The influence of pre-sleep cognitive arousal on sleep onset processes

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ABSTRACT

Cognitive hyperarousal, resulting in enhanced cognitive activation, has been cited as an important contributor to the development and preservation of insomnia. To further understand this process, our study examined the effects of acutely-induced pre-sleep cognitive hyperarousal on sleep onset processes in healthy volunteers. Following an adaptation night, 15 subjects slept two nights in our sleep laboratory: one reference night and another one with cognitive arousal induction, in a counterbalanced order. In the cognitive arousal condition, subjects worked through half an hour of cognitive tasks without interference of an emotional component prior to retiring to bed. Objective sleep onset latency was significantly prolonged in the cognitive arousal condition compared to the reference condition. Significantly more high frequency activity was recorded during the first and second deep-sleep period. Moreover, differences in heart rate and proximal temperature during and after sleep onset were observed in the nights after the cognitive induction. Pre-sleep cognitive activation successfully induced a significant cognitive load and activation in our subjects to influence subsequent sleep (onset) processes.

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1. Introduction

Years of research have shown that sleep onset – the transition from the waking to the sleeping state – does not initiate sleep from 1 s upon the other, but rather is a process with several semi-independent, yet interactive changes (Ogilvie, 2001). It is a progressive process of decline in control over mental activity and gradual changes in perceived state of consciousness (Freedman and Sattler, 1982), accompanied by gradual decrease in arousal (De Gennaro et al., 2001; Espie, 2002). This decrease is evidenced by a depression in cortical activity (Davis et al., 1938), a slowing of the heart rate (Burgess et al., 1999; Jurysta et al., 2003; Okamoto-Mizuno et al., 2008), a decline in core body temperature (Kräuchi et al., 2000; Kräuchi and Wirz-Justice, 2001; Okamoto-Mizuno et al., 2008; van den Heuvel et al., 1998), an increase in distal and proximal skin temperature (Kräuchi et al., 1999, 2000) and many more behavioural, cortical and physiological changes (Ogilvie, 2001). However, these are processes observed during sleep onset in healthy sleepers (Monroe, 1967). Subjects with sleep-onset problems, in particular those with sleep-onset insomnia, may not experience this specific series of events, or exhibit alterations of these processes (Espie, 2002; Monroe, 1967).

1.1. Hyperarousal models of insomnia

Hyperarousal is defined as either an enhanced basal level of arousal or the inability to down-regulate an excess of arousal (Pigeon and Perlis, 2006). It can express itself as somatic, cortical and cognitive arousal (Perlis, 2001; Pigeon and Perlis, 2006). In most models of insomnia, hyperarousal has been cited as an important contributor to the development and preservation of insomnia (Perlis et al., 2005). Depending on the model, emphasis is either put on cognitive (Espie, 2007; Hall et al., 1996; Harvey, 2002; Riemann et al., 2010; Wicklow and Espie, 2000), physiological/somatic (Bonnet, 2009; Bonnet and Arand, 2010) or cortical hyperarousal (Perlis et al., 1997).

Within the cognitive model of insomnia, a more important role is assigned to cognitive (hyper)arousal in the development of insomnia (Espie, 2007; Harvey, 2002; Wicklow and Espie, 2000). Being hyperaroused, insomniacs find it difficult to fall asleep and become highly attentive to sleep-promoting and sleep-disturbing factors (Tang et al., 2007). Other authors put insomnia in a more behavioural context, with cognitive hyperarousal as a factor in the maintenance of insomnia (Bonnet, 2009; Bonnet and Arand, 2010; Spielman et al., 1987). Several researchers incorporate both the behavioural and cognitive points of view into their models (Morin, 1993; Perlis et al., 1997; Riemann et al., 2010). In those models, a role for cognitive hyperarousal is formulated both in the development and maintenance of insomnia. It is hypothesized that by means of classic conditioning (of both

cognitions and behaviours), the sleep environment becomes an arousing trigger (Morin, 1993; Perlis et al., 1997; Riemann et al., 2010). An over-activation of the sensory and information-processing systems results from increased arousal (Pribram and McGuinness, 1975), making it more difficult to fall asleep.

Falling asleep should be a rather automatic and unconscious process. Conscious attempts to fall asleep might even disturb the sleep onset process (Espie, 2007; Harvey, 2000). Sleep itself, and worries, are the focus of pre-sleep cognitive activity in insomnia patients. More than good sleepers, they complain of non-intentional pre-sleep cognitive activity (Harvey, 2000). Intrusive pre-sleep thoughts cause stress and are highly correlated with sleep onset problems (Hall et al., 1996; Wicklow and Espie, 2000). These thoughts are an important target for such therapeutic interventions as thought-stopping and articulatory suppression (Bootzin and Rider, 1997; Levey et al., 1991; Morin, 1993). They all focus on short-term memory and working memory. Although insomnia patients tend to blame their sleep problems more on intrusive cognitive-arousing thoughts than on physical arousal (Lichstein and Rosenthal, 1980), the disturbing role of changes in processes controlled by the autonomous nervous system – such as heart rate (Hall et al., 2004) and body temperature (Morris et al., 1990; van den Heuvel et al., 1998) – must be taken into account.

It should also be noted that, in the context of sleep, cognitive activation is relevant not only with regard to patient populations. For instance, research has shown that such pre-sleep activities as computer gaming, television watching (Dworak et al., 2007) and using the internet (Reynolds et al., 2010; Van den Bulck, 2004) significantly disturb the weekly sleep patterns of young adolescents with negative effects on their learning and memory performance (Dworak et al., 2007). Higuchi et al. (2005) studied young adults and obtained the same results, but also eliminated the possibility that these effects of pre-sleep activity might be due to the exposure to bright light emitted by monitors.

1.2. Cognitive hyperarousal and its effects

Earlier research has shown that sleep is sensitive to the level of arousal induced by specific pre-sleep activities. However, this depends on the characteristics of the pre-sleep activity (physical and/or cognitive activities) (Bonnet and Arand, 2001; De Bruin et al., 2002; Hauri, 1969; Tang and Harvey, 2004) and the time elapsed between the activity and bedtime (Baekeland and Lasky, 1966; Hauri, 1968). de Bruin et al. (2002) and Hauri (1968) observed that neither a six- nor an eight-hour sustained mental workload immediately before going to sleep, had any effect on subsequent sleep macrostructure. However in both studies participants went to sleep immediately after finishing the cognitive tasks. Hauri (1968) analysed only the first 3, 5 h of sleep. He discussed this as an important shortcoming, because by referring to Baekeland and Lasky (1966), Hauri (1968) pointed out that pre-sleep activities have rather a delayed than an immediate effect on sleep-parameters. In another report (Hauri, 1969), however, Hauri observed a significant delay of 6 min in sleep onset after studying, compared to after physical activity and relaxation.

Gross and Borkovec (1982) found a significant effect of cognitive intrusions on the sleep onset latency of good sleepers (differences between experimental and control groups ranging between 5 and 12 min). In line with the previous experiment, two studies have identified the effects of induced intrusive thoughts — one on subsequent sleep onset and continuity (Hall et al., 1996) and another one on daytime sleep onset, using the Multiple Sleep Latency Test procedure (De Valck et al., 2004). Ansfield et al. (1996) found that under high mental load (March music) the urgency to fall asleep increased sleep onset latency in normal subjects whilst under a low mental load (new age music) the urgency to fall asleep caused subjects to fall asleep sooner than without the urgency under the same mental load. Kobayashi et al. (1998) found that mental activity affected the timing of REM-periods later at night and Koulack et al. (1985) found that both easy and

difficult versions of intelligence tests increased subsequent sleep onset latency and negatively influenced REM density. Moreover, pre-sleep engagement in exciting computer games increased sleep onset latency (Dworak et al., 2007; Higuchi et al., 2005). Yet, these studies did not differentiate between the pure cognitive effects of inducing cognitive arousal and the possible consequences of arousing emotional factors (Vandekerckhove and Cluydts, 2010) in their induction — e.g. a financial reward (Hauri, 1968), the stress of a 15-minute evaluated speech in the morning (Hall et al., 1996), musical preference (Ansfield et al., 1996), a television interview (De Valck et al., 2004) or speech (Gross and Borkovec, 1982), driving a car for 600 km on the highway (Kobayashi et al., 1998), fear of failure (Koulack et al., 1985) and playing shooting (Higuchi et al., 2005) or race games (Dworak et al., 2007). Research has shown that cognitive tasks with no emotional load (i.e. where performance is independent of reward or punishment) performed once or repeatedly prior to sleep onset have a significant detrimental effect on cortical and physiological processes. The effect on cortical processes was indicated by decreased EEG delta power density during the first non-REM-sleep cycle (Takahashi and Arito, 1994), whilst the lack of effect on physiological processes was indicated by changes in arterial blood pressure and R–R intervals (Takahashi and Arito, 1996a, 1996b). However, these last two studies also involved sleep restriction procedures (subjects slept from 02:00–07:00) that could have influenced the results.

1.3. Study aims and hypotheses

Since most of the afore-mentioned studies did not differentiate well between cognitive and emotional arousal, the aim of this study was to use a subset of cognitive tasks: a Digit Span task, a Stroop task, a Recognition task and the Symbol Substitution task (Wechsler, 1997), (see Materials and methods). These are maximally exclusive of emotional components. Earlier studies have demonstrated the effects of these tasks on cognitive loading and cortical and physiological arousal (Baddeley, 2003; Fairclough and Houston, 2004; Kamarck et al., 1994; Larson et al., 1995; Manuck et al., 1992; Matthews et al., 2004; Pattyn et al., 2010).

Within the framework of the cognitive behavioural model, this study investigates the way in which a set of cognitive tasks known to load on working memory, and known to induce a physiological response, influences the sleep onset process in healthy sleepers. Sleep onset is hypothesised to be prolonged, and during sleep onset, heart rate is expected to be elevated and the increase in proximal temperature to be reduced. An increased presence of high frequency EEG-activity is expected during deep sleep.

2. Materials and methods

2.1. Participants

15 volunteers (7 men and 8 women), between the ages of 18 and 28 (mean = 22.07 years; SE = 0.81) participated in our study. Subjects were recruited among a student population unknown to the experimenter and unaware of the purpose of the study. All were healthy sleepers, non-regular smokers and non-abusers of alcohol or other substances that influence the central nervous system. Adherence to these standards was ensured using the Pittsburgh Sleep Quality Index (Buysse et al., 1989), the Insomnia Interview Schedule (Morin, 1993), and a general intake interview. All participants signed informed consent forms. Complete participation to the study was rewarded with €150 independent of any results or performance.

2.2. Design and procedure

Two weeks prior to the experiment and during the experimental week, participants had to fill out sleep diaries, to control for any

abnormalities in their sleep–wake patterns. Participants spent three nights in the laboratory. After a first habituation night, the two test conditions (cognitive arousal – COG – and reference – REF – night) were counterbalanced. Participants slept one night at home between each night in the laboratory. Table 1 shows an overview of the experimental procedure. The study protocol was approved by the Institutional Review Board.

2.2.1. Questionnaires

Sleep diaries inform us about time awake in bed and drug intake or alcohol or caffeine use in the late evening. In the morning, subjects evaluated sleep quality and how refreshed they felt on an eleven-point visual analogue scale (VAS) from zero ('extremely bad') to ten ('extremely good').

The Cognitive VAS-Scale (CS) assesses two cognitive complaints – 'problems concentrating' and 'memory deficits' – on a four point VAS-scale from 1 ('absolutely not') to 4 ('seriously') giving a total score of min 2 and max 8. The higher the score on the CS, the more problems were experienced.

The Emotional VAS-Scale (ES) assesses four emotional complaints – 'feeling of being tense', 'feeling of being gloomy', 'feeling of being active'and 'feeling of being relaxed' – on a four point VAS-scale from 1 ('absolutely not') to 4 ('seriously') giving a total score of min 4 and max 16. The higher the score on the ES, the more problems were experienced.

Karolinska Sleepiness Scale (KSS) (Åkerstedt and Gillberg, 1990) runs from 1 ('extremely alert') to 9 ('very sleepy, having trouble staying awake').

The 32-item version of the Profile of Mood States (POMS) (Dutch translation by Cluydts, 1979) is a four-point adjective scale (from 1 — 'totally disagree' to 4 — 'totally agree') that results in five subscales: Tension (6–24), depression (8–32), anger (7–28), fatigue (6–24) and vigor (5–20). Higher scores are indicative of a higher intensity of the construct experienced.

The 27 items of the Activation/Deactivation Adjective Checklist (AD-ACL) (Mackay et al., 1978) is a four point adjective checklist (from 1 — 'totally disagree' to 4 — 'totally agree') that results in two subscales: stress and arousal. Higher scores are indicative of a higher intensity of the construct experienced.

2.2.2. Cognitive arousal induction

In subsequent order, a digit span task, a Stroop task, a recognition task and a symbol substitution task (Wechsler, 1997) were administered to induce cognitive arousal. The first three tasks were administered via E-Prime (Psychology Software Tools, Inc), whilst the symbol substitution task was conducted with paper and pencil. No feedback was offered and neither reward nor punishment was connected to

Table 1
Overview of the experimental procedure.

Time (hours)	Activity
0700 PM	Arrival at laboratory
	Completion of CS, ES, KSS, POMS and AD-ACL*
	Application of electrodes
0925 PM	Experimental COG
	REF
1015 PM	Completion of CS, ES, KSS, POMS, AD-ACL*
	Free time
1100 PM	Bedtime
	Start of PSG* recording
0700 AM	Awakening
	Electrodes removed
0715 AM	Completion of CS, ES, KSS, POMS, AD-ACL*
0730 AM	Shower and breakfast
0800–0900 AM	Start normal daytime activities

*CS = Cognitive VAS-Scale, ES = Emotional VAS-Scale, KSS = Karolinska Sleepiness Scale, POMS = Profile of Mood State, AD-ACL = Activation/Deactivation Adjective Checklist, PSG = polysomnography.

participants' performance. Tasks were completed in isolation in participants' bedrooms and lasted for approximately 30 min.

In accordance with the Digit Span Forwards task of the Wechsler Adult Intelligence Scale 3rd Edition (WAIS III) (Wechsler, 1997), a series of digit strings, ranging from three to eight digits (five strings for each amount of digits) were presented. After hearing the digit string, subjects were asked to type the same sequence of digits on a keyboard.

Two Stroop tasks were administered. Words were presented in yellow, green, blue or red — first against a white background and then against a black background (the difference in background colour was the relevant cue for the following recognition task). The words were either colour names, neutral or sleep-related. Subjects had to respond to the colour of the word by pushing the corresponding colour-key on the keyboard. A more detailed account of this task can be found elsewhere (Pattyn et al., 2010).

In the recognition task participants were instructed to recognise the words from the first Stroop task with white background and words from the second Stroop task with the black background, this by either pressing the 'Y' key (for the white background) or the 'N' key (for the black background) on the keyboard. New words were to be ignored. A more detailed account of this task can be found elsewhere (Pattyn et al., 2010).

The Symbol Substitution task in the Wais III (Wechsler, 1997) was applied. The experimenter was present to monitor the time limit of 1 min.

2.3. Polysomnography (PSG), fast Fourier analyses, heart rate, proximal skin temperature and EEG-power-ratio

According to the 10–20 system, electro-encephalogram (EEG) electrodes were placed at positions F3, C3, O1, F4, C4 and O2 together with electro-oculogram-, submental electro-myogram- and electro-cardiogram (ECG) electrodes. Skin temperature was measured at the top of the left ring finger, the forehead and peri-axillar at the inner side of the right upper arm [proximal skin temperature (proximal-T)]. All channels were measured at 200 Hz by the Medatec DREAM system (Medatec nv., Brussels, Belgium). Afterwards the PSG-recordings were blinded and scored by trained technicians, according to the Rechtschaffen and Kales criteria (1968). The amount of data of both proximal-T and heart rate (HR) was reduced in size by calculating within each individual night the mean proximal-T and HR for each five minute interval.

Fast Fourier analyses implied the computation of the following frequency bands for all consecutive 30-second epochs: delta (0.5–4 Hz), theta (5–8 Hz), alpha (8–12 Hz) and beta (12–24 Hz). In addition, beta-activity was subdivided into: sigma (12–16 Hz), low beta (16–20 Hz) (beta-lo) and high beta (20–24 Hz) (beta-hi). Hereby we focused ourselves on positions C3 and C4 during the first two deep sleep (DS) episodes (see below for how these episodes were defined). The 30-second sleep scores were matched with the power spectra analyses and epochs with artifacts were excluded by visually examination (Jenni and Carskadon, 2004) [mean percentage + SE (standard error) artifact-free 30-second epochs of DS-episode 1 (REF: 95.22 ± 1.11; COG: 94.78 ± 0.90; Wilcoxon paired t-test = 52.00; ns.; n = 15) and 2 (REF: 96.75 ± 0.78; COG: 95.07 ± 1.62; Wilcoxon paired t-test = 26.00; ns.; n = 14)].

Sleep onset (SO) was individually defined as the time between lights out and the first three consecutive epochs of stage 1. DS-episodes started with the first occurrence of stage 3 and included only stage 3 and stage 4 epochs. DS-episodes were separated from each other either by the occurrence of a REM sleep episode or in case of a skipped REM sleep episode, we treated two DS-episodes as distinguished from each other as soon they were separated by stages 1 or 2, wakefulness or movement time for at least 12 consecutive minutes (Jenni and Carskadon, 2004).

An EEG power ratio (EEG-PR) was calculated: EEG-PR = (delta + theta)/(alpha + beta) (adaptation of the formula by Muresanu et al.

(2008)). This was performed for the first and second DS-period of each REF- and COG-night. An increase in EEG-PR reflects a slowing of the EEG, whereas a decrease in EEG-PR is indicative of an EEG-acceleration (Muresanu et al., 2008).

2.4. Statistical analysis

Due to lack of normal distribution of the data and small sample size, non-parametric methods of analysis were used. The questionnaire data yielded by the CS, KSS, POMS and AD-ACL were analysed using non-parametric Friedman ANOVA. The condition (REF or COG) and time (upon arrival, in the evening and in the morning) were extracted as within-subject variables. Where appropriate Wilcoxon paired *t*-tests were used for post hoc analyses. Bonferroni correction was applied to adjust the α of 0.05 to correct for multiple comparisons. Considering the within subject design Wilcoxon paired *t*-tests were used to analyse the sleep diaries-, EEG- and FFT-data.

Effect size (d) was calculated using the formula for within subject data proposed by Dunlap et al. (1996) and recommended by Nakagawa and Cuthill (2007). All statistical analyses were executed using STATISTICA 10 (StatSoft, Inc. 1984–2011). Data were expressed as mean ± SE (standard error).

3. Results

For the sake of clarity, all questionnaire data were rescaled (if necessary) to make sure that each scale's minimum score would be zero. Sample size may differ due to technical problems in the PSG recordings of either the REF- or COG-night or due to missing questionnaire data. PSG- and FFT- variables that take the whole night into account are executed on 13 subjects. Recordings from 2 subjects contained insufficient data to be included in all the analyses. Heart rate analyses include 11 instead of 15 subjects due to loss of ECG-signal in either the REF- or COG-night.

3.1. Questionnaires

No significant main effects (neither of time nor between conditions) were found within the CS ($\chi^2_{df=5} = 8.19$; ns.; n = 13) or the ES ($\chi^2_{df=5} = 10.79$; ns.; n = 12).

When analysing the sleep diary data no significant differences were found between the REF- and COG-conditions, with regard to the subjective SO-latency (SOL), subjective time in bed (TIB), subjective sleep period time (SPT), subjective total sleep time (TST), percentage of subjective wake after sleep onset (WASO) or sleep efficiency (ns.; n = 14). The REF- and COG-conditions also did not differ in subjective sleep quality score (ns.; n = 14) and the feeling of being refreshed (ns.; n = 14).

A significant main effect for time was found ($\chi^2_{df=5} = 38.28$; p<0.0001; n = 13) within the KSS. Post hoc analyses (corrected $\alpha = 0.017$) showed that in both the REF- (p<0.017; n = 13) and COG- (p<0.017; n = 13) conditions subjects evaluated themselves as significantly sleepier in the evening than upon arrival. Only in the REF-condition, sleepiness was reduced significantly from the evening to the morning (p<0.017; n = 13).

With regard to the stress subscale of the AD-ACL no significant effects were found ($\chi^2_{df=5} = 8.44$; ns.; n = 13). Concerning the arousal subscale of the AD-ACL, a significant main effect for time was found ($\chi^2_{df=5} = 18.94$; p<0.01; n = 13). Further post hoc analyses (corrected $\alpha = 0.017$) showed only in the COG-condition a significant decrease in arousal from arrival in the laboratory to the evening (p<0.017; n = 13) and a significant increase in arousal from the evening to the morning both in the REF- (p<0.017; n = 13) and the COG- (p<0.017; n = 13) conditions.

For the POMS-scales tension, depression and anger, no significant differences were found between the REF- and COG-conditions at any

time point. With regard to the fatigue subscale of the POMS, there was a significant main effect of time ($\chi^2_{df=5} = 24.65$; p<0.001; n = 12). Post hoc (corrected $\alpha = 0.017$), in both conditions a significant decrease in fatigue was found from the evening to the morning (REF: p<0.017; COG: p<0.017; n = 12). Also, a significant main effect of time was found for the subscale vigor ($\chi^2_{df=5} = 13.72$; p<0.05; n = 13) of the POMS. In the REF-condition only, post hoc analysis (corrected $\alpha = 0.017$) showed a significant decrease in vigor from the time of arrival in the laboratory to the evening (p<0.017; n = 13). A significant increase in vigor from the evening to the morning was observed only in the REF-condition (p<0.017; n = 13).

3.2. Polysomnography

Compared to the REF-condition, objective SOL was significantly prolonged (p<0.01; d = 1.83; n = 15) in the COG-condition. No significant differences between conditions were found with regard to total sleep time (TST), sleep stage distribution, latency to the first (DS1) and second (DS2) DS episodes, and latency to 1st, 2nd, 3rd and 4th REM sleep periods (Table 2).

3.3. Fast Fourier analyses

Both at positions C3 (p<0.05; n = 13) and C4 (p<0.05; n = 14), there was a significant higher percentage of beta-activity in the second deep sleep period in the night after the cognitive induction. At position C4 the percentage beta-activity was also higher in the first deep sleep period in the night after the cognitive induction (p<0.05; n = 14). At position C3 a significant decrease in the percentage beta-activity from the first to the second deep sleep period was found within the reference nights (p<0.05; n = 13) (see Fig. 1).

When comparing more in detail the frequency distributions of DS1 and DS2 both at positions C3 and C4, a significantly higher percentage of sigma activity was found within the DS2-period of the COG-condition at position C3 (p<0.05; n = 13). At position C4, a significant higher amount of sigma activity was found within the DS1-period of the COG-condition (p<0.05; n = 14). Within the DS2-period of the COG-condition significantly more beta-low (p<0.01; n = 14) and beta-high (p<0.05; n = 14) activity was found compared to the activity within the DS2-period of the REF-condition. Both at positions C3 and C4 several trends were found (see Table 3 and Table 4 for an overview of the results).

In both REF- and COG-condition the EEG-PR was calculated both for DS1 and DS2. A significant main effect of condition was found both at position C3 ($\chi^2_{df=3} = 7.99$; p<0.05; n = 13) as at position C4

Table 2
PSG variables: both for the reference (REF) and cognitive arousal (COG) conditions (mean ± SE).

	REF	COG	P
*SOL (min)	12.60 ± 1.58	20.27 ± 2.33	<0.01
*TST (min)	422.08 ± 8.11	426.31 ± 7.04	ns.
*% S1	5.00 ± 1.10	4.16 ± 1.00	ns.
% S2	43.40 ± 1.93	47.79 ± 2.50	ns.
% DS	30.15 ± 1.96	27.35 ± 2.54	ns.
% REM	17.53 ± 1.20	15.85 ± 1.24	ns.
% Awake	3.12 ± 0.78	3.86 ± 0.63	ns.
% MT	0.80 ± 0.41	1.01 ± 0.54	ns.
*1st REM-L	103.12 ± 10.60	121.73 ± 15.54	ns.
2nd REM-L	202.15 ± 10.12	228.27 ± 16.46	ns.
3rd REM-L	302.92 ± 9.74	312.54 ± 14.43	ns.
4th REM-L	391.88 ± 7.97	391.94 ± 13.96	ns.
*1st DS-L	14.30 ± 1.65	13.80 ± 1.83	ns.
2nd DS-L	90.15 ± 5.19	90.85 ± 7.06	ns.

* SOL (min) = sleep onset latency in minutes, TST (min) = total sleep time in minutes, % S1/2/3/4 = percentage stage 1/2/3/4, % MT = percentage movement time, REM-L = latency to 1st/2nd/3rd/4th REM-period presented in minutes, DS-L = latency to 1st/2nd DS-period presented in minutes.
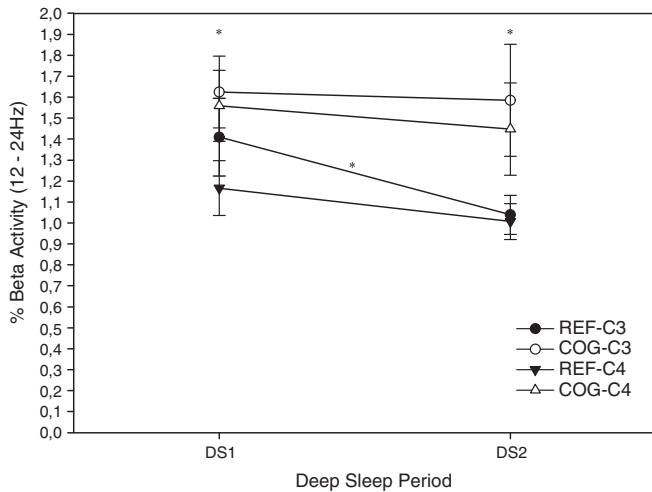
Fig. 1. % Beta activity (12–24 Hz) recorded both at positions C3 and C4 during the first and second deep sleep period (DS1 and DS2) of both reference (REF) and cognitive arousal (COG) condition (* p<0.05).

($\chi^2_{df=3} = 16.80$; p<0.001; n=14). Within DS1 a significant difference between REF- and COG-condition was found at position C4 (p<0.05; n=14). Within DS2, a significant difference between REF- and COG-condition was found at position C3 (p<0.05; n=13) and a trend at position C4 (p=0.05; n=14) (Fig. 2).

3.4. Physiologic variables

A significant main effect of time was found ($\chi^2_{df=15} = 89.30$; p<0.001; n=14) when analysing the first 40 min of proximal-T recordings after lights out (lo). Post hoc analyses (corrected $\alpha=0.0071$) showed that in both conditions proximal-T increased significantly over the first 20 min after lo. When analysing the 120 min after SO a significant main effect of time was found within the first 60 min ($\chi^2_{df=23} = 64.09$; p<0.001; n=14). In the first 5 min after SO only in the COG-condition there was a significant increase in proximal-T (p<0.0046; n=14). No significant main effects were observed within the second hour after SO ($\chi^2_{df=23} = 12.37$; ns.; n=14) (see Fig. 3).

A significant main effect of time was found ($\chi^2_{df=15} = 30.65$; p<0.01; n=11) when analysing the first 40 min of HR recordings after lo. In both conditions there was a decrease in HR over time. However, post hoc analyses with corrected $\alpha=0.0071$, showed no significant differences. When analysing the 120 min after SO no significant main effects were found, neither within the first 60 min ($\chi^2_{df=23} = 11.04$; ns.; n=11), nor the last 60 min ($\chi^2_{df=23} = 33.90$; ns.; n=11) (see Fig. 4).

Table 3
FFT-frequencies at location C3: both for DS1 and DS2 in the reference (REF) and cognitive arousal (COG) conditions (mean % ± SE).

C3	REF		COG	
	DS1	DS2	DS1	DS2
Delta	78.91 ± 1.72	83.92 ± 1.17	78.93 ± 1.33	81.44 ± 1.71
Theta	12.17 ± 0.95	8.97 ± 0.72	11.43 ± 0.77	9.37 ± 0.75
Alpha	3.72 ± 0.35	3.27 ± 0.28	3.94 ± 0.30	3.56 ± 0.25
Sigma	2.87 ± 0.36	2.08 ± 0.18[*]	3.33 ± 0.38	3.26 ± 0.59[*]
Beta-lo	0.86 ± 0.13	0.63 ± 0.07[a]	0.94 ± 0.11	0.90 ± 0.14[a]
Beta-hi	0.50 ± 0.10	0.40 ± 0.06	0.60 ± 0.08	0.59 ± 0.10

[a] Trend (0.05<p<0,10) between the REF and COG conditions.
[*] p<0.05.

Table 4
FFT-frequencies at location C4: both for DS1 and DS2 in the reference (REF) and cognitive arousal (COG) conditions (mean % ± SE).

C4	REF		COG	
	DS1	DS2	DS1	DS2
Delta	81.05 ± 1.61	84.57 ± 0.72[a]	79.73 ± 1.18	82.19 ± 1.59[a]
Theta	11.40 ± 0.90	8.77 ± 0.57	10.99 ± 0.63	9.19 ± 0.72
Alpha	3.43 ± 0.37[a]	3.21 ± 0.22	3.83 ± 0.31[a]	3.46 ± 0.28
Sigma	2.44 ± 0.22[*]	2.17 ± 0.17[a]	3.25 ± 0.41[*]	2.94 ± 0.47[a]
Beta-lo	0.67 ± 0.51	0.54 ± 0.08[**]	0.88 ± 0.09	0.85 ± 0.13[**]
Beta-hi	0.38 ± 0.29[a]	0.32 ± 0.04[*]	0.55 ± 0.07[a]	0.55 ± 0.08[*]

[a] Trend (0.05<p<0.10) between the REF and COG conditions.
[*] p<0.05.
[**] p<0.01.

4. Discussion

In accordance with the aim of our study we succeeded in inducing a cognitive load, void of an emotional component. That is, no statistically significant effects on emotional experience were observed. In line with our hypotheses and consistent with earlier research (Gross and Borkovec, 1982; Haynes et al., 1981), 80% of our subjects experienced longer sleep onset after pre-sleep induced cognitive arousal — a sleep onset latency with a mean increase of 8 min, compared to the reference condition.

No other significant differences in sleep macrostructure, neither in sleep stage distribution nor in latencies to REM- or to deep sleep-periods, between reference and cognitive arousal nights were found. Lack of any further differences in macrostructure might be due to the rather mild induction lasting 'only' half an hour. Still these results are also in line with earlier research among good sleepers (Bonnet and Arand, 1998, 2005).

However, when analysing the sleep microstructure, important differences between reference and cognitive induction nights were found. Since research has indicated that less restorative sleep might be due to a disruption of deep sleep as indicated by alterations of its micro-structure (Moldofsky et al., 1975). We focused our analysis on the first and second deep sleep period. A lower EEG-power ratio both for deep sleep periods 1 and 2 of the cognitive induction night in comparison with the reference night was observed. As stated before, an increase in the EEG-power ratio applied, reflects a slowing of the EEG, whereas a decrease in EEG-power ratio is indicative of an EEG-acceleration (Muresanu et al., 2008). Also an increase in relative power in the high frequency EEG bands was found in the night
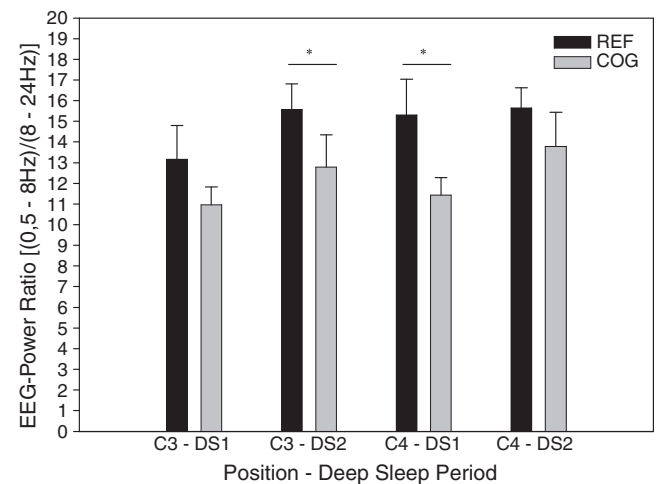

Fig. 2. EEG-power ratio [(0.5–8 Hz)/(8–24 Hz)] calculated for the recordings of both positions C3 as C4 during first and second deep sleep period (DS1 and DS2) both for the reference (REF) and cognitive arousal (COG) condition (* p<0.05).
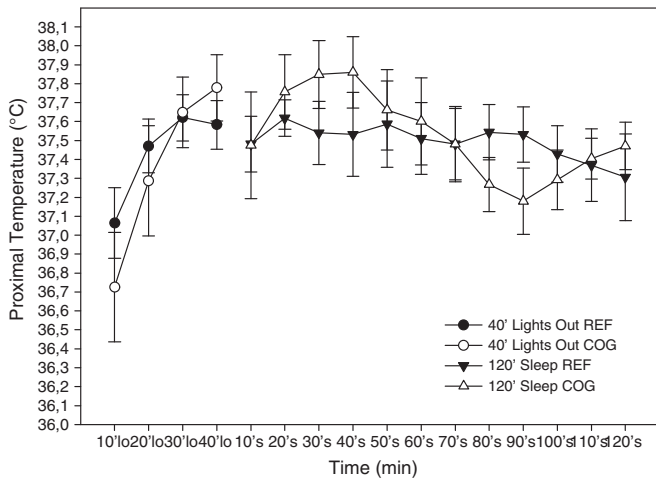
Fig. 3. Proximal skin temperatures (°C) of the reference (REF) and cognitive arousal (COG) conditions for the first 40 min after lights out (lo) and for the first 120 min of sleep (s).

after the cognitive induction. High frequency beta EEG activity during wake has been related to cognitive functioning, and it has been assigned as indicator of increased cortical arousal in insomnia (Huang et al., 2011; Perlis et al., 2001a; Perlis et al., 2001b). The findings that (at both positions C3 and C4) the percentages of beta-activity in the first and second deep sleep periods in the night after the cognitive induction was remarkably higher than after the reference night, is in accordance with earlier research (Morin et al., 2008; Schabus et al., 2006; Schmidt et al., 2006). Although at position C3 there was no significant difference in percentage of beta-activity within the first deep sleep period, within the reference night there was a significant decrease in beta activity from first to second deep sleep period. Since the beta range (12–24 Hz) is rather broad, more detailed analyses were required. This way, at position C4, we found a significant increased percentage of sigma activity in the first deep sleep period and a significant higher percentage of beta-low and beta-high activity within the second deep sleep period. At C3 an increased sigma activity was found within the second deep sleep period.

The role of specific task characteristics of the pre-sleep cognitive tests we used – such as for instance novelty – that may have contributed to the effects on subsequent sleep we observed, are methodologically difficult to entangle. The prolonged high frequency activation we observed might be the result of an implicit learning or memory

Fig. 4. Heart rates (bpm) of the reference (REF) and cognitive arousal (COG) conditions for the first 40 min after lights out (lo) and for the first 120 min of sleep (s).

effect we induced by the pre-sleep cognitive tasks. Although our tasks were not explicitly formulated as learning tasks, implicitly they all addressed working memory processes (Baddeley, 2003) with mainly a focus on recall (Digit Span, Stroop-recognition-task). Earlier research on pre-sleep learning of word pairs resulted in increased sigma activity [fast (>13 Hz) and slow spindle activity (<13 Hz)] during subsequent sleep (Schabus et al., 2008). With regard to the emotional component of the induction, we kept the tasks emotionally neutral, short lasting and without positive or negative feedback.

Although the tasks in this study were unconditional and although there was no prospect of any recall or retesting in the morning, the cognitive load of the tasks was sufficient to block the de-arousal of the brain and the cognitive processes, and thus hampering the normalcy of the automatic sleep onset process (Espie et al., 2006).

Although in the night after the cognitive arousal induction large fluctuations over time in proximal skin temperature were observed, opposite to a rather stable increase in the reference night, only few significant differences were observed within these fluctuations. After lights out a significant increase in the proximal skin temperature was observed in both conditions, lasting 20 min. Only in the first 5 min after sleep onset in the cognitive arousal condition a significant increase in the proximal skin temperature was observed. Although heart rate in the cognitive arousal condition dropped below and stayed below the reference level, no between condition differences were observed due to large inter-individual differences. Nonetheless these are limited observations in both proximal skin temperature and heart rate measurements, these observations might indicate that pre-sleep cognitive arousal induction might also have an influence at the physiological level, an influence which future research might take into account more closely.

The subjective sleep quality scores taken the following morning and the sleep diary variables did not indicate that our subjects experienced poorer sleep after being cognitively aroused than after neutral nights in the reference condition. However, taken into account that it was only a mild cognitive induction and that apart from sleep onset no further significant differences in sleep macrostructure were found, it is not remarkable that these subjects, who were all good sleepers, did not change their subjective account of the past night merely experiencing only once the negative impact of enhanced pre-sleep cognitive arousal. Attention focussing on the ongoing cognitive processes (e.g. prospect of recall in the morning) makes it less easy and more intentional to fall asleep. In the long run increased sleep intention might evolve into sleep effort making it ironically harder to sleep (Ansfield et al., 1996; Espie et al., 2006). Future research might however give insight into the impact of prolonged pre-sleep cognitive arousal on subsequent objective and subjective sleep.

Overall, we might conclude from these results that, for healthy sleepers, half an hour of cognitive tasks in the evening, maximally exclusive of emotional components induced substantial cognitive arousal that interfered with the sleep. More specifically, the sleep onset period was lengthened and the presence of high frequency EEG activity increased during the first and second deep sleep episodes which are highly important for recovery sleep. This means that, independent of their emotional components, cognitive-arousing pre-sleep activities have potentially sleep-disturbing effects. These pre-sleep cognitive activities might be important as focus of interest in therapeutic interventions with insomniacs, as well as for campaigns targeting the pre-sleep habits of adolescents and young adults.
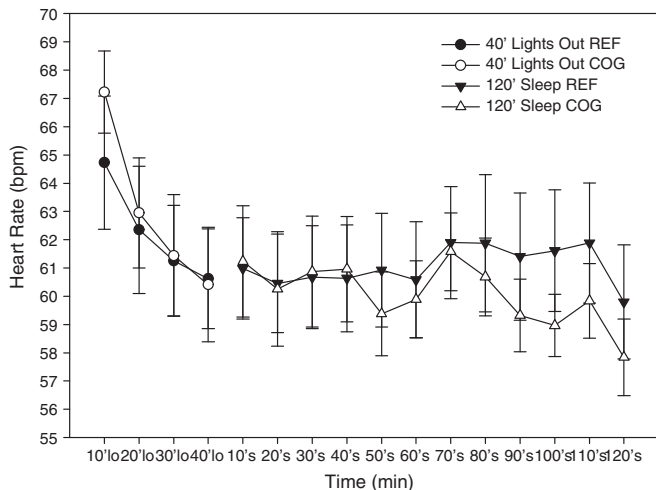
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