Sleep Deprivation – 1

P225
EEG spectral power and cognitive performance during sleep inertia: the effect of normal sleep duration and partial sleep deprivation
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Sleep inertia (SI) is a transient period occurring immediately after awakening, usually characterized by performance decrement. When sleep is sufficient, SI is moderate, and produces few or no deficit. When it is associated with prior sleep deprivation, SI shows dose-dependent negative effects on cognitive performance, especially when subjects have been awaken in slow wave sleep (SWS). In the present study, spectral analysis was applied during the last 10 min before and the first 10 min after awakening, and during 1 h after awakening while subjects performed the Stroop test. Seventeen subjects were divided into a Control group who slept 8 h, and a Sleep Deprived group who slept only 2 h. The results show that, after awakening, performance was normal in the Control group, whereas reaction time was increased during the first half hour and error level during the second half hour in the Sleep Deprived group. Spectral analysis applied on the waking EEG during the whole test session showed that alpha activity was increased in both groups, but theta power only in the Sleep Deprived group. There was a high positive correlation in sleep deprived subjects between delta power during the last 10 min before awakening and subsequent performance decrement in speed and accuracy during the whole test session, but most of all in speed during the 10 min after awakening. Comparison of individual records showed a high positive correlation between spectral power before and after awakening in the Control group (EEG moving progressively to a high frequency pattern), but no correlation was found in the Sleep Deprived group who exhibited a rather discontinuous EEG pattern from before to after awakening. This suggests that after normal sleep duration, sleep offset could be characterized by a progressive shift in EEG activity, while after prior sleep loss, this process could be more erratic. We discuss these results in terms of incoherence in the EEG continuity during sleep offset after prior sleep loss, which could partly account for the performance decrement observed during SI in sleep deprived subjects.

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Sleep, social roles and relationships of health professionals who work at night
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Most existing research concerning night work addresses physiological aspects, and indicates that night work profoundly disrupts the circadian system and sleep. Many studies acknowledge social factors influencing sleep, and some studies indicate associations between shift work and couple relationship breakdown. However, little research explores the lived experiences of night workers and their families, or how physiological and social processes may interact. The paper first provides a conceptual exploration of the social roles and relationships which may affect and be affected by health professionals’ work at night, together with linkages between physiological, social and emotional dimensions of night work. Secondly, findings are presented from a small-scale qualitative study exploring perspectives of night working doctors, nurses and their spouses/partners. Findings suggest health professionals’ physiological tolerance of work at night may be influenced by a combination of mediating action, social norms and structural social influences. For example, female nurses’ daytime sleep may be disrupted by delivering and collecting children from school, and completing other household tasks. Further, it seems severe sleepiness, especially in combination with social isolation, impedes motivation to follow a healthy lifestyle, which may contribute in part to associations between night work and cardiovascular disease, digestive problems and some cancers. In conclusion, despite night work’s significant negative effects on health professionals’ physiological health and well-being, it seems social responsibilities and well-being at household and professional levels are regarded as more important than physiological considerations, and may impede acceptance of night work’s physiological consequences.
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Effects of age and oral contraceptive use on women’s performance in the psychomotor vigilance task during total sleep deprivation
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The objective of this study was to investigate the effects of age and oral contraceptive (OC) use on women’s performance in the psychomotor vigilance task (PVT) during a 40-hour total sleep deprivation (SD) period. A total of 46 healthy women volunteered. They belonged to two age groups: young (n = 34; age range 19–30 years; 12 without, and 22 with OC) and aged (n = 12; age range 60–68; postmenopausal, without hormone therapy). The subjects underwent a 40-hour total SD with ambulatory EEG recordings, and performed PVT and subjective sleepiness scales at 2-hour intervals. SD increased mean reaction time (RT), slowest 10% RTs, number of lapses (RT > 500 ms), and subjective sleepiness scores similarly in the two age groups, when baseline performance levels were taken into account (area under curve analyses, Mann-Whitney U-tests NS). The increase in the fastest 10% RTs was more pronounced in the aged, while the increase in the number of false starts was more pronounced in the young. The SD-related deterioration in PVT performance (measured as number of lapses) occurred earlier in the aged than in the young women (survival analysis). OC use had no effects on any of the measures during SD. After recovery sleep, the young had better mean RT and fastest 10% RTs, but they also had more false starts and higher subjective sleepiness scores than the aged. In conclusion, aging affects the time course of the decline in PVT performance caused by SD, but has no effect on its amount, if baseline levels are taken into account. Young women seem to maintain their fastest RT scores better than the aged, but at the expense of making more mistakes. OC use does not significantly affect young women’s PVT performance during SD.
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Recovery of vigilance and multitask performance from cumulative sleep deprivation
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The present study examined the recovery process of cognitive functions from a simulated working week with reduced sleep.

Methods: A total of 16 healthy men (aged 21–27 years) participated in an experimental laboratory study. Thirteen of them underwent a sleep debt condition including two baseline days (8 h in bed per night), five sleep deprivation days (4 h in bed), and two recovery days (8 h in bed). The rest three of the subjects were allowed to sleep for 8 h per night throughout the experiment. On each day, the subjects performed four multitask sessions (20 or 50 min in duration) and three PVT (Psychomotor Vigilance Task) sessions (10 min). The subjects trained the tests prior to the experiment to flatten the practice effect. Subjective sleepiness was measured with the Karolinska Sleepiness Scale.

Results: After the five sleep deprivation days, there was a 25% reduction in multitask performance and an increase in the mean number of PVT lapses (from 0.9 to 3.5) and subjective sleepiness (from 4.0 to 5.7) compared to the baseline days. On the first recovery day, the mean multitask score was only 5% below the level observed during the baseline days. Also the mean number of PVT lapses (1.2, SD = 1.8) and subjective sleepiness (4.2, SD = 1.1) returned very close to their baseline levels. On the second recovery day, both performance and subjective sleepiness (4.2, SD = 1.1) returned very close to their baseline levels for a 10 h baseline sleep period. SWA underwent homeostatic increases after 5 nights of 4 h sleep restriction, but not to levels equivalent to the SWA during a baseline 10 h TIB. Thus, chronic restriction of sleep to 4 h TIB evokes insufficient SWA activity to prevent a sustained SWA deficit.

Acknowledgement: This study was supported by NIH NR004281 and RR00040.

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Dynamics of slow wave activity after five nights of 4-hour sleep restriction
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Restriction of sleep to 4h per night results in cumulative neurobehavioural deficits but much smaller increments in NREM EEG slow wave activity (SWA)…—the putative marker of sleep homeostasis. We sought to obtain a precise estimate of the time course and magnitude of the SWA response during sleep restriction in a cohort larger than previous studies. n = 28 healthy subjects (32 ± 7 years, 15 females) underwent 2 nights of baseline (B) 10 h TIB, followed by 5 nights at 4 h TIB. EEG was recorded at baseline and restriction nights 1 and 5 (SR1, SR5), and analyzed for NREM SWA (1-4 Hz, sampled at 120 Hz) for the C3-Ax derivation. After artifact removal, FFT analysis was performed in 5 s bins, and the average amplitude for every 30 s epoch was computed. SWA was measured as a function of total TIB, the first 4 h of baseline, and for equivalent amounts of NREM in baseline and restricted sleep. Compared to B10 h TIB, SWA was reduced on SR1 by 45% (P < 0.001). On SR5 SWA was increased over SR1 by 49% (P = 0.004), but remained 23% below SWA levels at B10 h TIB (P = 0.003). SWA was comparable on SR1 to the first 4 h of TIB on baseline (P = 0.52), but significantly higher by 28% at SR5 than at B4 h TIB (P = 0.016). Analyses on the equivalent duration of NREM sleep at baseline (B NREM) showed that SWA was again comparable on SR1 (P = 0.29) and higher by 21% at SR5 (P = 0.019). Thus, at SR5, SWA was elevated over comparable periods of NREM at baseline and SR1, but it remained below levels for a 10 h baseline sleep period. SWA underwent homeostatic increases after 5 nights of 4 h sleep restriction, but not to levels equivalent to the SWA during a baseline 10 h TIB. Thus, chronic restriction of sleep to 4 h TIB evokes insufficient SWA activity to prevent a sustained SWA deficit.
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Total sleep deprivation and partial sleep deprivation in young women
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The aim of our study was to compare the effects of 40 h of Sleep Deprivation with the effects of three nights of Sleep Restriction with 4 h of sleep per night. 20 women aged 20–30 years, healthy and non-smoker were included in the study after a careful selection. 10 women participated to the ‘Restriction Study’ (RS) in which they were admitted to the Sleep Laboratory for one Baseline Night (BN)-(11pm–7am), three nights of Sleep Restriction (01am–05am) and one Recovery Night (RN). The other 10 were included in the ‘Total Deprivation Study’ (TDS) in which the subjects had to stay in laboratory for two BN, 40 h of Sleep Deprivation and one RN. Continuous EEG recordings were performed day and night in both studies. The Stanford Sleepiness Scale (SSS) and the Psychomotor Vigilance Task (PVT) were performed during Baseline, Sleep Deprivation or Restriction and Recovery at 9am, 1pm and 5pm. Cognitive tests (Stroop, Wisconsin and Trail Making Test) were performed once during Sleep Deprivation or Restriction and during Recovery. The scores at PVT and SSS showed a condition main effect in both studies, but also a Interaction Condition x Hours x Study (P < 0.05) which revealed that in ‘TDS’ the time (PVT) and the scores at SSS more significantly increased in the afternoon after 30 h of Sleep Deprivation, whereas in ‘RS’ deterioration is strongest the morning at 9 am after the third night of Restriction. Recovery Night improved the scores in the ‘RS’, while in ‘TDS’ the scores at SSS were worse after RN than during Sleep Deprivation (Interaction Condition x Study: P < 0.005). For the Stroop and the Wisconsin, a Condition x Study Interaction (0.000 and 0.009) showed that in ‘RS’ time and errors were outside the norms after the third night of Restriction and improved significantly after RN while in ‘TDS’ the scores were in normal range during Total Deprivation but significantly increased after RN. Our data suggests that among young women, response to the two form of Sleep Deprivation differs, particularly concerning the recovery process.

Acknowledgement: This research was supported by the EU grant (QLK6-CT-2000-00499).

Keywords: Sleep deprivation, women, sleepiness

P232
Chronic sleep restriction alters the relationship between subjective daytime sleepiness and hypothalamic-pituitary-adrenal (HPA) axis
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Introduction: This study was conducted to investigate the effects of six days sleep restriction on daytime sleepiness and Hypothalamic-Pituitary-Adrenal (HPA) Axis.

Methods: Nine healthy male (age 23.4 ± 3.1 years) were enrolled in the study. Subjects spent 8 hours sleep (LS) or 5 hours sleep (SS) for 5 days the day before polysomnography and Multiple Sleep Latency Test (MSLT) were performed in the following day. Daytime sleepiness was evaluated using mean sleep latency (MSL) of MSLT and Epworth sleepiness scale. The combined Dexamethasone (DEX)/Corticotropin-releasing hormone (CRH) test was performed. Subjects received an oral dose of 1mg DEX at 23:00 h on the sixth day. At 15:00 h the following day 100 μg of human CRH was injected intravenously and blood specimens were drawn until 17:00 h. Plasma levels of ACTH and cortisol were measured and area under the concentration curve (AUC) values of ACTH and cortisol was calculated.

Results: MSL were shortened (8.0 ± 1.4 to 5.2 ± 1.6 m, P = 0.008), and ESS were increased (4.7 ± 0.7 to 10.4 ± 0.8, P = 0.007) in SS as compared with those of LS. MSL of LS were negatively correlated with ESS (Spearman’s ρ = -0.710, P = 0.032), but not in SS. AUC values of ACTH and cortisol did not differ between LS and SS. In LS condition, AUC values of ACTH and ESS showed significant positive correlation (ρ = 0.681, P = 0.043), however, those in SS condition showed significant negative correlation (ρ = -0.695, P = 0.038). Those correlations were not observed between AUC values of ACTH and MSL of MSLT.

Conclusion: This study suggests that HPA axis is suppressed as subjective daytime sleepiness is increased in sleep restricted condition continued for about one week.

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Sleep deprivation reduces hippocampal expression of the transcription regulatory protein zif-268
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One theory about the function of sleep is that it plays an important role in neuronal and synaptic plasticity necessary for memory formation. In line with this hypothesis, the expression of various genes appear to change with behavioral state, including the immediate early gene zif-268 (also known as Egr-1, Krox-24, NGFI-A). The zif-268 gene encodes for a transcription factor protein that controls various other genes with important roles in neuronal function and synaptic plasticity. Furthermore, studies with knockout mice have shown that deletion of zif-268 impairs learning and memory in a variety of tasks. In the present study, we examined the daily profile and effect of sleep deprivation on zif-268 protein expression in the hippocampus and other brain regions involved in learning and memory. The experiments were performed with adult male C57BL/6J mice. In one group of animals brains were collected at 3 h intervals throughout the 12 h light /12 h dark cycle. In a second series of animals, brains were collected after 12 h of sleep deprivation by gentle handling during the light phase (the circadian resting phase). The brains were processed for immunocytochemistry with a polyclonal antibody against zif-268. The data show that the 24 h pattern of zif expression in the dentate gyrus of the hippocampus parallels the pattern of sleep, with higher numbers of zif-268 positive cells during the light phase than during the dark phase. Sleep deprivation during the light phase caused a significant reduction in the number of zif-positive cells in various hippocampal regions, including the dentate gyrus. The results suggest that sleep promotes the hippocampal expression of the transcription regulator zif-268. A decrease in the expression of zif-268 due to sleep loss may eventually result in reduced neuronal plasticity and cognitive function.

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Chronic partial sleep deprivation gradually desensitizes the serotonin-1A receptor system  
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Chronic sleep loss is a rapidly increasing problem in modern society. Several studies have shown that shortage and disruption of sleep is linked to poor mood and psychopathology. One possible explanation is that chronic sleep restriction sensitizes individuals to depression by gradually altering serotonin signaling in the brain. The aim of the present study was: (i) to examine if chronic partial sleep deprivation gradually desensitizes the serotonin-1A receptor system; and (ii) to establish the role of stress and adrenal hormones in this process of desensitization. Our model of chronic sleep loss consisted of keeping rats awake for 20 h each day by forced locomotion in slowly rotating drums. The remainder of the time rats were allowed to sleep. In order to examine serotonin-1A receptor sensitivity, rats received injections of the agonist 8-OH-DPAT. The sensitivity of the receptors to 8-OH-DPAT was determined by measuring the acute hypotensive response (by means of radio telemetry) or the neuroendocrine response (by taking blood samples). To study the role of stress hormones we performed similar experiments after surgical removal of the adrenal glands, the main source of adrenaline and corticosterone. The sensitivity of the serotonin-1A receptor system was not affected by 2 days of restricted sleep, but it was significantly reduced after 8 days of sleep restriction, as indicated by attenuated temperature and ACTH responses to 8-OH-DPAT. This desensitization did not appear to be a consequence of forced locomotion since it did not occur in forced activity controls that walked a similar distance in half the time and had sufficient time to sleep. Moreover, the desensitization was not likely a consequence of stress or adrenal hormones since it persisted in sleep restricted animals even when their adrenals were removed. These data show that chronic partial sleep deprivation gradually alters serotonin-1A receptor signaling in a direction that is similar to what is seen in depression. Thus, our data support the hypothesis that chronically restricted sleep may gradually change the brain and sensitivity to disease.

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Chronic partial sleep deprivation alters amygdala function and stress reactivity  
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Frequently disrupted and restricted sleep is a rapidly growing problem in our modern society. Chronic sleep loss may have adverse effects on brain function and may increase the sensitivity to stress-related diseases and mood disorders, perhaps by gradually altering serotonergic neurotransmission. In the present study, we examined whether chronic partial sleep deprivation alters the serotonin-1A receptor signalling cascade in brain areas involved in stress and emotionality. Adult male Wistar rats were subjected to a schedule of partial sleep deprivation that consisted of 20 h sleep deprivation and 4 h of rest per day. Sleep deprivation was achieved by placing the animals in slowly rotating wheels. After 2 or 8 days of sleep restriction, brain material was collected and processed for autoradiographic analysis of serotonin-1A receptors and 1A receptor-associated Gi-proteins. In a second experiment we examined the effects of chronic partial sleep deprivation on neuroendocrine stress reactivity and behavioural fear response. In the brain areas that were examined, 2 and 8 days of sleep restriction did not lead to changes in serotonin-1A receptor numbers. However, 8 days of sleep restriction significantly increased serotonin-1A receptor-associated Gi-protein density in the amygdala. In agreement with this, sleep restricted animals showed altered neuroendocrine and behavioural response to stress. Thus, whereas restricted sleep for one or two days may not have noticeable effects, chronic partial sleep deprivation for over a week gradually alters serotonin-1A receptor-mediated signalling cascades in the amygdala, which in turn may affect the regulation of emotionality and stress-reactivity.

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Five days with partial sleep deprivation and the effects on performance, sleepiness and effort  
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Introduction: The aim of the present study was to evaluate performance and subjective ratings during 5 days of partial sleep deprivation (PSD) and during recovery. A second aim was to describe the intra-individual correlation between performance and subjective ratings of sleepiness.  

Method: Nine subjects slept in the laboratory for 12 days, which included 5 days with PSD (03–07 h) and 4 recovery days (23–07 h).  

Results: RT (median) deteriorated across PSD days and the highest value was found the 5th PSD day (342 ± 41 ms, P < 0.05). The values for the second baseline day was 284 ± 19 ms, RT was also elevated for the first recovery day (319 ± 26 ms) but declined across recovery days. Lapses (≥500 ms) showed a similar pattern, but the highest number of lapses was observed on the 7th recovery day (8 versus 3 for the 1st baseline day). KSS showed an increase across PSD days, but the recovery was more immediate and no elevated levels were observed during the 7th recovery day. The mean KSS was 5.0 ± 0.2 for the 1st baseline day, 7.6 ± 0.2 for the 5th PSD day, and 5.5 ± 0.3 for the 1st recovery day. The rating ‘tried to do my best’ (0 not at all-6 almost all the time) showed a different pattern (P < 0.05). It decreased across days, and the lowest value was observed for the 2nd recovery day (3.1 ± 0.3 versus 4.2 ± 0.2 for the 1st baseline day). The intra-individual correlation (based on pooled data and with subjects as forced dummy variables) between median RT and KSS was 0.34 (P < 0.001).  

Conclusion: Reaction times and subjective sleepiness increased across days with PSD. The recovery of reaction time was, however, slower compared to subjective sleepiness, and lapses occurred frequently during the 7th recovery day. The decline in ‘tried to do my best’ across days might contribute to the elevated reaction time levels during the recovery days. The correlation between subjective sleepiness and reaction time was moderate.

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Sleep deprivation during the first week of pregnancy differently affects male and female offspring' spatial learning in Morris water maze  
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Introduction: It is known that prenatal stress can affect differently female and male rats, and that sleep pressure is strongly increased...
during pregnancy. To differentiate specific effects of sleep loss and stress responses, gentle and non-stressful sleep deprivation of pregnant rats was administered in order to know if may affect offspring spatial learning behaviour and if those effects would show gender differences.

Methods: Eight males and eight females Long-Evans rats were disposed in couples in eight different cages. Each day the vaginal smear was observed in a microscope. The first day in which sperm appeared was considered day zero of gestation. Rats of experimental group were gently deprived 3 h, during 3 consecutive days on the first week of pregnancy. Rats of control group were in the identical environmental conditions but their sleep was not disturbed. When offspring of experimental group (OEG) and control group (OCG) were 4 month old, training of Morris Water Maze task was started. 16 OEG and 16 OCG rats were used; 8 males and 8 females in each group. The task consisted of 2 days of habituation to the swimming pool, 11 days of entrainment to find the platform and the trial day test.

Results: (i) Escape trials: The average time (seconds) was analysed by a three-factor repeated measures two-way ANOVA procedure with OEG and OCG and the sex (males or females) as the ‘Between Subjects’ factor and Trial Day (1,2,.. 11) as the ‘Within Subjects’ factor. OEG rats have a greater escape latency when compared to OCG ($P < 0.05$). Also females have a greater latency when compared to males ($P < 0.01$). (ii) Test trial: The average time (in seconds) was analysed by a two-factor ANOVA procedure with the sex and OEG or OCG as factors. OEG females performed worse than the other 3 groups ($P < 0.02$).

Conclusions: Sleep deprivation of the mother during first week of pregnancy seems to affect equally both sexes offspring in this escape training task, but a sex-specific response to the pregnant mother’ sleep deprivation is observed in the spatial-learning test.

Keywords: Sleep deprivation

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Changes in daytime heart rate variability after cumulative partial sleep deprivation

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Cumulative partial sleep deprivation has been shown to influence the activity of the sympathetic nervous system. This study aims to specify which aspects in the frequency domain of heart rate variability (HRV), a marker of autonomic activity, are altered after partial sleep deprivation. After 2 nights of normal 8 h sleep, sleep of 11 young and healthy male subjects (mean age 23.4 years) was restricted to 4 h during 5 nights. Hereafter, 2 nights of 8 h recovery sleep were allowed. HRV measurements were obtained after the first 2 nights of normal sleep (baseline), after 5 nights of restricted sleep (sleep deprived), and after 2 night of recovery sleep (recovery). For HRV analysis, 5 min epochs were selected each 2 h (from 08.00 AM until 10.00 PM) during which the subjects were sitting and activity was as low as possible. Artefacts in the ECG were manually identified and removed. Periods of hemodynamically significant cardiac ectopy were excluded. Parametric spectrum (autoregressive model) was used to obtain absolute power and normalised power of both the high (0.15–0.4 Hz) and low (0.04–0.15 Hz) frequency band. Finally, the LF/HF power ratio was obtained. For each variable, a daytime plot was drawn for each of the 3 experimental days and a 3 (day) x 8 (time) repeated measurements analysis was used to reveal statistical differences. Although ANOVA revealed no significant effects based on the preliminary results of the 11 subjects, a trend was observed towards a lower HF power, both in absolute and normalised terms. Although HF power became higher again after recovery compared to the sleep deprived condition, a full return to baseline levels did not occur. As the LF power remained unaffected, a trend towards an increase in LF/HF power ratio was observed. The HF power component of HRV tends to decrease after partial cumulative sleep deprivation, whereas LF power remained unaffected. This indicates a decrease in parasympathetic activity and thereby an increased sympathetic dominance.

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Sleep deprivation and suppression of a prepotent response

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Introduction: Sleep deprivation effects on basic cognitive functions have been extensively studied, and described. However, its the impact on higher-order cognitive processes, usually termed ‘executive functions’, is less clear. Recently, Jennings et al (Psych. Sc. 2003, 14: 473–79) studying the effect of one night of sleep deprivation on supervisory attention, found no effects on the participants’ ability to inhibit a prepotent response within a ‘go-no go’ paradigm. One of the aims of the present study was to evaluate the impact of one night of sleep deprivation on response inhibition using a different paradigm: the stop signal paradigm (Logan et al, Psych. Rev. 1984, 91:295–327). It consists of two concurrent tasks: a go task (a choice reaction time task) and a stop task involving the presentation of a signal following the target, which instructs the participant to stop his ongoing response. The stop signal paradigm permits estimation of the latency of the inhibition process.

Method: Fifteen participants, university students, aged 19–24. They were underwent the Stop Response Task in two different conditions: Baseline (participants were tested at approximately 9 a.m. after a regular night of sleep at home); and Deprivation (participants were tested at the same time after one night of total sleep deprivation).

Results: Mean GO RT after sleep deprivation was significantly higher ($P < 0.01$) than in base-line condition, while accuracy was significantly higher ($P < 0.01$) in base-line than in deprivation condition. As far as response inhibition is concerned we found that, after sleep deprivation, mean Stop Signal RT (the estimate of the latency of the inhibition process) was significantly higher ($P = 0.01$) than in base-line condition, suggesting an impairment of the efficiency of the inhibition process following sleep loss.

Conclusions: Our results are in agreement with the hypothesis of an impairment of the frontal lobe functions when normal architecture of sleep is disrupted. The failure to replicate Jennings et al. (2003) could be due to the fact that the paradigm we used is more sensitive to subtle variations in the efficiency of the inhibition process.

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The inhibition of basal forebrain neurons during sleep deprivation

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Sleep deprivation (SD) evokes significant increase of the adenosine level in the basal forebrain (BF), which may be the cause for increased sleep propensity. It can be hypothesized that adenosine inhibits BF neurons responsible for the active state, but no experimental data have been available. We have studied unit discharge activity in the BF in

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7–9 months old rats during SD. 73 BF neurons were recorded extracellularly for the first time in freely moving rats during 1h of baseline (BL), 3 h of SD, and 1 h of recovery. Preliminary comparison of the average discharge activity level during BL and 3rd h of SD showed significant difference in more than 70% of cells. Almost half of all recorded neurons were inhibited. The rest increased the average discharge activity or had no significant changes. During 1 h recovery about 20% of neurons returned their discharge frequency to BL level. State-dependency of neurons was determined based on 20% difference in mean discharge rate during BL in active waking (AW) compared to slow-wave sleep (SWS) and in paradoxical sleep (PS) compared to SWS. 45 cells were active in AW and PS (AP-on), 9 were AW-on, 12 were PS-on, 6 neurons were state-independent, and one neuron was SWS-on. Preliminary analysis showed that most neurons in all state-dependency groups decreased their discharge rate during the 3rd h of SD compared to BL. The single SWS-on neuron was activated. The largest number of inhibited neurons was in the AW-on group, more than half of those decreased their activity. Thus, these results show for the first time a significant inhibition of BF unit activity during SD, involving especially AW-on neurons. These data are consistent with the notion that the increasing adenosine level in BF during SD inhibits neuronal activity. Moreover, inhibition of most BF neurons may explain the decrease in active vigilance and increased sleepiness during prolonged wakefulness.

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P241
Age-related changes in homeostatic sleep regulation after multiple naps
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The EEG delta response to high sleep pressure (40-h of sleep deprivation) is attenuated in frontal brain areas in older subjects (1). Here we investigated the EEG delta response to low sleep pressure attained by scheduling multiple naps across a 40-h constant posture protocol. After an 8-h baseline (BL) sleep episode at habitual bedtimes, 16 healthy young (20–31 years; 8F) and 16 healthy older volunteers (57–74 years; 8F) underwent 10 cycles of 150-min scheduled wakefulness and of 75-min scheduled sleep under constant posture conditions (in bed, light levels < 8 lux). An 8-h recovery (RC) sleep episode followed the 40-h nap paradigm. EEGs were continuously recorded throughout the entire study and subjected to spectral analysis. The total amount of sleep accumulated across the ten naps was similar for the young and the elderly (460.1 ± 18.9 versus 448.3 ± 22.1 min). EEG delta activity (0.5–1.25 Hz) decreased significantly in response to sleep satiation during the first NREM sleep episode, but did not significantly differ between age groups. However, the decrease in EEG delta activity lasted significantly longer in the young (across the first two NREM sleep episodes) than in the older participants (only the first NREM sleep episode; \( P < 0.05 \)). Therefore, the decay of EEG delta activity was shallower for the elderly. Estimation of its time constant was only possible for frontal brain regions such that older subjects had a slower decay rate during the RC night than the young (0.074 ± 0.02 versus 0.119 ± 0.024; mean per hour). The immediate homeostatic response of EEG delta activity to sleep satiation does not change with age. Our results suggest age-related changes in the time course of the dynamics in EEG delta activity rather than regional differences in response to low sleep pressure.

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Reference: