model

There is considerable evidence for the involvement of clock-like mechanisms in the regulation of human sleep duration. Under normal conditions (Åkerstedt and Gillberg 1981) and in the absence of Zeitgebers (Czeisler et al. 1980; Zulley et al. 1981), sleep duration is dependent on the circadian phase of sleep onset. Shifting the circadian pacemaker, by repetitive exposure to bright light in the early morning, resulted in an advance of the rise in plasma melatonin in the evening, an advance of the rise of body temperature during sleep and an

advance of sleep termination (Dijk et al. 1987a, 1989). All these data underscore the circadian influence on sleep duration. Evidence for homeostatic involvement in the regulation of sleep duration has been scarce. In his analysis of sleep timing in internally desynchronized subjects, Strogatz (1986) failed to find any contribution of the variation in the duration of activity to the variation in subsequent sleep duration after correction for the circadian influence (see, however, Beersma 1987). Under normal laboratory conditions it has long been known that loss of 1 night of sleep lengthens the subsequent night sleep by only approximately 10–20% (Patrick and Gilbert 1896; Webb and Agnew 1975; Benoit et al. 1980). This lack of a clear homeostatic component in the regulation of sleep duration may be explained by assuming that NREM sleep has an intensity di-

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mension. Evidence for variations in the intensity of sleep has been derived from different areas. In the first place, the arousal threshold during sleep might be an indicator of sleep intensity. Arousal threshold studies revealed that during the nocturnal sleep period arousal thresholds become progressively lower (Loomis et al. 1937). As shown by EEG studies, the modulation of the arousal threshold is paralleled by the modulation of NREM sleep: arousal thresholds for acoustic stimuli are highest during stages 3 and 4 (slow wave sleep, SWS) and lowest during stage 2. They are, furthermore, enhanced after sleep deprivation (Williams et al. 1964). SWS diminishes over the sleep period for at least the first 11 h of sleep, and after sleep deprivation SWS is enhanced. The relation between the duration of prior wakefulness and SWS has been extensively described (Webb and Agnew 1971; Knowles et al. 1986). Computer assisted analysis of the changes within NREM sleep has resulted in a quantitative description of the relation between the duration of activity and the intensity of NREM sleep (Dijk et al. 1987c). Spectral analysis of the sleep EEG further revealed that the changes within NREM sleep are not limited to the delta frequencies but extend up to 7 or 8 Hz (Borbély et al. 1981; Dijk et al. 1987c).

The 2-process model of sleep regulation (Borbély 1982; Daan et al. 1984) attributes a role in sleep regulation to both a circadian and a homeostatic component. The homeostatic component is represented by process S. S increases during wakefulness in a saturating exponential way and decreases exponentially during sleep. Sleep is initiated and terminated when S reaches an upper threshold and a lower threshold respectively. The circadian component in the regulation of sleep duration is represented by a circadian variation in these two thresholds. The intensity aspect is accounted for by the assumption that S not only represents sleep debt but, during undisturbed sleep, also sleep intensity. During undisturbed sleep process S is thought to be reflected in EEG power density. One of the predictions of this model is that a positive correlation between sleep debt at sleep onset and sleep duration should emerge when the circadian phase of sleep onset is

kept constant, even though part of the sleep debt is compensated by changes in sleep intensity. Åkerstedt and Gillberg (1986a,b) could verify this prediction in an experiment in which sleep debt at sleep onset was manipulated by varying the duration of nocturnal sleep preceding the daytime recovery sleep period. They also showed that as the length of the nocturnal sleep period preceding recovery sleep increased, EEG power at the beginning of recovery sleep decreased. This latter finding indicates that NREM sleep intensity is a function of the prior history of sleeping and waking. In terms of the 2-process model of sleep regulation, these findings indeed indicate that at a particular circadian phase the level of S at sleep onset is positively correlated with subsequent sleep duration. A second prediction of the model is that despite equal levels of S at sleep onset, sleep duration should increase if the rate of decay of S during sleep is slowed down. The rate of decay of S can be thought of as the intensity of NREM sleep and is proportional to EEG power. This was shown in an experiment in which subjects were deprived of SWS during the first 3 h of their nocturnal sleep period without inducing wakefulness (Dijk et al. 1987b). In the undisturbed part of the night, an enhancement of SWS and EEG power density relative to the same interval of the baseline night was observed, indicating that S was higher in the second part of the experimental night as compared to the same interval of the baseline night. Since it can be assumed that the levels of S were identical at the beginning of the baseline night and the experimental night, it must be concluded that during SWS deprivation the decay of S is slowed down. Under the assumption that during SWS deprivation EEG power density is proportional to the rate of decay of S, this rebound could be accurately predicted from the reduction of EEG power during SWS deprivation. The experimental conditions did not allow us to assess spontaneous sleep duration. The purpose of the two experiments presented here is to investigate further the effects of SWS deprivation in the first part of the sleep period on EEG power density during the second part and to assess the effects of SWS deprivation on the spontaneous termination of sleep.

Methods

EEG recording and analysis

EMG, EOG and EEG were recorded on paper with a paper speed of 10 mm/sec and scored according to the criteria of Rechtschaffen and Kales (1968). EEGs were derived from C4-A1 and C3-A2 and low pass filtered at 25 Hz (24 dB/oct). The time constant of the preamplifier was 1 sec. All signals were digitized with a sampling rate of 64 Hz and stored on magnetic tape. Both EEG signals were subjected to spectral analysis with a fast Fourier routine on a PDP11/34 computer. Power densities per 4 sec epoch from 0.25 to 15.0 Hz were collapsed into 1 Hz bins by adding adjacent 0.25 Hz bins. Visual scores (30 sec epochs) were also fed into the computer and synchronized with the power data. This allowed us to calculate power densities per sleep stage and to remove epochs of stage 0 and MT (movement time). Brief disruptions of EEG signals due to short-lasting EMG arousals were removed on the basis of the value of the rectified EMG. For all computations the better of the two EEG leads was used. Body temperature was recorded with a rectal probe (Vita-log). The sampling rate was 0.25 min (data not shown). After each sleep period subjects filled out the Groningen Sleep Quality Scale (Mulder-Hajonides van der Meulen and Van den Hoofd-akker 1984).

Experiment 1

Eight healthy adult male subjects (mean age 23.1 years, range: 21–26) participated in a balanced cross-over design. They were free of sleep complaints and had regular sleep habits. After an adaptation night (00.00–08.00 h) sleep was recorded. Lights went off at midnight. The subjects were instructed not to rise before they felt that they had completed their normal sleep quota. Before the sleep recording they were told that their sleep would be disturbed by acoustic stimuli. Two weeks later the subjects came to the laboratory again and the entire procedure (including the adaptation night) was repeated. Again they were told that their sleep would be disturbed. The subjects were, however, deprived of SWS during 1 of the 2 sleep recording nights. The night in which

the deprivation took place will be called the 'experimental night,' and the other the 'baseline night.' The order of SWS deprivation was balanced. The deprivation was achieved by acoustic stimuli which were administered through an intercom. Whenever a subject entered stage 3, clicks of which the loudness could be varied were administered. Care was taken not to induce wakefulness. The SWS deprivation lasted for the first 5 h after sleep onset, to allow spontaneous termination of sleep during the undisturbed remaining part of the night.

Experiment 2

Subjects were 8 healthy male subjects (mean age 24.5 years; range 23–26). They were free of sleep complaints and had regular sleep habits. The experiment consisted of 2 sessions with an interval of 2 weeks. After an adaptation night, baseline sleep from 00.00 to 08.00 h was recorded. The following evening the subjects came to the laboratory and were kept awake until 11.00 the next morning when recovery sleep was allowed. During the sleep deprivation night subjects were sitting in a room together with the experimenter. EEG was recorded throughout the night. At the beginning of the daytime recovery sleep, subjects were instructed not to rise before they felt wide awake and were told that their sleep would be disturbed by acoustic stimuli. Two weeks later the subjects returned to the laboratory and the entire procedure was repeated, including the adaptation and baseline night. Before the recovery sleep the subjects were told again that their sleep would be disturbed. As in experiment 1, only 1 of the 2 recovery sleep periods was disturbed. In 4 subjects the first recovery sleep was the sleep period in which they were deprived of SWS, whereas in the remaining 4 subjects the second recovery sleep was disturbed. Acoustic stimuli were administered during the first 3 h of recovery sleep.

Results

Experiment 1

Sleep stages. For the 2 nights time spent in the various sleep stages was calculated for 2 inter-

TABLE I

Sleep stages (min) during 2 intervals of the baseline night and experimental night of experiment 1. $n = 8$. Values between brackets are S.E.M.s.

Stage	Interval			
	0–5 h		5–6 h	
	Baseline	Experimental	Baseline	Experimental
0	8.6 (4.5)	13.2 (7.8)	0.5 (0.5)	0.0 (0.0)
1	9.0 (1.8)	11.1 (3.1)	2.5 (1.2)	0.5 (0.2)
2	140.7 (8.1)	173.4 (5.4) *	39.2 (4.5)	19.0 (2.5) *
3	26.3 (3.8)	35.7 (4.6)	1.4 (1.3)	4.9 (1.7)
4	54.0 (6.2)	13.0 (5.2) *	0.6 (0.3)	12.0 (4.7) *
MT	11.9 (1.4)	10.9 (0.8)	3.1 (0.5)	2.5 (0.7)
REM	49.6 (7.0)	42.7 (4.5)	12.7 (4.3)	21.1 (6.7)

* $P \leq 0.05$ Wilcoxon matched pairs signed ranks test. Baseline vs. experimental. MT = movement time.

vals. The first covered the first 5 h after sleep onset (the deprivation interval), whereas the second started 5 h after sleep onset and ended 1 h later (recovery interval). The reason for this short duration of the second interval is that the longest common sleep duration was 6 h. Compared to the first 5 h interval of the baseline night, no significant enhancement of stages 0, 1 and movement time was observed during the deprivation interval (Table I). Stage 4 was virtually absent during the deprivation, but stage 3 was hardly affected by the deprivation procedure. The reduction in SWS resulted in an increase of stage 2. Averaged over the entire interval, the amount of REM sleep was not affected although, in the first 3 h of sleep, more REM sleep was produced in the experimental night. Between the 2 nights no significant differences in REM latency were present (117.8 ± 17.8 (S.E.) and 91.7 ± 12.2 min for the baseline and experimental nights respectively).

During the recovery interval of the experimental night significantly more stage 4 was present than in the comparable interval of the baseline night. Stage 4 was increased at the expense of stage 2. Time spent in stages 0, 1 and MT was also slightly reduced. For this interval no significant differences in REM sleep were observed. Sleep termination was operationally defined as the first 15 min interval in which no epoch of stages 2, 3, 4 or REM sleep was present. By this definition the

resulting sleep durations were 451.8 ± 22.0 (S.E.) and 466.1 ± 17.9 min for the baseline and experimental night respectively (for the individual values see Table III). These values were not significantly different from each other.

Power density. In Fig. 1 power densities during the first 5 h of the experimental night are depicted as percentages of power density during the first 5 h of the baseline night. Power densities were calculated for stages 1, 2, 3, 4 and REM sleep combined and for NREM sleep (stages 2, 3 and 4) and REM sleep separately. From the 8 subjects, 1 subject was excluded from this analysis because the quality of the EEG signal did not allow reliable calculation of spectral power densities. During the SWS deprivation power densities during NREM sleep were attenuated from 0.25 to 7 Hz although only in the 2 (= 1.25–2.0 Hz) and 3 Hz bins this reduction was statistically significant. During REM sleep no significant changes in power densities were observed. Averaged over all sleep stages there was also a significant reduction in power density in the 2 and 3 Hz bins. In the first hour after termination of the SWS deprivation, power densities during NREM sleep were signifi-

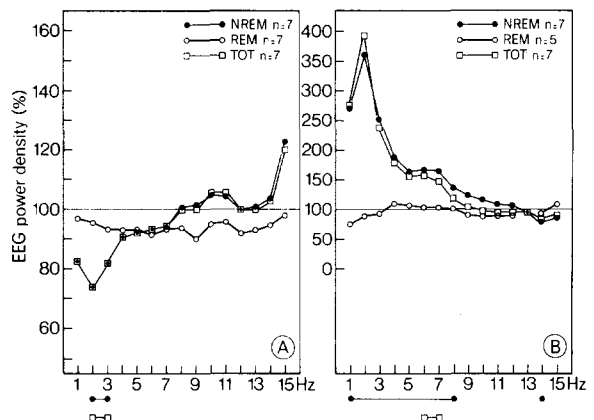


Fig. 1. A: EEG power density during SWS deprivation (0–5 h) expressed as percentage of EEG power density during the same interval of the baseline night. B: EEG power density during the first hour after SWS deprivation (interval: 5–6 h) expressed as percentage of EEG power density during the same interval of the baseline night. TOT = stage 1 + 2 + 3 + 4 + REM. Values are plotted at the upper boundary of the frequency bins. Horizontal bars indicate significant differences between the two conditions. Wilcoxon matched pairs signed rank test ($P < 0.05$).

cantly enhanced compared to the same interval of the baseline night. These changes were, however, limited to the delta and theta frequencies and the largest increase was found in the 2 Hz bin. During REM sleep no significant changes were observed. Calculated over all sleep stages the observed changes were similar to the changes in NREM sleep, albeit only significant in the 6 and 7 Hz bins.

For an estimation of the remaining deficit after 6 h, the total EEG energy (called total power by others; cf., Åkerstedt and Gillberg 1986b) was calculated by multiplying the power densities in the delta and theta frequencies in all sleep stages by time. Compared to the energy accumulated during the first 6 h of the baseline night (= 100%), the largest remaining deficit was located in the 2 Hz bin. During the first 6 h of the experimental night energy accumulated in this bin amounted to 88.7 ± 12.7 (S.D.) % of the energy accumulated during the same interval of the baseline night. To investigate whether this deficit was related to sleep duration, the rank correlation between this deficit and the difference in sleep duration between the baseline and the experimental nights was calculated. This correlation was not significant ($r_s = 0.142$, $n = 7$). Finally, EEG energy (0.25–8.0 Hz) was accumulated from sleep onset to sleep end for the baseline and experimental nights. Expressed as percentage of energy accumulated during baseline sleep, the subjects woke up in the experimental

night at an accumulated EEG energy value of, on average, 101.1 ± 25.1 (S.D.) %.

Experiment 2

Sleep stages. For a description of the effects of sleep deprivation on sleep stages, comparisons were made between the first 3 h of baseline sleep (averaged over the 2 baseline nights) and the first 3 h of the undisturbed recovery sleep (Table II). The effects of SWS deprivation were evaluated by comparing the first 3 h of the undisturbed and the disturbed daytime recovery sleep.

In the 6 subjects who slept for at least 1 h after termination of the SWS deprivation, a comparison was made between this 1 h interval and the comparable interval of the undisturbed recovery sleep. Sleep deprivation resulted in a significant reduction of latency to stage 2. REM sleep latency was not significantly affected by the sleep deprivation. Time in stages 3 and 4 was enhanced although the difference was only significant for stage 4.

SWS deprivation by the acoustic stimuli resulted in a significant reduction of stage 4. Stage 2 was enhanced as was stage 0. REM latency was not significantly altered by the SWS deprivation, although the variation was much larger than during the undisturbed recovery sleep. Time in REM sleep was not significantly changed. Two subjects woke up at the end of or immediately after the 3 h SWS deprivation interval. As a result, sleep duration in the SWS deprivation condition was on

TABLE II

Sleep stages in minutes during the first 3 h of baseline sleep (average of 2 nights) and during 2 intervals of recovery sleep starting at 11 a.m. For the first 3 h of baseline and recovery sleep $n = 8$. For the second interval of recovery sleep $n = 6$. Values between brackets are S.E.M.s.

Stage	Interval				
	0–3 h			3–4 h	
	Baseline	Undisturbed recovery	Disturbed recovery	Undisturbed recovery	Disturbed recovery
0	2.2 (1.1)	0.6 (0.4)	11.8 (5.9) *	0.4 (0.3)	0.2 (0.2)
1	6.3 (0.9)	5.0 (0.9)	15.1 (4.2)	1.5 (0.4)	0.7 (0.3)
2	80.2 (5.8)	71.9 (12.3)	93.0 (9.4)	39.3 (3.0)	17.9 (3.0) *
3	23.6 (2.1)	15.7 (2.0)	20.4 (4.6)	4.4 (4.6)	9.3 (2.0)
4	48.5 (3.8)	60.1 (4.5) *	18.9 (8.2) *	2.5 (0.9)	25.2 (2.9) *
MT	2.8 (0.4)	2.9 (0.4)	3.4 (0.9)	1.3 (0.2)	1.1 (0.2)
REM	17.9 (2.6)	23.9 (3.7)	17.2 (5.4)	10.6 (2.2)	5.7 (2.4)

* $P < 0.05$ WMP (variable vs. variable in preceding column).

TABLE III

Individual sleep durations in experiments 1 and 2. Values are in minutes. No subjects participated in both experiments.

Subject	Experiment 1		Experiment 2	
	Baseline night	Experimental night	Undisturbed recovery sleep	Disturbed recovery sleep
1	460.0	408.0	313.5	317.5
2	414.0	485.0	253.5	344.0
3	380.0	489.0	284.0	—
4	472.5	563.5	375.0	338.0
5	493.0	448.5	537.5	368.5
6	472.0	456.5	608.5	532.5
7	366.5	405.0	390.0	—
8	556.0	473.5	565.0	475.0

average somewhat reduced. Exclusion of these 2 subjects did not, however, result in a significantly longer sleep duration in the SWS deprivation condition (442.2 ± 60.1 (S.E.) and 395.9 ± 35.5 min for the undisturbed and disturbed conditions respectively; see Table III for the individual values).

In the 6 subjects who slept for at least 1 h after termination of the SWS deprivation, a significant increase in stage 4 was present compared to the same interval of the undisturbed recovery sleep. This increase was at the expense of stage 2.

Power density. Power densities during the first 3 h of undisturbed daytime recovery sleep were expressed as percentage of power densities during the first 3 h of the 2 baseline nights (Fig. 2A). During NREM sleep, power densities were significantly enhanced from 2 to 7 Hz and in the 15 Hz bin. The largest increase was observed in the 2 Hz band. In the spindle frequencies (12.25–14.0 Hz), power densities were attenuated during recovery sleep. During REM sleep, power densities in the delta and theta frequencies were also increased by the sleep deprivation, although only the change in the 3 Hz bin was statistically significant. When all sleep stages were taken together power densities were enhanced in the delta and theta frequencies, whereas in the spindle frequencies an attenuation was observed.

As a consequence of SWS deprivation power densities in the delta frequencies were attenuated

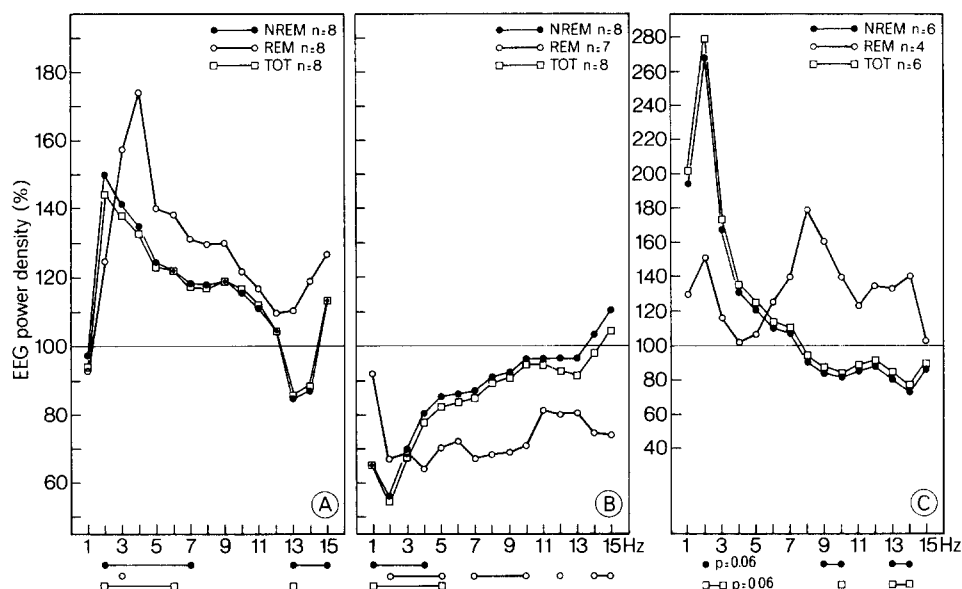


Fig. 2. A: EEG power density during recovery sleep (0–3 h) expressed as percentage of EEG power density during the same interval of the baseline night. B: EEG power density during SWS deprivation (0–3 h) expressed as percentage of EEG power density during the same interval of undisturbed recovery sleep. C: EEG power density during the first hour after SWS deprivation (3–4 h) expressed as percentage of EEG power density during the same interval of the undisturbed recovery sleep. For further details see Fig. 1.

during NREM sleep (Fig. 2B). The largest reduction was present in the 2 Hz bin. In the other frequencies no significant changes were present. Interestingly, during REM sleep power densities were also reduced by the SWS deprivation. Although the largest effects were observed in the delta frequencies, significant reductions were also present in the other frequency bins. When all sleep stages were taken together, power densities in the delta and theta frequencies were reduced. In the higher frequencies no significant changes were observed. After termination of the SWS deprivation, power densities during NREM sleep were higher in the delta and theta frequencies than those measured in the same interval of the undisturbed recovery sleep (Fig. 2C). Reductions were observed in power densities from 9 to 14 Hz. During REM sleep power densities in theta frequencies were also somewhat enhanced, although not significantly. When all sleep stages were taken together a similar picture emerged.

For the 6 subjects who still slept at the end of the SWS deprivation, EEG energy from 0.25 to 8.0 Hz was accumulated from sleep onset till the end of sleep for both the undisturbed and the disturbed recovery sleep. In the disturbed condition all subjects woke up before they had accumulated the amount of EEG energy produced in the undisturbed condition. The average amount produced, expressed as percentage of EEG energy produced during the undisturbed condition, was 78.9 ± 8.0 (S.D.) %.

Discussion

SWS deprivation during the first 5 h of night sleep (experiment 1) resulted in a reduction of stage 4. SWS deprivation was, however, not complete. Despite the modest SWS deficit a significant compensatory response was observed at the end of this nocturnal sleep period. This finding is in accordance with a previous experiment in which subjects were deprived of SWS during the first 3 h of a nocturnal sleep episode (Dijk et al. 1987b). As in the former experiment also in the present study, during SWS deprivation power densities during NREM sleep were reduced in the delta frequen-

cies while a significant enhancement in the delta and theta frequencies was present after termination of SWS deprivation. The data from the visual scoring and the spectral analysis show that the time course of SWS and delta and theta power is not determined by the time passed since sleep onset but depends on the events within NREM sleep. Even at the end of a nocturnal sleep period where REM sleep pressure is thought to be very high, a deficit in NREM energy can be compensated for. As a consequence of the compensatory response the energy deficit at the end of the sixth hour of the experimental night was very small. In terms of the 2-process model of sleep regulation, this means that the level of S at the end of the sixth hour was not much different for the two conditions. It is, therefore, not surprising that SWS deprivation during the first 5 h did not result in an increase in sleep duration.

The first 3 h of recovery sleep starting at 11 a.m., after 1 night of sleep deprivation, were characterized by a significant enhancement of power density during NREM sleep in the delta and theta frequencies and an enhancement of stage 4. The largest increase was observed in the 2 Hz bin. REM sleep power densities were also higher than during the first 3 h of baseline night sleep. These findings correspond with those from a sleep deprivation experiment by Borbély et al. (1981), in which recovery sleep started at 11 p.m. They also reported significant enhancements of power density in the delta and theta frequencies during NREM sleep. Furthermore, in their data also the effects of sleep deprivation on power densities were not limited to NREM sleep, but slow wave activity was increased also in REM sleep.

From a nap study (Dijk et al. 1987c) we concluded that power densities of the delta and theta frequencies during the first 30 min of sleep are a monotonic rising function of the duration of prior wakefulness, independent of the circadian phase at which sleep is taken. In this nap experiment the range in the duration of prior wakefulness was 2–20 h. The results of the present experiment, in which the duration of prior wakefulness was 27 h and sleep was initiated at 11 a.m., confirm this conclusion. The reduction of power densities in the 13 and 14 Hz bins was not observed in the nap

study. There we found a reduction in the 15 Hz bin, as reported by Borbély et al. (1981) in their sleep deprivation experiment. This discrepancy may not be incidental. In another sleep deprivation experiment (Dijk et al. in prep.), in which recovery sleep started at 11 a.m., we also found a significant reduction in the 14 Hz bin and no significant attenuation in the 15 Hz bin. At present we do not have an explanation for these findings, although the reductions of power densities in these higher frequencies may be related to changes in the occurrence and/or amplitude of sleep spindles.

To deprive subjects of SWS during recovery sleep from total sleep deprivation turned out to be difficult. The amount of SWS was, however, significantly reduced. Again this deficit was partially compensated for by an increase in the amount of stage 4 in the first hour after termination of the SWS deprivation. The effects of SWS deprivation on power densities during NREM sleep were qualitatively similar to the changes observed in the first experiment. The enhancement of power densities in the delta and theta frequencies during NREM sleep after SWS deprivation shows that also during recovery sleep after total sleep deprivation at this circadian phase the time course of delta and theta power is not solely dependent on the time passed since sleep onset.

In contrast to the findings in the first experiment, in the second we now found a significant effect of SWS deprivation on power densities during REM sleep. Over the entire frequency range analysed power densities during SWS deprivation were somewhat lower, especially in the delta frequencies. This finding is reminiscent of the reductions in power density from REM period 1 to 3 (Borbély et al. 1981). For an explanation of these phenomena, a better understanding of the interactions between slow wave activity during NREM sleep and slow wave activity during the subsequent REM period is needed.

The rather short duration of undisturbed day sleep after sleep deprivation underscores the circadian influence on sleep termination. Compared to the data of Åkerstedt and Gillberg (1981) which were used to derive the lower threshold, the present durations are somewhat longer, although

the interindividual variation is considerable.

The absence of an increase in sleep duration after the SWS deprivation in the daytime recovery sleep experiment cannot be explained in the same way as in the first experiment, by a small energy deficit at the end of the fourth hour. All 6 subjects woke up with a considerable energy deficit but they did not sleep longer than in the undisturbed condition. The 2-process model predicts that the level of S at sleep termination depends on the circadian phase. Nevertheless, if the decay of S is slowed down, sleep duration should increase irrespective of circadian phase.

There are several possible explanations for the discrepancy between the data and the predictions of the model. Firstly, SWS deprivation may not slow down the decay of the regulating variable S. This explanation is very unlikely in view of the similarities in the changes in the EEG induced by SWS deprivation and those observed over the sleep period (Borbély et al. 1981). The changes in the sleep EEG after termination of the SWS deprivation resemble the changes seen after sleep deprivation in substantial detail, indicating that the level of S at the end of the SWS deprivation interval is higher than at the end of the comparable interval of undisturbed sleep. One could argue that the changes in the sleep EEG are merely correlates of changes in S and that a regulatory process responsible for the time course of power density during sleep, which is normally correlated with changes in S, dissociates during SWS deprivation from the time course of S during SWS deprivation. This hypothesis cannot easily be tested experimentally and is therefore not very attractive. Alternatively, SWS deprivation may raise the lower threshold, either as a direct result of the acoustic stimuli or as a consequence of the reduced slow wave activity. Although this explanation is intuitively appealing, it cannot be investigated until a measurable correlate of the level of the wake up threshold has been identified.

In conclusion, the data presented underscore the homeostatic properties of the processes involved in the regulation of slow wave activity, but they do not support the hypothesis that a reduction of NREM sleep intensity increases sleep duration at all circadian phases.

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