The role of presleep negative emotion in sleep physiology

Vandekerckhove, Marie; Weiss, Rolf; Schotte, Christiaan; Haex, Bart; Exadaktylos, Vasilis; Verbraecken, Johan; Cluydts, Raymond

Published in:
Psychophysiology

Publication date:
2011

Psychophysiology, ■■■ (2011), 1–7. Wiley Periodicals, Inc. Printed in the USA.

DOI: 10.1111/j.1469-8986.2011.01281.x

The role of presleep negative emotion in sleep physiology

M. VANDEKERCKHOVE,[a] R. WEISS,[b] C. SCHOTTE,[c] V. EXADAKTYLOS,[d] B. HAEX,[e] J. VERBRAECKEN,[f] AND R. CLUYDTS[g]

[a]Faculty of Psychological and Educational Sciences, Department of Experimental and Applied Psychology, Research Group of Biological Psychology, Department of Clinical and Life Span Psychology, Vrije Universiteit Brussel, Brussels, Belgium
[b]Department of Biological Psychology, University of Cologne, Cologne, Germany
[c]Department of Clinical Psychology, Vrije Universiteit Brussel, Brussels, Belgium
[d]Division of Measure, Model and Manage Bioresponses, Katholieke Universiteit Leuven, Heverlee, Belgium
[e]Department of Biomechanics and Engineering Design, Katholieke Universiteit Leuven, Leuven, Belgium
[f]Multidisciplinary Sleep Disorders Centre, Antwerp University Hospital, Edegem, and University of Antwerp, Antwerp, Belgium
[g]Faculty of Psychological and Educational Sciences, Department of Experimental and Applied Psychology, Research Group of Biological Psychology, Vrije Universiteit Brussel, Brussels, Belgium

Abstract

Although daytime emotional stressful events are often presumed to cause sleep disturbances, the few studies of stressful life events on sleep physiology have resulted in various and contradictory findings. As research has focused in particular on stress in itself, the present study is the first to investigate the effect using polysomnography (PSG). Results indicate a significant increase in sleep fragmentation, as expressed by decreased sleep efficiency, total sleep time, percentage of rapid eye movement (REM) sleep, and an increased wake after sleep onset latency, total time awake, latency to SWS, number of awakenings and number of awakenings from REM sleep. The results demonstrate that negative emotion correlates with enhanced sleep fragmentation helping us to understand why sleep patterns change and how sleep disturbances may develop.

Descriptors: Affect, Emotion, Stress, Sleep, Polysomnography, Sleep disturbances

Daytime events, and especially emotionally stressful events and their subsequent presleep affective and cognitive activity, may affect each phase within sleep physiology (Harvey, 2005; Thomsen, Mehlsen, Christensen, & Zachariae, 2003; for a review, see Vandekerckhove & Cluydts, 2010). Each sleep phase has a distinct set of associated physiological, neurological, and psychological characteristics. Sleep proceeds in cycles of rapid eye movement (REM) and nonrapid eye movement (NREM) sleep. REM (or dream) sleep is characterized by rapid eye movements, but also low muscle tone and rapid, low-voltage waves (Iber, Ancoli-Israel, Chesson, & Quan, 2007). NREM involves three stages: N1 (initial sleep stage), followed by N2 characterized by high amplitude slow wave brain activity, and then stage N3, which is also called deep sleep, delta sleep, or SWS. In particular, REM sleep seems to be highly influenced by daily emotional stress. Although the exact effects of emotional stress on sleep and REM sleep has yielded inconsistent results, sleep problems such as difficulties in falling asleep, fragmentation of sleep, and negative emotions within a dream often might be the result. Other effects that also have been reported are an exaggerated startle response, decreased dream recall, elevated awakening thresholds from REM sleep, increased or decreased latency to sleep and REM sleep, and both decreased and increased percentage of REM and REM sleep duration (Cartwright, Luten, Young, Mercer, & Bears, 1998; Cui, Li, Suemaru, & Araki, 2007; Hobson, Stickgold, & Pace-Schott, 1998). For instance, watching aversive films before sleep has been found to influence sleep pattern and to change the level of dream content and emotional experience in the first REM periods of the night (Lauer & Lund, 1987). In another study, stress exposure, such as watching an exciting film, has been inconsistently associated with increased percentage of REM sleep and REM sleep duration (Goodenough, Witkin, Lewis, Koulack, & Cohen, 1974). Other forms of emotional stress, such as interpersonal conflict, have been associated with sleep disturbances (Brisette & Cohen, 2002). Edéll-Gustavsson (2002) studied patients two days before coronary bypass and found that involuntary thoughts predicted sleep fragmentation. Even when subjects performed cognitive tasks during half an hour before sleep, sleep onset latency was significantly prolonged (Wuyts et al., 2011). In still another study (Germain, Buysse, Ombao, Kupfer, & Hall, 2003), the subjects were told before sleep that they would have to give a speech in the morning, where their performance would be evaluated. An increase in REM sleep density across REM periods, a decrease in late-night average REM count, and a slower rate of increase across successive REM periods immediately after the stress exposure have been observed. The finding that average REM count is increased is in line with previous longitudinal studies on the effects of direct stress exposure (Buysse et al., 1992;

This study was financially supported by the agency for Innovation by Science and Technology (IWT). We thank David Emmans for editing the text and Johan Steen for helpful comments.

Address correspondence to: Marie Vandekerckhove, Faculty of Psychology & Educational Sciences, Department of Clinical & Life Span Psychology & Experimental and Applied Psychology, Pleinlaan 2 | B - 1050 Brussel, Belgium. E-mail: Marie.Vandekerckhove@vub.ac.be

Monroe, Thase, & Simons, 1992). Also, the effects of more longtime stress exposure resulted in increased REM count (Pillar, Malhotra, & Lavie, 2000; Reynolds et al., 1993). In a study of Ackerstedt and coworkers (Ackerstedt, Kecklund, & Axelsson, 2007) in which sleep was recorded following days of low, high, and intermediate stress, a decrease in sleep efficiency, a higher percentage of being awake, and an increased latency to deep sleep or SWS in periods of high stress have been observed. However, no REM sleep or other sleep variables were affected. The above-mentioned findings on the impact of presleep negative affect and worry on subsequent sleep are partly similar to those showing sleep-related problems during on-call nights such as reduced SWS, longer latency to SWS, more awakenings, but with less REM sleep (Nicol & Botterill, 2004). Also, in a review of Kim and Dimsdale (2007), experimentally induced stress appeared to result in decreased SWS, REM sleep, and sleep efficiency as well as in increases in the number of awakenings. Taken together, alterations or abnormalities in sleep can be related to variables associated with the state of the individuals during the day (Hauri & Hawkins, 1971; Stickgold, Hobson, Fosse, & Fosse, 2001). However, it is clear that, in particular, the effects published to date in the literature of emotional presleep stress on sleep, especially REM sleep are still limited and contradictory. Some research findings indicate that REM sleep appears to modulate our daily mood as well as to facilitate the integration of affective life events into long-term memory (Agargun & Cartwright, 2003; Cartwright, Baehr, Kirkby, Pandi-Perumal, & Kabat, 2003; Hu, Stylos-Allen, & Walker, 2006; Payne & Kensinger, 2010; Payne, Stickgold, Swanberg, & Kensinger, 2008; Wagner, Hallschmid, Rasch, & Born, 2006). In correspondence, sleep deprivation such as REM sleep deprivation due to changes of normal sleep patterns has a strong impact on the way we process and recover from our daily experiences. People with disturbed sleep seem to show almost the same physiological changes as those under stress during the day, including increased cortisol, heart rate, body temperature, and oxygen consumption (Ackerstedt, 2006). As both sleep disturbance and daytime stress influence each other negatively, a dysfunctional or even psychopathological cycle might develop. Sleep disturbances due to our affective state during the day not only restrict our feeling of everyday happiness, but may even have a prognostic significance in predicting mental well-being. Without sufficient healthy sleep, negative emotional reactivity seems to be significantly enhanced, and positive reactions to pleasant events are often subdued (Zohar, Tzischinsky, Epstein, & Lavie, 2005). Similarly, studies in children and adolescents found that sleep deprivation increases subjective feelings of frustration (Chelette, Albery, Esken, & Tripp, 1998), confusion and anger (Dahl, 1996; James & Gregg, 2004), irritability/aggression, somatic complaints, anxiety, depression, and paranoia (Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007).

The contradictory results on the role of a stressed state of the individual on subsequent sleep only enhance the lack of existing theoretical consensus in sleep research and theorizing on the function of sleep (Vandekerckhove & Cluydts, 2010; Walker & Harvey, 2010). The discussed findings suggest a necessity for more insight about the relation between normal day-to-day emotions and physiological sleep parameters and vice versa. Situational and dispositional factors (Kuhl, 1992) affect sleep physiology within its different stages, but this is still not well understood. As a consequence, it could prove useful to investigate not only how stress as a global phenomenon affects sleep, but also, and more specifically, how a specific daytime emotion affects sleep in particular. The goal of this study is to contribute to integrating the psychology and the neurobiology of sleep, in order to explain why sleep patterns change or do not so and in which direction changes occur, as a response to presleep emotion. Within the understanding of situational influences on sleep physiology, the effects of a specific emotion such as an emotional failure experience have, as far as we know, never been investigated previously. Studies on specific presleep emotions should help to clarify when and how sleep becomes fragmented and sleep phases change, especially REM sleep. They will brings us a step further towards a better insight into the function of sleep, the definition of sleep quality, and the mechanisms underlying the development and maintenance of sleep disturbances in people experiencing transient but stressful life events.

Methods

Participants

Participants were recruited for a study on cognition and sleep. They were contacted via different sources, such as mailing lists in the electronic communication platform of the Vrije Universiteit Brussel and via acquaintances and colleagues of the researchers. Fourteen healthy Dutch-speaking participants were included in this study with an age range of 19 to 56 yrs ($M = 32.38$, $SD = 12.23$). The participants had a good baseline for quality of sleep and no sleep disturbances (including apnea and insomnia), with an average of 6 to 9 hours of sleep per night and no psychiatric disorder or intake of medication or drugs influencing sleep, and no psychotherapeutic treatment, pregnancy, shift working, smoking habits, or intake of more than three beverages containing caffeine or alcohol. To fulfill these inclusion criteria, the subjects completed the Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Only moderate to good sleepers were included, who reported having their sleep-onset latency below 20 min on average. There were no habitual short or long sleepers. These subjects were also screened with the semistructured interview M.I.N.I. (Mini International Neuropsychiatric Interview; Van Vliet & De Beurs, 2007) as well as with the Symptom Check List 90 (Arrindell & Ettema, 2003), to exclude psychiatric and psychological problems. In the debriefing procedure, one participant suspected some of the rationale of the study and was excluded, too. As a result of these criteria, 13 participants were finally selected (men = 6; women = 7). Participants gave their written informed consent and received 120 Euros for their participation of four nights. The study was approved by the Ethics Committee at the Vrije Universiteit Brussel.

Procedure

Participants were scheduled for four nights each with 8 h of time to spend in bed in the sleep laboratory of the Vrije Universiteit Brussel (Table 1). The first night included a diagnostic sleep study to rule out any sleep disorder and to adapt the participants to the laboratory procedures. The second night served as baseline for the sleep parameters. Following the adaptation and the baseline nights, subjects underwent two experimental nights: one night with the experimental failure condition, and one night with a

Table 1. *Overview of the Experimental Procedure*

Time (hours)	Activity
19:30	Arrival at the laboratory Completion of PANAS Application of the electrodes
20:00	Experimental emotion induction of failure by feedback on a cognitive performance task Control condition: neutral feedback on a cognitive performance task
22:00	Completion of PANAS Free time with relaxing literature
23:00	Bedtime Start of PSG recording
07:00	Awakening Electrodes removed
07:15	Completion of PANAS
07:30	Breakfast
08:00–09:00	Start of normal daytime activities

Note. PANAS = Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988); PSG = polysomnography.

control or neutral condition. Both conditions were done by the same experimenter. Although there could be a possible negative effect from having the neutral condition occur after the failure condition, we gave priority to counterbalancing the two experimental nights, in order to avoid natural order effects. Order effects in sleep physiology occur when an individual has to sleep for 2 or more days in a new setting. In addition, between each night at the laboratory, there was always at least one night or at most four nights at home without any measurements.

Prior to the study, participants were asked to keep a sleep diary for the previous 2 weeks, to check for irregular sleep-wake schedules, and were asked to maintain their normal sleep-wake patterns 2 weeks before the experiment.

Adaptation and baseline night. On the adaptation night, participants arrived at the lab at 20:00. After detailed explanation of the course of the study, the procedure that they would be asked to perform, and the completion of informed consent forms, electrodes were attached. In order to assess the influence of failing at the tasks and the subjects' actual level of affect, emotional experience was assessed by the Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988). Between 21:00 and 23:00, only recreational activities were allowed, such as reading relaxing literature or watching some nature documentaries. At 22:45, a wake-electroencephalogram (EEG) was recorded. At 23:00, the participants were asked to go to bed. At 07:00 the next morning, they were awakened and completed the PANAS. At 07:15, the electrodes were removed and breakfast was served. At about 08:00, the participants left the lab.

Experimental night: Failure experience. The induction consisted of a failure feedback in response to different cognitive tasks, which the participants were told constituted an intelligence test that they had to perform. In several prior pilot tests, the level of difficulty within the tasks was adapted until all participants had a score between zero and three. After this phase, we validated the test on 12 persons. The tasks and failure feedback were presented on a computer in order to attain maximum standardization and also to be able to individualize the scores based on their real performances.

For the experimental night, participants arrived at the lab at 20:00. After the electrodes were attached, participants had to watch an emotionally neutral film on nature of 5 min duration, which served as a baseline condition (Mauss, Cook, Cheng, & Gross, 2007). After rating their emotional experience with the PANAS, the experimenter entered the room again to introduce the intelligence test, which the participants were asked to complete. The experimenter informed the participant that he or she would be participating in a new cognitive test, reflecting their level of intelligence and predicting future professional achievement. The exact translation of the wording is: "It is very important that you concentrate and do this intelligence test to the best of your ability. The test is being developed for international use and reflects your potential regarding general intelligence and professional achievements."

The test (taking about 20 min) contained six subtests: (1) spatial ability tasks, (2) logical steps tasks (figure completion) and mathematical problem-solving tasks (e.g., number completion: 100, 95, 79, 68, ?, 40, 23; see Figure 1 below), (3) a numeric and visual memory test as the first part of the memory test (a series of negative pictures: IAPS; Lang, Bradley, & Cuthbert, 2008) was presented accompanied by a number below each picture. After the presentation of these pictures and the respective numbers in the second part of the task, the participant had to recall the number given with the picture, (4) counting tasks: Participants were asked to count backwards quickly in increments of 7 and 13 from 15,732 and to repeat this five times, (5) a semantic reasoning task: This final task involved an impossible semantic test, whereby three unrelated words (such as blood, music, cheese) were presented ten times and the participants had to find the (impossible) related word. Between the different tasks, the experimenter entered the room several times, giving various comments in an annoying and irritated way. The first time, the following feedback was given: "You are moving around too much, causing physiological artifacts which make the data useless. Please sit still." After 5 min, the experimenter entered the room again, clearly annoyed, checking the electrodes and then leaving the room without talking to the participants. After each subtest and at the end of the test, the score obtained was presented on the computer screen accompanied by a failure feedback saying that the test achievements were weak and clearly under the mean. At the end of the testing, the experimenter informed the participants that the physiological measurements so far were useless. In reality, physiological data were not obtained and the test was too difficult to obtain good results. Before the participants went to bed at 23:00, they completed the PANAS to assess their emotional experience and recovery of negative affect by the Level of Ruminative Thought and the Impact of Event Scale (IES; Horowitz, Alden, Wiggins, & Pincus, 1979).

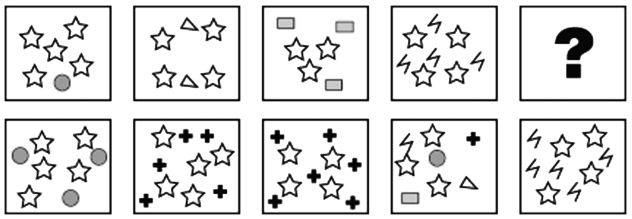

Figure 1. Example of a question: Which figure from the lower series best fits the frame with the question mark?

After their morning wakening at 07:00, mood and intrusions were assessed again. After the electrodes were removed and before leaving the lab, a "funneled debriefing" procedure was used: If the night of the failure induction came before the night of the neutral condition with the relatively easy tasks, they were told: "The task you performed yesterday is in general still far too difficult. Everyone gets a score between zero and three, which means that your score was not bad at all, but rather good and that we definitely have to adapt our tests to a more feasible level of difficulty." It was emphasized that the tasks appeared far too difficult and this made their performance unrelated to intelligence and either academic or career success.

The debriefing was done in a manner that subjects could vent their feelings about the experience. It was done in such a way that participants fully understood the nature of the study. To confirm that they felt good again, they were asked specifically about their experience again at the end of the debriefing.

For the conditions in which the failure night came after the neutral condition, the same but more profound debriefing was given except that participants were explicitly asked whether they had any idea about the aim of the study. The debriefing was done in a way that participants understood the true nature of the study and the reason for the different treatments until they felt good again.

Experimental night: Neutral experience. For the control condition, the same procedure was followed as in the night of the failure induction with identical but less difficult tasks than in the experimental condition. Prior pilot testing allowed adjusting the tasks to a medium-range level of difficulty and calibrated about the same time for solving the tasks, so that participants could perform 60% and get neutral feedback. Although the experimenter entered the room, she did not give irritated comments to the participants. After each subtest and at the end of the test, the score obtained was presented on the computer screen, accompanied by a neutral feedback saying that the test achievements were okay and approximated the mean. At the end of the testing, the experimenter informed the participants that the physiological measurements were adequate. In the debriefing phase, the experimenter said, "The task you performed yesterday was okay and your cooperation meant a step further in the validation of the study."

Statistical Analyses

Statistical analyses were performed using SPSS 19.0 software. Before analyses, normality, sphericity, and homogeneity of variances were verified. Separate 3 × 2 (emotional experience at 'time of moment' × experimental night [failure experience versus neutral experience]) mixed repeated measures ANOVA were calculated. Emotional experience measured with the Panas at the different moments throughout the night—'after the neutral film,' 'after the cognitive tasks,' and 'the following morning'—was extracted as within-subject variables and the experimental failure and neutral experiences as between-subject variables.

Measurements

Polysomnography

Polysomnography (PSG) was performed in the experimental sleep laboratory at the Vrije Universiteit Brussel. EEG electrodes were placed at positions F3, C3, O1, F4, C4, and O2 together with electrooculogram (EOG), submental electromyogram (EMG), and electrocardiogram (ECG) electrodes. A 32-channel Embla N7000 recording system was used (Medcare) with a DC offset of 500 mV max and a fixed DC low-cut filter at 0.3 Hz. The PSG and EOG signals were high-pass filtered at 0.5 Hz and low-pass filtered at 40 Hz, EMG channels were high-pass filtered at 5 Hz and low-pass filtered at 70 Hz. Absolute power (mV^2) in the delta (1–3.5 Hz), theta (4–7.5 Hz), alpha (8–12 Hz), sigma (12–14 Hz), beta-1 (12–20 Hz), beta-2 (20–35 Hz), and high beta (25.5–30 Hz) bandwidths was computed for both PSG sites of the brain. The data were scored blindly by two trained specialists reaching an inter-reliability of 90%. Sleep stages were scored based on the rules of the American Academy of Sleep Medicine (Iber et al., 2007) with the following sleep variables: percentage 1 sleep (N1), and percent stage 2 sleep (N2), total sleep time (TST), total time awake, sleep onset latency (SOL) (from the moment the lights are turned off until the first minute of N1), wake after sleep onset (WASO), sleep efficiency (SE), percentage slow wave sleep (SWS), or percentage 3 sleep (N3), latency to SWS, latency to REM sleep, percentage of REM sleep, and number of REM All epochs containing movements or EMG artifacts were excluded from analysis.

Wake-EEG

A 5-min measurement with eyes open and eyes closed was performed. An EOG and EMG submentalis were done to exclude artifacts of eye movements or muscle activity.

Sampling rate was 256 Hz, and impedances were kept below 10 kOhm. The recording montage was identical to that used for PSG. Absolute and relative values were calculated. A fast Fourier analysis was performed on the wake-EEG for a minimum of 90 s artifact-free data using Neuroguide software (Applied Neuroscience, Inc.).

Results

Negative Emotional Experience

At first we looked at the effects of the failure experience on emotional experience as measured with the PANAS compared to the effects of the neutral experience at the different moments throughout the night and on the following morning. As expected, this resulted a significant difference 'after the tasks' ($F(1,12) = 13.19$, $p = .003$, $\eta_p^2 = .52$) (neutral: $M = 1.27$, $SD = 0.21$; failure: $M = 2.10$, $SD = 0.78$) and 'the following morning' ($F(1,12) = 6.35$, $p = .027$, $\eta_p^2 = .35$) (neutral: $M = 1.16$, $SD = 0.19$; failure: $M = 1.59$, $SD = 0.57$) (see Table 2).

Effects of Failure Induction on Sleep Physiology

The experience of the failure experience in comparison with the neutral experience resulted in a significant decrease of sleep efficiency (SE) ($F(1,12) = 8.23$, $p = .014$, $\eta_p^2 = .41$). After the failure experience compared to the neutral experience, wake after sleep onset latency (WASO) increased significantly ($F(1,12) = 5.51$, $p = .037$, $\eta_p^2 = .31$). A significant effect of the failure experience on the increase of total time awake ($F(1,12) = 7.20$, $p = .020$, $\eta_p^2 = .37$) and number of awakenings ($F(1,12) = 5.22$, $p = .041$, $\eta_p^2 = .30$) in the failure night was

Table 2. *Means and Standard Deviations for Emotional Experience and Sleep Parameters*

Emotional experience	Baseline night	Neutral night	Failure night
NA before the tasks (PANAS)	—	1.12 (± 0.16)	1.30 (± 0.42)
NA after the tasks (PANAS)	1.23 (± 0.26)	1.27 (± 0.21)	2.10 (± 0.78)
NA postsleep (PANAS)	1.29 (± 0.30)	1.16 (± 0.19)	1.59 (± 0.57)
Sleep parameters	**Baseline night**	**Neutral night**	**Failure night**
% SE	95.68 (± 4.70)	94.01 (± 8.82)	88.81 (± 14.19)
WASO (min)	13.11 (± 6.54)	21.69 (± 42.25)	44.38 (± 65.73)
SOL (min)	8.85 (± 6.36)	7.93 (± 5.53)	12.77 (± 14.13)
% Total time awake	4.55 (± 4.72)	5.99 (± 8.82)	11.61 (± 15.50)
Awakenings	7.62 (± 9.64)	7.85 (± 6.45)	11.69 (± 9.23)
TST (min)	457.42 (± 32.65)	445.46 (± 45.77)	422.88 (± 71.99)
% REM	24.12 (± 4.88)	22.04 (± 6.58)	18.46 (± 6.19)
REM periods	5.61 (± 1.38)	4.92 (± 1.75)	4.76 (± 1.16)
Latency to REM (min)	96.00 (± 31.54)	107.23 (± 50.24)	101.31 (± 32.62)
Awakenings from REM	0.92 (± 0.76)	0.85 (± 1.28)	1.54 (± 1.66)
% N1	6.10 (± 3.67)	7.60 (± 2.29)	7.54 (± 3.61)
% N2	39.72 (± 9.95)	36.82 (± 4.69)	39.89 (± 13.68)
% SWS (N3)	26.78 (± 8.75)	27.02 (± 9.45)	25.33 (± 7.70)
Latency to SWS (min)	25.04 (± 11.12)	22.96 (± 9.31)	35.69 (± 25.59)

Note. PANAS = Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988); SE = sleep efficiency; WASO = wake after sleep onset; SOL = sleep onset latency; TST = total sleep time; REM = rapid eye movements; % N1 = % stage 1 sleep; N2 = % stage 2 sleep; % SWS = slow wave sleep (N3).

found. Furthermore, an increased number of awakenings from REM sleep ($F(1,12) = 8.53$, $p = .013$, $\eta_p^2 = .41$), an increase in latency to SWS ($F(1,12) = 5.16$, $p = .042$, $\eta_p^2 = .30$) was observed. On the other hand, percentage REM sleep ($F(1,12) = 5.77$, $p = .003$, $\eta_p^2 = .32$) and total sleep time (TST) ($F(1,12) = 4.27$, $p = .042$, $\eta_p^2 = .26$) decreased significantly in the failure night compared to the neutral night. Latency to REM sleep, sleep onset latency (SOL), percent SWS, N1 and N2 sleep time, and number of REM periods did not differ significantly in the failure compared to the neutral night. All results are reported in Table 2.

Possible confounding was controlled for by comparing the neutral night with the baseline night for sleep physiology, which did not reveal significant results. These effects were also most likely not due directly to other factors such as effort or arousal associated with the performance of the cognitive tasks. The tasks were controlled and tested before in a pilot study for degree of similarity in their kind and the time needed to complete them. When controlling for negative emotion as covariate with ANCOVA, no difference between both conditions could be observed, confirming the effects of negative emotion within the failure condition into sleep physiology. Also, no differences for gender in the effects of the failure experience on emotional experience or sleep were found.

Discussion

Emotional stress has been considered as the main etiological factor for primary psychophysiological sleep disorders, insomnia, and depression. An increased incidence of stressful life events has been found to precipitate such signs of sleep pathology as insomnia (Healey et al., 1981). The current study examined the effects of an emotionally distressing event on mood and sleep physiology. A worsening of the mood of participants after the failure experience was observed. This effect was also obvious at the level of sleep physiology, where a clear impact of the failure experience was observed with decreases in sleep efficiency, total sleep time, and percentage REM sleep. After the failure experience, increases were observed in wake after sleep onset, total time awake, latency to SWS, number of awakenings, and in the number of awakenings from REM sleep. These effects on sleep are in line with findings from other studies on emotional stress or cognitive stress and sleep where poorer sleep has been found as seen in decreased sleep efficiency, increase in the number of awakenings, and decreased SWS, as well as a longer REM latency and decreased REM sleep (Edéll-Gustafsson, 2002; Germain et al., 2003). Others have also found reductions in total sleep time on the night before an exam (Lester, Burch, & Dosset, 1967) and reduced SWS during nights on call (Torsvall & Ackerstedt, 1988). Ackerstedt and coworkers (2007) also documented, aside from a decrease in sleep efficiency, a higher percentage of wake after sleep onset (WASO) and a longer SWS latency. Similarly, increased stress at bedtime has been related to the report of increased sleep onset latency and more awakenings during sleep (Haynes, Adams, & Franzen, 1982; Tang & Harvey, 2004; Wuyts et al., 2011). The decrease in REM sleep in our study contradicts the results of some studies involving the presentation of unpleasant films before sleep (Buysse et al., 1992; Goodenough et al., 1975; Perlis & Nielsen, 1993; Pillar et al., 2000), where a decreased latency to REM sleep and increased REM sleep duration (Perlis & Nielsen, 1993) have been observed. The question remains whether this increase in REM sleep could be a secondary effect of processing and recovering from a negative experience. An emotionally negative experience may interfere with sleep, leading to decreased REM duration, which may be compensated for only after the level of emotional distress has been normalized. When the level of emotional experience before falling asleep is too intense, sleep fragmentation may also affect the amount of REM phases. One could assume that if a negative event has not been processed and regulated enough during the day, it needs to be additionally processed during the night. The regulatory function of REM sleep could then possibly explain the increase in REM sleep in some studies. It is plausible to

consider a ''ceiling and floor effect,'' as suggested by Cartwright (Cartwright et al., 1998; Vandekerckhove & Cluydts, 2010), which is in line with the findings reviewed above and the hypotheses of a normalization effect in the processes of (a) recovery, (b) continuing processing, and (c) recovery of an emotional stressful or negative experience. The more immediate positive regulating effects of REM sleep occur in more 'normal' levels of negative mood. It possibly also occurs less directly after a very stressful experience or in more pathological states as in anxiety disorder (Reynolds et al., 1993), or even in posttraumatic stress disorder where an increased REM latency and decreased REM time have also been observed (Cartwright et al., 1998; Vandekerckhove & Cluydts, 2010). An alternative explanation could be that when distress appears uncontrollable, the stress system may 'turn off' and preserve energy until circumstances change. This would be another possible explanation for the fact that in some studies sleep physiology is not disturbed, but even extended (Sadeh, Keinan, & Daon, 2004) and REM sleep is in fact enhanced. Considering these factors, the extent to which some stressful events need to be additionally regulated (or not), resulting in these different findings of the mentioned studies, could be moderated by individual differences in emotion regulation or coping strategies, a question we are actually investigating.

Taking these possibilities into consideration, it would be interesting to investigate in a subsequent study whether a rebound effect in REM sleep takes place on the second night, when negative emotion is not that strong anymore. The level of emotional recovery might be associated with a normalizing effect on the second, third, or on a later night, after the failure experience. In addition, it has to be noted that, although we saw clear effects of a presleep failure experience not only on the level of emotional experience, but also on the level of objective physiological parameters, further exploration and validation of these preliminary findings in larger groups is needed. In addition, more longitudinal studies should clarify the longer term effects of intermittent stress, negative life experiences, or of chronic stress and their relationship with sleep. These studies should highlight the time needed to develop chronic changes in sleep physiology and the role of impaired sleep in the development of stress-related diseases (Ackerstedt, 2006). In keeping with this line of thought, it is highly probable that REM sleep plays an adaptive role in the processing of negative emotional experiences into memory and in the regulation of emotion (Payne & Kensinger, 2010; Vandekerckhove & Cluydts, 2010; Walker & Harvey, 2010; Yoo, Hu, Gujar, Jolesz, & Walker, 2007).

In summary, the present study highlights the important interaction of emotion and sleep and therefore of situational and dispositional factors that may increase vulnerability to psychological distress, sleep disruption, and the development of sleep disturbances, chronic fatigue, and affective disorders such as depression.

References

Ackerstedt, T. (2006). Psychosocial stress and impaired sleep. *Scandinavian Journal of Work Environment and Health*, *32*, 493–501. Retrieved from http://www.sjweh.fi.

Ackerstedt, T., Kecklund, G., & Axelsson, J. (2007). Impaired sleep after bedtime stress and worries. *Biological Psychology*, *76*, 170–173. doi: 10.1016/j.biopsycho.2007.07.010.

Agargun, M. Y., & Cartwright, R. (2003). REM sleep, dream variables and suicidality in depressed patients. *Psychiatry Research*, *119*, 33–39. doi: 10.1016/S0165-1781(03)00111-2.

Arrindell, W. A., & Ettema, J. H. M. (2003). *SCL-90. Handleiding bij een multidimensionele psychopathologie-indicator*. Tilburg, The Netherlands: Swets Zeitliger. Retrieved from http://www.pearson-nl.com/scl-90-handleiding-revised.

Brissette, I., & Cohen, S. (2002). The contribution of individual differences in hostility to the associations between daily interpersonal conflict, affect, and sleep. *Personality and Social Psychology Bulletin*, *28*, 1265–1274. doi: 10.1177/01461672022812011.

Buysse, D. J., Kupfer, D. J., Frank, E., Monk, T. H., Ritenour, A., & Ehlers, C. L. (1992). Electroencephalographic sleep studies in depressed outpatients treated with interpersonal psychotherapy: I. Baseline studies in responders and nonresponders. *Psychiatry Research*, *42*, 13–26. doi: 10.1016/0165-1781(92)90036-3.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index (PSQI): A new instrument for psychiatric research and practice. *Psychiatry Research*, *28*, 193–213. doi: 10.1016/0165-1781(89)90047-4.

Cartwright, R., Baehr, E., Kirkby, J., Pandi-Perumal, S. R., & Kabat, J. (2003). REM sleep reduction, mood regulation and remission in untreated depression. *Psychiatry Research*, *121*, 159–167. doi: 10.1016/S0165-1781(03)00236-1.

Cartwright, R. D., Luten, A., Young, M., Mercer, P., & Bears, M. (1998). Role of REM sleep and dream affect in overnight mood regulation: A study of normal volunteers. *Psychiatry Research*, *81*, 1–8. doi: 10.1016/S0165-1781(98)00089-4.

Chelette, T. L., Albery, W. B., Esken, R. L., & Tripp, L. D. (1998). Female exposure to high G: Performance of simulated flight after 24 hours of sleep deprivation. *Aviation, Space, and Environmental Medicine*, *69*, 862–868. doi: 10.1016/j.paid.2006.06.002.

Cui, R., Li, B., Suemaru, K., & Araki, H. (2007). Psychological stress-induced changes in sleep patterns and its generation mechanism. *Yakugaku Zasshi*, *128*, 405–411. doi: 10.1254/jphs.08068FP.

Dahl, R. E. (1996). The impact of inadequate sleep on children's daytime cognitive functioning. *Seminars in Pediatric Neurology*, *3*, 44–50. doi: 10.16/S1071-9091(96)80028-3.

Edéll-Gustafsson, U. M. (2002). Insufficient sleep, cognitive anxiety and health transition in men with coronary artery disease: A self-report and polysomnographic study. *Journal of Advanced Nursing*, *37*, 414–422. doi: 10.1046/j.1365-2648.2002.02106.x.

Germain, A., Buysse, D. J., Ombao, H., Kupfer, D. J., & Hall, M. (2003). Psychophysiological reactivity and coping styles influence the effects of acute stress exposure on REM sleep. *Psychosomatic Medicine*, *65*, 857–864. doi: 10.1097.PSY.0000079376.87711.B0.

Goodenough, D. R., Witkin, H. A., Koulack, D., & Cohen, H. (1975). The effects of stress films on dream affect and on respiration and eye-movement activity during rapid-eye-movement sleep. *Psychophysiology*, *12*, 313–320. doi: 10.1111/j.1469-8986.1975.tb01298.x.

Harvey, A. G. (2005). Unwanted intrusive thoughts in insomnia. In D. A. Clark (Ed.), *Intrusive thoughts in clinical disorders: Theory, research, and treatment* (pp. 86–118). New York, NY: Guilford Press.

Hauri, P., & Hawkins, D. (1971). Phasic REM, depression and the relationship between sleeping and waking. *Archives of General Psychiatry*, *25*, 56–63. doi: 10.1037/0022-006X.62.1.83.

Haynes, S. N., Adams, A., & Franzen, M. (1982). The effects of presleep stress on sleep-onset insomnia. *Journal of Abnormal of Psychology*, *90*, 601–606. doi: 10.1037//0021-843X.90.6.601.

Healey, E. S., Kales, A., Monroe, L. J., Bixler, E. O., Chamberlin, K., & Soldatos, C. R. (1981). Onset of insomnia: Role of life-stress events. *Psychosomatic Medicine*, *43*, 439–451. http://www.psychosomaticmedicine.org/.

Hobson, J. A., Stickgold, R., & Pace-Schott, E. F. (1998). The neuropsychology of REM sleep dreaming. *NeuroReport*, *9*, 1–14. doi: 10.1097/00001756-199802160-00033.

Horowitz, M., Wilner, N. J., & Alvarez, W. (1979). Impact of events scale: A measure of subjective stress. *Psychosomatic Medicine*., *41*, 209–218. Retrieved from http://www.psychosomaticmedicine.org/content/41/3/209.long.

Hu, P., Stylos-Allen, M., & Walker, M. P. (2006). Sleep facilitates consolidation of emotional declarative memory. *Psychological Science*, *17*, 891–898. doi: 10.1111/j.1467-9280.2006.01799.x.

Iber, C., Ancoli-Israel, S., Chesson, A., & Quan, S. F. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications*. Westchester, IL: American Academy of Sleep Medicine. Retrieved from http://www.aasmnet.org/store/product.aspx?pid = 176.

James, J. E., & Gregg, M. E. (2004). Effects of dietary caffeine on mood when rested and sleep restricted. *Human Psychopharmacology: Clinical and Experimental*, *19*, 333–341. doi: 10.1002/hup.589.

Kahn-Greene, E. T., Killgore, D. B., Kamimori, G. H., Balkin, T. J., & Killgore, W. D. (2007). The effects of sleep deprivation on symptoms of psychopathology in healthy adults. *Sleep Medicine*, *8*, 215–221. doi: 10.1016/j.sleep.2007.07.003.

Kim, E. J., & Dimsdale, J. E. (2007). The effect of psychosocial stress on polysomnographic evidence. *Behavioral Sleep Medicine*, *5*, 256–278. doi: 10.1080/15402000701557383.

Kuhl, J. (1992). A theory of self-regulation: Action versus state orientation, self-discrimination, and some applications. *Applied Psychology: An International Review*, *41*, 95–173. doi: 10.1111/j.1464-0597.1992.tb00688.x.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). *International Affective Picture System (IAPS): Affective ratings of pictures and instruction manual* (Technical Report A-8). University of Florida, Gainesville, FL. Retrieved from http://my.ilstu.edu/ ∼ jhkahn/psy331/Langetal2008.pdf

Lauer, C. D. R., & Lund, R. M. B. (1987). Shortened REM latency: A consequence of psychological strain. *Psychophysiology*, *24*, 263–271. doi: 10.1111/j/1469-8986.1987.tb00293.x.

Lester, B. K., Burch, N. R., & Dosset, R. C. (1967). Nocturnal EEG—GSR profiles: The influence of pre-sleep states. *Psychophysiology*, *3*, 238–248. doi: 10.1111/j.1469-8986.1967.tb02701.x.

Mauss, I. B., Cook, C. L., Cheng, J. Y. J., & Gross, J. J. (2007). Individual differences in cognitive reappraisal: Experiential and physiological responses to an anger provocation. *International Journal of Psychophysiology*, *66*, 116–124. doi: 10.1016/j/ijpsycho.2007.03.017.

Monroe, S. M., Thase, M. E., & Simons, A. D. (1992). Social factors and the psychobiology of depression: Relations between life stress and rapid eye movement sleep latency. *Journal of Abnormal Psychology*, *101*, 528–537. doi: 10.1037/0021-843X.101.3.528.

Nicol, A., & Botterill, J. S. (2004). On-call work and health: A review. *Environmental Health*, *3*, 1–7. doi: 10.1186/1476-069X-3-15.

Payne, J. D., & Kensinger, E. A. (2010). Sleep leads to changes in the emotional memory trace: Evidence from fMRI. *Journal of Cognitive Neuroscience*, *23*, 1285–1297. doi: 10.1162/jocn.2010.21526.

Payne, J. D., Stickgold, R., Swanberg, K., & Kensinger, E. A. (2008). Sleep preferentially enhanced memory for emotional components of scenes. *Psychological Sciences*, *19*, 781–788. doi: 10.1111/j.1467-9280.2008.02157.x.

Perlis, M. L., & Nielsen, T. A. (1993). Mood regulation, dreaming and nightmares: Evaluation of a desensitization function for REM sleep. *Dreaming*, *3*, 243–257. Retrieved from http://psycnet.apa.org/journals/drm/20/4/227/.

Pillar, P., Malhotra, A., & Lavie, P. (2000). Posttraumatic stress disorder and sleep—what a nightmare!. *Sleep Medicine Review*, *4*, 183–200. doi: 10.1053/smrv.1999.0095.

Reynolds, C. F., Hoch, C. C., Buysse, D. J., Monk, T. H., Houck, P. R., & Kupfer, D. J. (1993). REM sleep in successful, usual, and pathological aging: The Pittsburgh experience 1980–1993. *Journal of Sleep Research*, *2*, 203–210. doi: 10.1111/j.1365-2869.1993.tb00091.x.

Sadeh, A., Keinan, G., & Daon, K. (2004). Effects of stress on sleep: The moderating role of coping style. *Health Psychology*, *23*, 542–545. doi: 10.1037/0278-6133.23.5.542.

Stickgold, R., Hobson, J. A., Fosse, R., & Fosse, M. (2001). Sleep, learning, and dreams: Off-line memory reprocessing. *Science*, *2*, 1052–1057. doi: 10.1126/science.1063530.

Tang, N. K. Y., & Harvey, A. G. (2004). Effects of cognitive arousal and physiological arousal on sleep perception. *Sleep*, *27*, 69–78. Retrieved from http://www.journalsleep.org/.

Thomsen, D. K., Mehlsen, M. Y., Christensen, S., & Zachariae, R. (2003). Rumination-relationship with negative mood and sleep quality. *Personality and Individual Differences*, *34*, 1293–1301. doi: 10.1016/S0191-8869(02)00120-4.

Torsvall, L. A., & Ackerstedt, T. (1998). Disturbed sleep while being on call. An EEG study of apprehension in ships'engineers. *Sleep*, *11*, 35–38. Retrieved from http://www.journalsleep.org/.

Vandekerckhove, M., & Cluydts, R. (2010). Sleep and the emotional brain: A bi-directional relationship? *Sleep Medicine Review*, *14*, 219–226. doi: 10.1016/j.smrv.2010.01.002.

Van Vliet, I. M., & De Beurs, E. (2007). Het Mini Internationaal Neuropsychiatrisch Interview (MINI): Een kort gestructureerd diagnostisch psychiatrisch interview voor DSM-IV en ICD-10 stoornissen. *Tijdschrift voor Psychiatrie*, *49*, 393–397. Retrieved from http://www.tijdschriftvoorpsychiatrie.nl/.

Wagner, U., Hallschmid, M., Rasch, B., & Born, J. (2006). Brief sleep after learning keeps emotional memories alive for years. *Biological of Psychiatry*, *60*, 788–790. doi: 10.16/j.biopsych.2006.03.061.

Walker, M. P., & Harvey, A. G. (2010). Obligate symbiosis: Sleep and affect. *Sleep Medicine Review*, *14*, 215–217. doi: 10/1016/j.smrv.2010.02.003.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Psychology and Social Psychology*, *54*, 1063–1070. doi :10.1037//0022-3514.54.6.1063.

Wuyts, J., De Valck, E., Vandekerckhove, M., Pattyn, N., Bulckaert, A., Berckmans, D., . . . Cluydts, R. (2011). The influence of pre-sleep cognitive arousal on sleep onset processes. *International Journal of Psychophysiology*. Retrieved from http://www.elsevier.com/wps/find/journaldescription.cws_home/506061

Yoo, S.-S., Hu, P. T., Gujar, N., Jolesz, F. A., & Walker, M. P. (2007). A deficit in the ability to form new human memories without sleep. *Nature Neuroscience*, *10*, 385–392. doi: 10.1038/nn1851.

Zohar, D., Tzischinsky, O., Epstein, R., & Lavie, P. (2005). The effects of sleep loss on medical residents' emotional reactions to work events: A cognitive-energy model. *Sleep*, *28*, 47–54. Retrieved from http://www.journalsleep.org/.

(Received April 6, 2011; Accepted July 7, 2011)