OXFORD

Cerebral Cortex, 2023, **33**, 96–113
https://doi.org/10.1093/cercor/bhac055
Advance access publication date 23 February 2022
Original Article

Stress dynamically reduces sleep depth: temporal proximity to the stressor is crucial

Jonas Beck [1], Erna Loretz [2], Björn Rasch [1,*]

[1]Department of Psychology, University of Fribourg, Rue P.-A-de-Faucigny 2, CH-1700 Fribourg, Switzerland,
[2]The Siesta Group Schlafanalyse GmbH, Schlosshofer Strasse 11, 1210 Vienna, Austria
*Corresponding author: Department of Psychology, University of Fribourg, Rue P.-A-de-Faucigny 2, CH-1700 Fribourg, Switzerland. Email: Bjoern.rasch@unifr.ch

The anticipation of a future stressor can increase worry and cognitive arousal and has a detrimental effect on sleep. Similarly, experiencing a stressful event directly before sleep increases physiological and cognitive arousal and impairs subsequent sleep. However, the effects of post- vs. pre-sleep stress on sleep and their temporal dynamics have never been directly compared. Here, we examined the effect of an anticipated psychosocial stressor on sleep and arousal in a 90-min daytime nap, in 33 healthy female participants compared to an anticipated within-subject relaxation task. We compared the results to an additional group ($n = 34$) performing the same tasks directly before sleep. Anticipating stress after sleep reduced slow-wave activity/beta power ratio, slow-wave sleep, sleep spindles, and slow-wave parameters, in particular during late sleep, without a concomitant increase in physiological arousal. In contrast, pre-sleep psychosocial stress deteriorated the same parameters during early sleep with a concomitant increase in physiological arousal. Our results show that presleep cognitions directly affect sleep in temporal proximity to the stressor. While physiological arousal mediates the effects of presleep stress on early sleep, we suggest that effects during late sleep originate from a repeated reactivation of mental concepts associated with the stressful event during sleep.

Key words: sleep; stress; cognition; arousal; slow-waves.

Introduction

Stress is our response to threats and challenges in order to adapt to such situations. On a physiological level, the stress response is modulated by activation of the hypothalamic pituitary adrenal (HPA) axis and the autonomic nervous system comprising sympathetic (SNS) and parasympathetic nervous system (PNS). Most stressors induce stress responses at both the physiological and cognitive levels, particularly those that involve a psychosocial component (Kogler et al. 2015). A widely used and standardized stress paradigm to induce acute psychosocial stress is the Trier Social Stress Test (TSST; Kirschbaum et al. 1993), which targets a combination of social-evaluative threat and uncontrollability (Dickerson and Kemeny 2004), reliably activates the HPA-axis and induces cognitive arousal in real and virtual reality settings (Allen et al. 2017; Zimmer et al. 2019). Research suggests that stress and stress regulatory systems have a major impact on our sleep and the development of sleep disturbances (van Reeth et al. 2000; Riemann et al. 2010; Lattova et al. 2011; Drake et al. 2017). Sleep is important for our recovery: impaired sleep is associated with several health problems such as obesity, cardiovascular diseases, cognitive impairments as well as neurodegenerative diseases (Xie et al. 2013; Fan et al. 2020; Hale et al. 2020) and has been suggested to make people more sensitive to negative social experiences (Gordon et al. 2019).

Experimental studies inducing psychosocial stress and cognitive tasks in a laboratory directly before sleep and collecting objective polysomnographic data (Wuyts et al. 2012a; Ackermann et al. 2019), reported a prolonged sleep onset latency (SOL) and a decrease in low-/high-frequency power in the electroencephalogram (EEG) during nonrapid eye movement (NREM) sleep, which is a measure of objective sleep quality (e.g. Hall et al. 2007; Maes et al. 2014; Cordi et al. 2019; Hogan et al. 2020). These changes were limited to early periods of sleep and presleep stress did not affect sleep parameters during later sleep periods. Other studies reported inconsistent results: While Vandekerckhove and colleagues observed changes in sleep architecture (but not on sleep onset, amount of slow-wave sleep [SWS] or EEG power bands) after a negative experience before sleep (Vandekerckhove et al. 2011), Kim and coauthors reported no changes on sleep architecture at all after a presleep TSST (Kim et al. 2019). Therefore, the effects of presleep stress induction on sleep seem to be smaller than expected and mostly affecting early sleep. Interestingly during wakefulness,

Received: October 7, 2021. **Revised:** January 22, 2022. **Accepted:** January 23, 2022

stress-induced changes in heart rate recovers between 10 and 30 min after the TSST induction, while cortisol recovers about 30–90 min after finishing the task (Kirschbaum et al. 1993; Janson and Rohleder 2017; Yamanaka et al. 2019). Thus, changes in sleep after presleep stress induction might be mainly related to the physiological component of stress, which covers the sleep onset and early sleep periods but vanishes throughout later periods of sleep.

These limited effects of acute stress on sleep stand in contrast to the well-documented notion that stress is a major factor causing sleep disturbances (van Reeth et al. 2000; Riemann et al. 2010; Lattova et al. 2011; Drake et al. 2017), possibly throughout the whole sleep period. In contrast to time-limited physiological responses after acute stress, anticipation of future stress preserves negative cognitive activity over a longer time period (Brosschot et al. 2006). Importantly, anticipation of future stress—also referred to as repetitive negative thinking (Watkins et al. 2005) or more generally cognitive arousal—is a main factor for the development and maintenance of sleep disturbances (Ballesio et al. 2020; Clancy et al. 2020; Kalmbach et al. 2020; Lemyre et al. 2020). Thus, Brosschot proposed that unconscious cognitive representations of anticipation of future stressful events are causing the detrimental long-term effects of stress and might also be responsible for the detrimental effect of stress on sleep (Brosschot 2010; Brosschot et al. 2018).

Several studies have already examined the effects of anticipating stressful events on sleep: Anticipating an early awakening or a stressful workday negatively affects sleep architecture (Kecklund et al. 1997; Kecklund and Åkerstedt 2004) and induces changes in stress-related hormone levels during later parts of sleep (Born et al. 1999). Anticipating a stressful speech after a nap delays sleep onset, decreases the amount of N2 sleep and increases the amount of wake (Gross and Borkovec 1982). Similar trends were observed in a night study, where subjects anticipated demanding cognitive tasks after sleep (Elder et al. 2018). Two additional studies investigated the effect of anticipating a stressful speech after sleep and did not find any effects on overall sleep parameters (Germain et al. 2003; Hall et al. 2004). However, they observed physiological changes during later periods of sleep, including the number of rapid eye-movements during REM sleep at the end of the night (Germain et al. 2003) and a constant increase of high frequency power in the electrocardiography (ECG) across successive NREM periods (Hall et al. 2004). Thus, studies examining the effect of anticipating a stressful event on sleep support the notion that changes occur during later sleep periods which are closer to the time-point when the event is expected.

In sum, the effects of stress on sleep are probably dynamic: While acute and completed stressful events before sleep (presleep stress) might mainly impact early sleep periods (possibly mediated by the time-limited occurrence of physiological stress-responses), anticipation of stressful events after sleep (postsleep stress) might mainly rely on maintained cognitive arousal and occurs during later sleep periods. To our knowledge, no study so far has directly compared the dynamic effects of direct pre- vs. anticipated post-sleep stress on sleep. Moreover, previous studies are lacking and in-depth analysis of sleep physiology by including analysis of EEG power and sleep oscillations such as slow-waves and sleep spindles. Our study aims to fill these gaps.

Due to inconsistent results of anticipating stress on sleep physiology, we aimed to investigate the effect of anticipating stress on subjective and objectively measured sleep. In addition, we examined how the effect of stress on sleep differs when stress is anticipated vs. conducted directly prior to sleep. In two counterbalanced within-subject experimental nap sessions, 67 healthy young subjects performed the TSST and a relaxation task in a virtual reality environment. In addition, half of the subjects only received the task instructions directly before sleep and anticipated to perform the tasks after sleep (postsleep), while the other half performed the tasks directly before sleep (presleep). We preregistered our hypothesis that cognitive arousal is increased in the stress condition compared with the relaxation condition and decreases objective (SOL and slow-wave activity [SWA]/beta power ratio) and subjective sleep quality in the postsleep group. Furthermore, we explored effects of stress compared with relaxation on heart rate, sleep stages and sleep parameters including an in-depth analysis of slow-waves, slow and fast spindles, and their progression across the nap. In addition, we explored effects of the type of stress (postsleep vs. presleep) on all parameters. We were particularly interested in possible differences in dynamic changes of sleep parameters and assumed that the effect of stress on sleep occurs more strongly in temporal proximity to the stressor. Thus, we hypothesized that direct stress before sleep mainly affects early sleep, whereas anticipating stress mainly affects later periods of sleep.

Materials and methods

Participants

The experiment examined the effect of a stress-inducing task compared with a relaxation task on sleep according to a within-subject design. Seventy-one healthy German- or French-speaking subjects participated in the experiment. Four subjects were excluded from all analysis due to no sleep in at least one of the naps (3 subjects) or a sleep period time lower than 3 SD below the mean (7 min; 1 subject). The final sample consisted of 67 young females (mean age = 21.84 ± 2.84 y [M ± SD], age range 18–30 y). In an additional group factor, half of the subjects ($n = 33$) anticipated the stress and the relaxation tasks after sleep (postsleep group; mean age = 22.30 ± 2.87 y [M ± SD], age range 18–30 y), while another group of subjects ($n = 34$) conducted both tasks directly before sleep

(presleep group; mean age = 21.38 ± 2.79 y [M ± SD], age range 18–30 y).

Subjects neither took any sleep influencing medication nor reported any neurological, psychiatric or sleep-related disorders and confirmed that no surgical procedures had been performed within the 3 months prior to the experiment. None of the subjects reported taking regular naps, working shift work, or having been on an intercontinental flight 6 weeks prior to the experiment. All participants were instructed to wake up before 08:00 h and not to drink alcohol or caffeine on experimental days. Subjects were compensated CHF 110 for attending all three sessions. The study was approved by the internal review board of the University of Fribourg (No. 475). Participants signed an informed consent form after an experimenter explained the study procedure and possible consequences.

Design and procedure

Subjects participated in three sessions including a 90-min nap. During a first adaptation nap, subjects were familiarized with sleeping with a polysomnographic setup (EEG, electromyography (EMG) and electrooculography (EOG)) in the laboratory environment and conducted no additional tasks. The adaptation session was followed by 2 experimental sessions and polysomnographic data were recorded during sleep in all 3 sessions. Participants arrived at the sleep laboratory between 10:30 am and 1:00 pm. Both experimental sessions took place on the same day of the week, 1 week apart, and participants completed questionnaires throughout the experiment. During the experimental sessions, either a stressful or a relaxing task was performed using a head-mounted display. The order of condition was counterbalanced across participants according to a within-subjects design. In an additional between-subject factor, half of the sample received the instruction for the tasks before sleep and performed the tasks after the nap (postsleep group), while the other half of subjects conducted the tasks directly before sleep (presleep group).

Stress and relaxation tasks in virtual reality

Tasks were conducted using an HTC VIVE PRO head-mounted display (https://www.vive.com) with 2 3.5" AMOLED displays with a resolution of 1440 x 1600 per eye (2880 x 1600 combined), 90 Hz refresh rate, 110° field-of-view and attached stereo headsets (HTC Corporation, Taoyuan, Taiwan). Tracking of the headset was achieved with 2 SteamVR Base Stations 2.0 using the runtime software SteamVR (Valve Corporation). The headset was connected to a PC running Windows 10 Enterprise (64-bit), an Intel Core i7-8700k, 64 GB RAM, NVIDIA GeForce GTX 1080 Ti and 512 GB SSD.

In the stress condition, a virtual reality version of the TSST was performed (Kirschbaum et al. 1993). The TSST is an established test method to induce acute social stress in a laboratory setting (Allen et al. 2017) and induces a similar stress response, when conducted in virtual reality (Liszio et al. 2018; Zimmer et al. 2019). During this test, subjects had to give a 5-min speech to convince a panel of 3 people why they would be the best candidate for a job position. If participants stopped their speech before the 5 min expired, an experimenter asked standard questions according to the TSST manual. Subsequently, in a second math task in front of the panel, participants were requested to count continuously backwards from 2023 in increments of 17, again for 5 min. If subjects made a mistake, they were asked by an experimenter to start again at 2023. The panel was presented using a prerecorded virtual reality video showing 3 university professors sitting behind a table. The 15-min video was played using the Skybox VR Player (https://skybox.xyz/en/). According to the to the standardized TSST protocol, subjects were asked to indicate their subjective stress level at the beginning, after the speech and after the math task. Subjects were standing throughout the task and instructions and questions during the task were provided by an experimenter located behind the subject.

In addition, participants were informed on-screen prior to the task that they would be recorded with a video camera and microphone for later behavioral and vocal analyses and that the task has been shown to successfully induce psychological and physiological stress. Moreover, they were informed that high task performance reflects good resilience and stress management and is associated with job satisfaction and career success, while low performance is associated with a higher risk for cardiovascular diseases, sleep disturbance, depression, and burnout. Additionally, they were told that they would receive results on their performance with comparative values for their age group and an additional payment of 10 CHF if they performed very well in both stress-inducing tasks. Lastly, it was pointed out that high performance is crucial for the bachelor and master theses and dissertations associated with the project.

In the relaxation condition, subjects were told to relax in a beautiful virtual reality environment, without further instructions. We used Nature treks VR (https://greenergames.net/), a relaxation game that allows the user to explore nature-based virtual reality environments such as tropical beaches, green meadows, and underwater oceans. The game was launched via Steam (Valve Corporation). First, subjects selected 2 out of 9 virtual reality environments based on a representative image of an environment in the game's menu, where they assumed to be able to relax best. Next, participants were familiarized with the motion controls using the righthand controller. To avoid motion sickness, movement was only possible via a teleport function. All other functions of the game were disabled. Subjects were instructed to search for a relaxing place within both environments. After visiting both environments, they were asked to choose the environment perceived

as more relaxing and to continue the task in this environment at the previously chosen relaxing place. At this location, the experimenter withdrew the controller and informed participants that they would now remain at this place for about 10 min and relax. No further instructions were provided and subjects were allowed to design the relaxation exercise individually. Similar to the standardized TSST procedure, subjects were asked to indicate their subjective stress level at the beginning, after 5 min and at the end of the task. Subjects sat during the task and questions were delivered by an experimenter who was located behind the subject.

In addition, participants were informed on-screen prior to the task that no video or audio recording would be made and that the task has been shown to successfully induce psychological and physiological relaxation. Moreover, they were informed that relaxation tasks are helpful for good health and lasting well-being, and that frequent practice of relaxation tasks is associated with high resilience and stress management, job satisfaction and career success, and reduces the risk of cardiovascular disease, sleep disturbances, depression and burnout. Furthermore, they were told that no other tasks would follow after the relaxation task, there will be no comparison to other subjects and that the payment would not be affected by this task.

After reading through the on-screen task instructions, subjects were given 3 min to take notes, either for their speech to the panel or on how they planned to relax in the virtual reality environment. They were informed that the notes could not be accessed at a later time. The postsleep group received the task instructions and completed taking notes on the tasks before sleep. Again, after sleep, they received the task instructions and conducted the tasks. Subjects in both groups were verbally debriefed directly after performing the stressful task and again in writing at the end of the experiment. In the presleep group, subjects were verbally debriefed directly after the task before sleep. During the verbal debriefing, subjects were provided with their best performance in the arithmetic task and were informed that no further feedback on their performance can be given, as neither a video of their performance nor their voice was recorded and analyzed. In addition, they were debriefed that all subjects receive the additional payment of CHF 10 and that subjects should not share this information with possible future participants. A written debriefing sheet, which included the same points and information about the aim of the study, was signed by each subject at the end of the experiment.

Questionnaires

During the adaptation session, subjects completed a questionnaire on general personal information (sex, age, education status, handedness, native language), the Pittsburg Sleep Questionnaire Inventory (Buysse et al. 1989), the Morningness-Eveningness-Questionnaire (Griefahn et al. 2001), the Tellegen Absorption Scale (Tellegen and Atkinson 1974), the Resilience Scale for Adults (Friborg et al. 2003), the rumination subscale of the Rumination-Reflection Questionnaire (Trapnell and Campbell 1999) and the trait-anxiety subscale of the State-trait Anxiety Inventory (Spielberger 1970). At the beginning of all three sessions, subjects were asked about their sleep the previous night, anticipation of an important or stressful task, general stress level within the past week and consumption of alcohol, caffeine and drugs. After sleep, subjects filled out the Presleep Arousal Scale (Nicassio et al. 1985) and a subjective sleep quality questionnaire (SF-A/R, Görtelmeyer 2011). The Multidimensional Mood Questionnaire (MDBF, short form A; Steyer et al. 1997) and a single question about their stress level (10 – point Likert scale from 1 = not at all to 10 = very much) was performed at the beginning of the each session, directly before sleep as well as after sleep in all 3 sessions. After the virtual reality tasks, subjects filled out the Simulator Sickness Questionnaire (Kennedy et al. 1993) and the Igroup Presence Questionnaire (Schubert et al. 2001). In addition, the single stress question was asked at the beginning, after 5 min and at the end of the virtual reality tasks. One additional measurement of the MDBF was conducted after the virtual reality tasks for participants in the postsleep group. For the purposes of this study, we focused our analysis on the Presleep Arousal Scale (PSAS) and on the SF-A/R questionnaire on subjective sleep quality.

The PSAS (Nicassio et al. 1985) was the first questionnaire completed after waking up to assess presleep cognitive and somatic arousal. Subjects rated how intensely they experienced 16 presleep symptoms during the presleep period before the nap on a 5-point likert-scale from 1 (not at all) to 5 (extremely). Eight items referred to cognitive arousal (PSAS-C; e.g. worry about falling asleep; thinking or ruminating about events of the day) and another 8 concerned somatic arousal (PSAS-S; e.g. heart racing, pounding, or beating irregularly; a cold feeling in your hands, feet or body in general). Subscale scores ranged from 8 to 40 with higher scores indicating increased cognitive or somatic arousal. Both subscales are internally consistent with a Cronbach's alpha of 0.88 for the PSAS-C and 0.79 for the PSAS-S in college students (Nicassio et al. 1985). For the PSAS-C, values > 16 (with a mean item-level response higher than "slightly") have been suggested as high cognitive arousal and values ≤ 16 as low cognitive arousal (Kalmbach et al. 2020) and increased values for insomnia patients (Vochem et al. 2019). In addition, subjects were asked on a single item, if they were worried about the announced task or about the already conducted task before sleep.

Subjective sleep quality was assessed via the sleep quality subscale of the SF-A/R (Görtelmeyer 2011) after the nap. The scale includes 4 indices indicating difficulty in initiating sleep (1 item), difficulty in maintaining sleep (2 items), early waking with inability to return to sleep (1 item), and general sleep characteristics (6 items). Scores between 1–5 indicate absent (1) or strongly distinct (5)

characteristics of good sleep quality. Cronbach's alpha with respect to the subscale sleep quality is 0.89 in healthy subjects.

Polysomnographic recording

Electroencephalographic data were recorded at F3, F4, C3, C4, P3, P4, O1, O2 and left and right mastoids using single (Ag/AgCl) electrodes with a BrainAmp amplifier (Brain Products, Gilching, Germany), a sampling rate of 500 Hz, Cz as a physical online reference and Fpz as a ground electrode. Two electrodes were placed laterally to the outer canthi of the left and right eye to collect EOG data. Two bipolar chin electrodes collected EMG data, and 2 bipolar electrodes collected electrocardiogram data. Impedances were kept below 10 kΩ for EEG, EOG, and EMG electrodes.

For sleep scoring, data were rereferenced against contralateral mastoids, and standard filter settings suggested by the AASM (Iber et al. 2007) were applied (e.g. EEG 0.3–35 Hz) with an additional notch filter (50 Hz). Data were exported in EDF format and scored by a central scoring facility following the AASM guidelines with a validated scoring algorithm and visual quality control (Anderer et al. 2010). Results were manually checked by additional scorers, who were blind to the experimental condition. In addition to sleep scoring data, the scoring algorithm also provided microstructural arousal and stage shift parameters. Sleep scoring parameters were computed using the SleepTrip toolbox (https://www.sleeptrip.org/; RRID: SCR_017318).

Preprocessing and artifact rejection

EEG data preprocessing was conducted using BrainVision Analyzer software (2.2; Brain Products, Gilching, Germany). Data were filtered using a high- (0.1 Hz) and low-pass (40 Hz) filter with an additional notch filter at 50 Hz and rereferenced to averaged mastoids. Next, data were segmented in 30 s epochs of NREM sleep based on sleep scoring results. Afterwards, data were further segmented into equally sized segments of 2048 data points (4 s, 102 points overlap). Next, an automatic artifact rejection was applied (Ackermann et al. 2015) based on the following 3 criteria: (1) the maximum difference in EMG activity < 150 μV, (2) maximum voltage step in all EEG channels < 50 μV/ms, (3) maximum difference in EEG activity < 500 μV in all EEG channels. The number of removed segments were manually checked. For analysis of oscillatory activity during sleep (power analysis, spindle detection, and slow-wave detection), artifact rejected data were exported as continuous data and further analyzed using the SleepTrip toolbox (https://www.sleeptrip.org/; RRID: SCR_017318), which is based on FieldTrip functions (http://fieldtriptoolbox.org; RRID: SCR_004849; Oostenveld et al. 2011) and the SpiSOP tool (www.spisop.org; RRID: SCR_015673).

Power analysis

To investigate differences in EEG power during sleep, we used the default settings of SleepTrip (10% segment overlap, 20% Hanning window). Mean power values (μV^2) of each channel were exported for SWA (0.5–4.5 Hz), theta activity (4.5–8 Hz), alpha activity (8–11 Hz), slow spindle activity (11–13 Hz), fast spindle activity (13–15 Hz), and beta activity (15–30 Hz) during NREM sleep. As preregistered, we computed the ratio between SWA and beta activity.

Outliers were identified based on SWA values in both studies (outlier criterion: 3 SD ± mean) and replaced with values from the contralateral electrode. If both hemispheres exceeded the outlier criterion over one lobe, we replaced the values with data from the nearest electrode. One subject was excluded from the power analysis due to SWA values exceeding the outlier criterion in all electrodes in the stress condition and 3 electrodes in the relaxation condition. Additional exploratory analysis on 5 min segments of sleep was conducted on the sleep scoring data without an artifact rejection procedure.

Slow-wave detection

Slow-wave detection was conducted with the default settings from SleepTrip, which are comparable to previously reported settings (Beck, Cordi, et al. 2021). Two parameters were adjusted compared with the previously reported setting, which are a reduced amplitude threshold for artifact detection of 600 μV (previously 1000 μV) and a decreased factor of 1.00 (previously 1.25) for the means of the amplitudes and the negative half-wave peak potential. As a result, number of slow-waves, density per 30 s epoch NREM sleep, mean amplitude, duration, down slope (value of the negative half-wave peak divided by the time from the first zero-crossing to the trough in μV/s) and up slope (absolute value of the negative half-wave peak divided by the time from the trough to the next zero-crossing in μV/s) were calculated for each participant and channel during NREM sleep.

Spindle detection

Prior to the detection of sleep spindles, individual slow and fast spindle frequency peaks were visually determined based on the NREM power spectrum of each dataset. Slow spindle peaks were determined in frontal channels (F3, F4) and fast spindle peaks in parietal channels (P3, P4) due to expected power maxima over those regions (Mölle et al. 2011). Similar to a previous nap study (Beck, Cordi, et al. 2021), average slow spindle peaks ranged between 9.5 and 13.3 Hz with an average frequency of 11.71 ± 0.81 Hz (M ± SD), and fast spindle peaks ranged between 12.5 and 15.5 Hz with an average frequency of 14.16 ± 0.45 Hz (M ± SD).

To detect sleep spindles, default settings from SleepTrip toolbox were used. They are based on algorithms used in Mölle et al. (2002) and Mölle et al. (2011) and have already been explained in detail (Beck, Cordi, et al. 2021). Two parameters were adjusted, which are an increased

minimal amplitude threshold factor of 1.75 SD (previously 1.5 SD) and a reduced maximum duration of each spindle to 2 s (previously 3 s).

Electrocardiography

ECG data were cut into the whole sleep time and 15 min segments and exported in EDF+ format using the BrainVision Analyzer software (2.2; Brain Products, Gilching, Germany). Data were further analyzed using Kubios HRV Premium 3.2.0 (Kubios Oy, Kuopio, Finnland). The software includes an automatic artifact correction based on successive RR peak intervals. Data were analyzed in the time and frequency domain and the following variables were included in our analysis: mean heart rate (as an index for physiological arousal (Kogler et al. 2015)) and the activity of the parasympathetic (PNS-index, based on mean RR peak intervals) as well as sympathetic nervous system (SNS-index, based on mean heart rate) to assess physiological stress.

Statistical analysis

Statistical analyses were performed using Rstudio version 1.1.456 (R Core Team 2018). To examine whether anticipating a stress or relaxation task after sleep affects arousal and sleep differently than performing a stress or relaxation task before sleep, we performed a mixed-design analysis of variance (ANOVA) containing the within-subject factor condition (stress vs. relaxation) and the between-subject factor group (postsleep vs. presleep). First, we analyzed if the group factor affected the objective arousal (ECG) and subjectively rated cognitive (PSAS-C) and somatic presleep arousal (PSAS-S). Second, we analyzed effects on subjective sleep quality (subscale SQ, SOL) and objective sleep quality (SOL, SWA/beta power ratio). Next, we explored effects on sleep architecture (SWS, N2, N1, REM, WASO) and sleep oscillations (sleep spindles and slow-wave parameters). In addition, we explored the progression over the nap in 5-min segments for SWA/beta power ratio, SWS, slow-wave parameters and sleep spindle parameters by adding the within-subject factor time (0, 5, 10, … 80 min). We further explored the progression of the heart rate across sleep in 15-min time segments. In case of statistically significant results, effect sizes are reported with partial eta squared (η_p) for main effects (ME) and interactions and Cohen's d for t-tests. Posthoc tests for significant interactions comprised paired Student's t-tests and Welch's t-test. If the assumption of sphericity was violated, Greenhouse–Geisser corrected P-values are reported. Data are presented as means ± standard error. The level of significance was set at $P < 0.05$ (two-tailed).

Preregistration

We preregistered the postsleep group of this study, which can be viewed via the following link: https://osf.io/rf6tc.

Results

Presleep subjective arousal

The analysis on cognitive arousal revealed a ME of condition (stress vs. relaxation; $F_{1,65} = 10.79$, $P = 0.002$, $\eta_p = 0.14$), but neither a significant interaction with the time of the intervention (postsleep vs. presleep, $F_{1,65} = 1.08$, $P > 0.30$) nor a ME of this group factor ($F_{1,65} = 1.00$, $P > 0.30$). Thus, both the anticipation of postsleep stress and real presleep stress increase cognitive arousal to a similar extent compared with the relaxation condition (see Fig. 1C). In contrast, we observed a significant interaction between condition and the time of intervention for somatic arousal ($F_{1,65} = 6.44$, $P = 0.014$, $\eta_p = 0.09$; see Fig. 1B): While somatic arousal was rated comparably between both conditions in the postsleep group ($t_{32} = -0.13$, $P > 0.90$), subjects reported significantly higher somatic arousal in the presleep group after stress compared with the relaxation condition ($t_{33} = -3.09$, $P = 0.004$, $d = 0.53$). Moreover, the result that anticipating postsleep stress selectively increased cognitive but not somatic arousal was further supported by an additional analysis in the postsleep group, which yielded an interaction between the 2 arousal scales and the experimental condition ($F_{1,32} = 4.19$, $P = 0.049$, $\eta_p = 0.12$).

Objective arousal

The findings for subjective somatic arousal were supported by heart rate averaged over the whole the nap period. In the postsleep group, which anticipated stress or relaxation task after the sleep period, the mean heart rate (beats per minute, bpm) during sleep was comparable between the stress (66.36 ± 1.73 bpm) and the relaxation conditions (65.77 ± 1.79 bpm; $t_{32} = -0.42$, $P > 0.60$). In contrast, mean heart rate was increased in the stress (63.72 ± 1.94 bpm) compared with the relaxation condition in the presleep group (60.96 ± 1.78 bpm; $t_{33} = -3.50$, $P = 0.001$, $d = 0.60$). The interaction between condition and group (i.e. timing of the intervention) was significant ($F_{1,65} = 4.39$, $P = 0.040$, $\eta_p = 0.06$). Exploratory analysis on the progression of heart rate across the nap period revealed that the differences between the stress and relaxation condition in the presleep group were only present during the first half of the nap, whereas later time periods did not differ (interaction condition × time in the presleep group: $F_{5,165} = 5.18$, $P = 0.002$, $\eta_p = 0.14$; see Fig. 1D, right plot). This was further supported by a significantly increased SNS index and decreased PNS index only during the first half of the nap in the stress compared with the relaxation condition in the presleep group (interaction condition × time: SNS: $F_{5,165} = 4.29$, $P = 0.004$, $\eta_p = 0.12$; PNS: $F_{5,165} = 2.20$, $P = 0.10$). In contrast, the heart rate, SNS and PNS indices in the postsleep group were comparable between the stress and relaxation condition at all timepoints (interaction condition × time: heart rate: $F_{5,160} = 1.05$, $P = 0.37$; SNS:

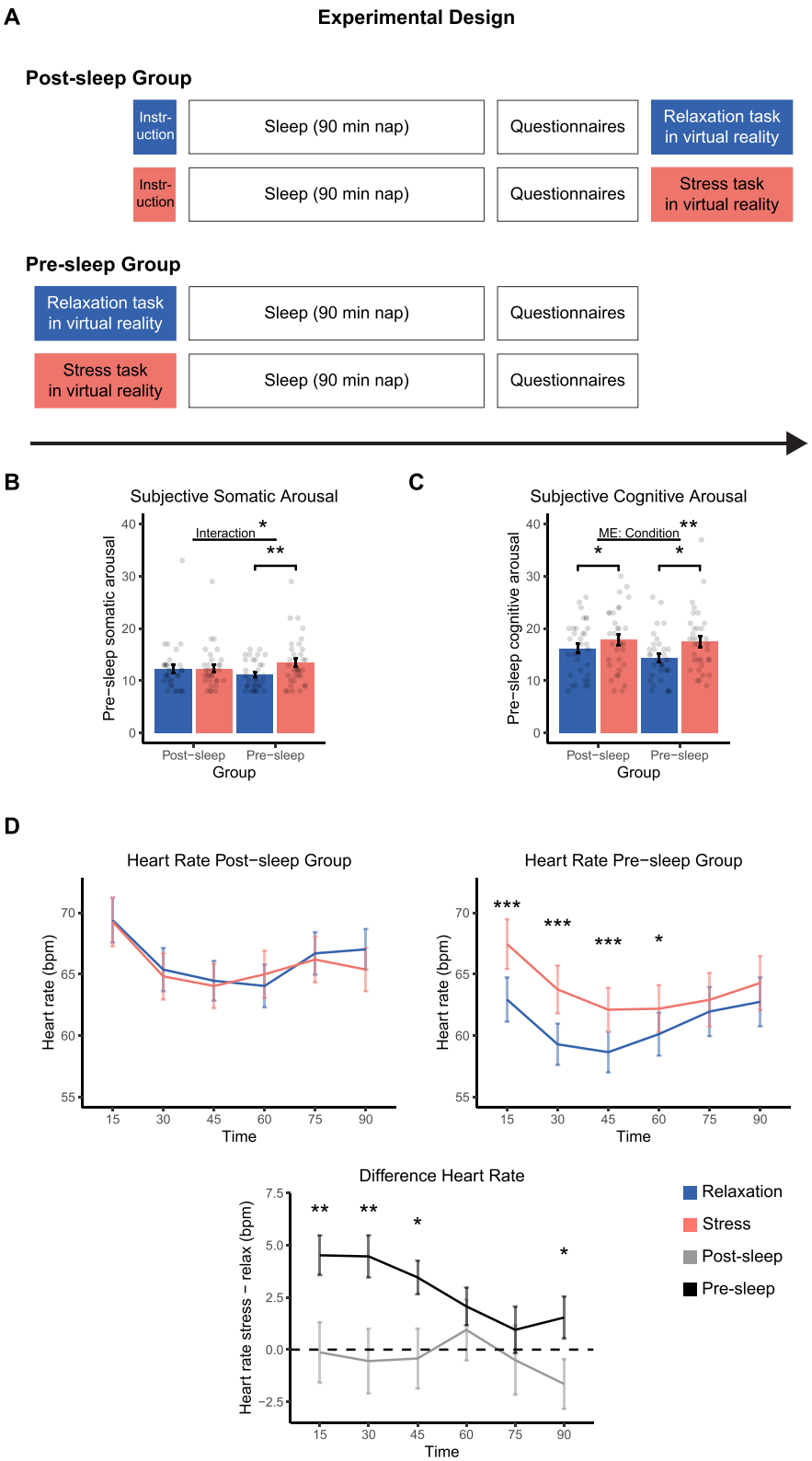

Fig. 1. Experimental design and results of subjective and objective arousal. (A) Sixty-seven healthy participants either anticipated a stress or a relaxation task after sleep (postsleep, $n = 33$) or conducted the tasks before sleep (presleep group, $n = 34$). In the stress condition, a virtual reality version of the TSST was performed, while in the relaxation task, subjects were told to relax in a beautiful virtual reality environment without further instructions. (B) Subjective presleep somatic arousal was comparable between the stress and the relaxation condition in the postsleep group. In contrast, subjective somatic presleep arousal was increased in the stress compared with the relaxation condition in the presleep group. These differences between groups were supported by a significant interaction between condition and group (interaction; $F_{1,65} = 6.44$, $P = 0.014$, $\eta_p = 0.09$). (C) Subjective cognitive presleep arousal was increased in the stress compared with the relaxation condition in both groups (ME: condition; $F_{1,65} = 10.79$, $P = 0.002$, $\eta_p = 0.14$). (D) in the postsleep group, objective physiological arousal (heart rate, bpm) was comparable between the stress and the relaxation condition across the sleep period (D, left plot). In the presleep group, objective physiological arousal was increased in the stress compared with the relaxation condition during the first hour of sleep (D, right plot). Changes in heart rate from the relaxation to the stress condition in the presleep group mainly differed during early sleep from the postsleep (D, bottom plot). Values are displayed as mean ± SEM. $^{***}P < 0.001$, $^{**}P < 0.01$, $^{*}P < 0.05$.

$F_{5,160} = 0.61$, $P > 0.60$; PNS: $F_{5,160} = 1.10$, $P > 0.30$; see Fig. 1D, left plot).

In addition, exploratory analysis of the data in the adaptation nap yielded that heart rate in the presleep group was comparable at all time-points with the heart rate in the relaxation condition (see Supplementary Fig. S2B). Thus, the differences in heart rate between conditions in the presleep group are likely to be driven by an increased heart rate in the stress condition. In the postsleep group, anticipation of a stress ($t_{32} = -2.24$, $P = 0.033$, $d = 0.39$) and of a relaxation task ($t_{32} = -2.15$, $P = 0.039$, $d = 0.37$) similarly increased the heart rate in comparison with the adaptation nap during early sleep (timepoint 30 min; see Supplementary Fig. S2A). Interestingly, heart rate in the adaptation nap was statistically comparable between groups at the beginning (timepoint 15 min; $t_{61.41} = 1.14$, $P > 0.20$) and averaged over the whole nap period (ME group: $F_{1,65} = 2.01$, $P = 0.16$). Therefore, anticipation of a task might generally increase objective arousal levels during early sleep.

Subjective sleep quality

Ratings of subjective sleep quality were not strongly affected by the stress vs. relaxation condition, irrespective of whether the intervention occurred before sleep or was only anticipated. We observed neither a significant ME of condition ($F_{1,65} = 2.05$, $P = 0.16$), of group ($F_{1,65} = 0.24$, $P > 0.60$) nor an interaction between both factors ($F_{1,65} = 1.85$, $P = 0.18$; see Fig. 2B). Exploratory posthoc tests yielded a trend for an increased sleep quality in the presleep group in the relaxation compared with the stress condition ($t_{33} = 1.86$, $P = 0.07$, $d = 0.32$). For subjective SOL, we observed a significant ME of condition over both studies ($F_{1,61} = 5.26$, $P = 0.025$, $\eta_p = 0.08$; Fig. 2A). Four subjects had to be excluded due to values larger than 3 SD of the mean. The increase in subjective SOL in the stress compared to the relaxation condition occurred similarly in the postsleep and the presleep group, as we neither observed a ME of the group factor ($F_{1,61} = 0.73$, $P > 0.40$) nor an interaction between both factors ($F_{1,61} = 1.85$, $P > 0.60$).

Objective sleep quality

Similar to subjective SOL ratings, stress increased objective SOL in both groups from 11.75 ± 1.17 min in the relaxation condition to 18.87 ± 1.87 min in the stress condition (ME of condition: $F_{1,65} = 17.99$, $P < 0.001$, $\eta_p = 0.22$; see Fig. 2C). We did not observe a ME of group ($F_{1,65} = 0.12$, $P > 0.70$), however, the increase in objective SOL tended to be larger in the presleep group compared with the postsleep group (interaction condition × group: $F_{1,65} = 3.82$, $P = 0.06$, $\eta_p = 0.06$). For objective sleep quality measured by the SWA/beta power ratio, a ME of group suggested a generally decreased sleep quality in the presleep group (226.25 ± 22.46) compared with the postsleep group (373.64 ± 42.21; $F_{1,64} = 9.82$, $P = 0.003$, $\eta_p = 0.13$). One subject had to be excluded in this analysis due to values larger than 3 SD of the mean. In addition, we also observed a significant ME of condition ($F_{1,64} = 5.30$, $P = 0.025$, $\eta_p = 0.08$). The SWA/beta power ratio was decreased in the stress condition (263.28 ± 24.52) compared with the relaxation condition (332.15 ± 33.22; see Fig. 2D). Moreover, the SWA/beta power ratio was equally decreased by anticipated and direct stress before the nap, as we observed no significant interaction between the condition and group factor ($F_{1,64} = 0.40$, $P > 0.50$). Interestingly, beta power was increased in the presleep group compared with the postsleep group ($F_{1,64} = 13.51$, $P < 0.001$, $\eta_p = 0.17$) and comparable between the stress and the relaxation condition in both groups (ME condition: $F_{1,64} = 0.06$, $P > 0.80$; interaction condition × group: $F_{1,64} = 0.79$, $P > 0.30$; see Supplementary Fig. S1A).

Sleep architecture, slow-waves, and sleep spindles

Anticipated postsleep stress and direct presleep stress had also similar effects on sleep architecture. Participants spent less time in SWS in the stress condition (13.07 ± 1.50 min) compared with the relaxation condition (17.57 ± 1.78 min; ME of condition: $F_{1,65} = 7.54$, $P = 0.008$, $\eta_p = 0.10$; Fig. 2E). This decrease occurred similarly in the postsleep and the presleep group (interaction condition × group: $F_{1,65} = 0.04$, $P > 0.80$; see Table 1). A similar results pattern with a ME of condition was observed for sleep efficiency ($F_{1,65} = 12.16$, $P < 0.001$, $\eta_p = 0.16$) and total sleep time (TST, $F_{1,65} = 11.73$, $P = 0.001$, $\eta_p = 0.15$). Trends for ME of condition without an interaction with the group factor were also observed for wake after sleep onset (WASO; $F_{1,65} = 3.70$, $P = 0.06$, $\eta_p = 0.05$) and sleep stage N2 ($F_{1,65} = 3.53$, $P = 0.07$, $\eta_p = 0.05$). In contrast, N1 and REM sleep remained unaffected by our manipulation (see Table 1). In addition, a ME of group suggested an increased duration of N2 sleep in the presleep group compared with the postsleep condition ($F_{1,65} = 4.62$, $P = 0.036$), but we did not observe a ME of the factor group for TST, WASO, N1, SWS, REM, and sleep efficiency (all $P > 0.16$).

To further explore whether certain characteristics of slow-waves might have responded differently to anticipation vs. real presleep stress (in spite of the comparable effects in SWS and SWA/beta power ratio), we analyzed various parameters of single slow-waves during NREM sleep (count, density, amplitude, up- and down-slope, duration). Significant MEs of condition (stress vs. relax) independent of the timing of the intervention (pre- vs. post-sleep group) were observed for the number of slow-waves ($F_{1,65} = 11.88$, $P = 0.001$, $\eta_p = 0.15$, see Fig. 2F), the density of slow-waves ($F_{1,65} = 4.77$, $P = 0.032$, $\eta_p = 0.07$), and the down-slope of slow-waves ($F_{1,65} = 4.08$, $P = 0.048$, $\eta_p = 0.06$). The amplitude of slow-waves revealed a statistical trend for the factor condition ($F_{1,65} = 3.78$, $P = 0.06$, $\eta_p = 0.05$), while the up-slope ($F_{1,65} = 0.01$, $P > 0.90$), mean frequency ($F_{1,65} = 0.44$, $P > 0.50$) and duration ($F_{1,65} = 0.36$, $P > 0.50$)

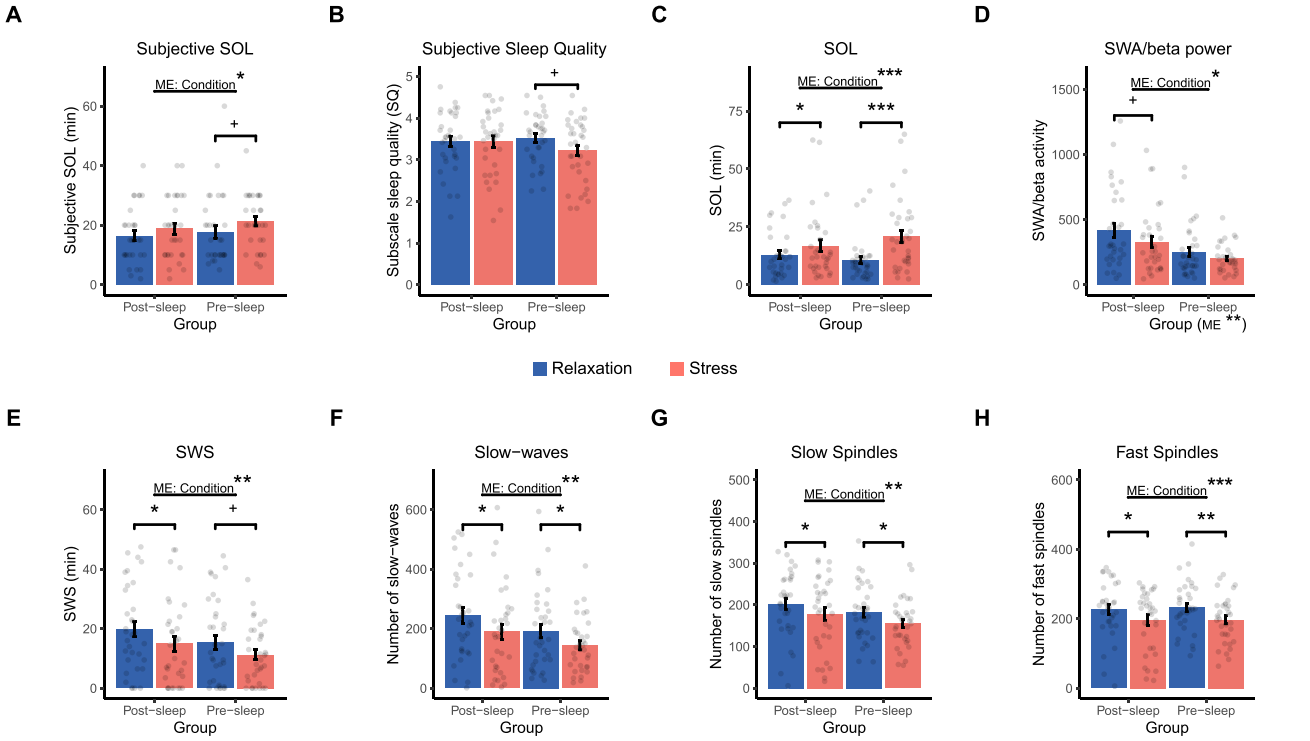

Fig. 2. Effect of anticipated postsleep stress vs. direct presleep stress on sleep. Data are shown for the relaxation (blue bars) and the stress condition (red bars) separately for the postsleep group anticipating the tasks after sleep and the presleep group conducting the tasks before sleep. Significant MEs of the factor condition (relaxation vs. stress) are displayed at the top of each graph as ME: Condition. Significant MEs of the factor group are indicated at the bottom of each graph as group (ME). We did not observe any significant interactions between both factors. Significant ME of condition suggested an increased subjective (A) and objective (C) SOL in the stress condition, while subjective sleep quality was comparable between conditions (B). In addition, the ratio between SWA and beta power (SWA/beta power, D), the amount of SWS (E) and the number of slow-waves (F), slow spindles (G) and fast spindles (H) was similarly decreased in the stress condition of the postsleep and the presleep group. Values are displayed as mean ± SEM. $^{***}P < 0.001$, $^{**}P < 0.01$, $^{*}P < 0.05$, $^{+}P < 0.10$.

Table 1. Sleep parameters in the stress and relaxation condition in the postsleep and presleep group.

Parameter	Postsleep relaxation	Postsleep stress	Presleep relaxation	Presleep stress	*P*-value main effect condition	*P*-value interaction
TST	68.30 ± 3.16	63.67 ± 3.47	**70.82 ± 2.72**	**60.49 ± 3.30**[a]	**0.001**[a]	0.20
WASO	**3.72 ± 0.84**	**7.18 ± 1.89**[b]	3.40 ± 0.66	3.88 ± 1.12	**0.06**[b]	0.14
N1	11.26 ± 1.35	13.14 ± 1.64	11.34 ± 1.06	11.04 ± 1.02	0.36	0.21
N2	28.50 ± 2.06	26.50 ± 2.21	**34.62 ± 1.91**	**31.31 ± 2.04**[b]	**0.07**[b]	0.64
SWS	**19.80 ± 2.56**	**14.97 ± 2.52**[a]	**15.40 ± 2.44**	**11.24 ± 1.65**[b]	**0.008**[a]	0.84
REM	8.74 ± 1.42	9.06 ± 1.38	9.47 ± 1.65	6.90 ± 1.43	0.37	0.25
Sleep efficiency	76.45 ± 3.54	71.11 ± 3.88	**79.11 ± 3.01**	**67.50 ± 3.69**[a]	<**0.001**[a]	0.20

Note: Means in minutes ± SEM for total sleep time (TST), wake after sleep onset (WASO), sleep stage N1 and N2, slow-wave sleep (SWS), rapid eye movement (REM) sleep, and sleep efficiency (time asleep/time-in-bed * 100). Bold values indicate significant differences between the relaxation and stress condition with [a]$P < 0.05$ and [b]$P < 0.10$.

of slow-waves were comparable between conditions. In addition, a ME of group indicated a higher density of slow-waves in the postsleep group (2.33 ± 0.17), which anticipated the tasks after sleep, compared with the presleep group (1.88 ± 0.12; $F_{1,65} = 4.80$, $P = 0.032$, $\eta_p = 0.07$). However, both groups showed a comparable number ($F_{1,65} = 2.66$, $P = 0.11$), amplitude ($F_{1,65} = 0.18$, $P > 0.60$), down-slope ($F_{1,65} = 0.17$, $P > 0.60$), up-slope ($F_{1,65} = 0.39$, $P > 0.50$), mean frequency ($F_{1,65} = 1.99$, $P = 0.16$) and duration ($F_{1,65} = 1.93$, $P = 0.17$) of slow-waves. Moreover, we did not observe an interaction between the factors condition and group for any of the slow-wave parameters (all $P > 0.40$).

Moreover, we observed a similar results pattern for slow and fast spindles. The number of both frontal slow and parietal fast spindles was significantly decreased in the stress condition compared to the relaxation condition (ME condition, slow spindles: $F_{1,65} = 10.36$, $P = 0.002$, $\eta_p = 0.14$; fast spindles: $F_{1,65} = 14.87$, $P < 0.001$, $\eta_p = 0.19$; see Fig. 2G and H). In addition, a trend for a ME of condition was also observed for frontal slow spindle density ($F_{1,65} = 5.34$, $P = 0.08$, $\eta_p = 0.05$). The density of fast spindles ($F_{1,65} = 0.66$, $P > 0.40$), amplitude of slow and fast spindles (slow: $F_{1,65} = 0.15$, $P > 0.70$; fast: $F_{1,65} = 1.46$, $P > 0.20$), duration of slow and fast spindles (slow: $F_{1,65} = 1.43$, $P > 0.20$; fast: $F_{1,65} = 1.86$, $P = 0.18$) and the frequency of

slow and fast spindles (slow: $F_{1,65} = 1.53$, $P > 0.20$; fast: $F_{1,65} = 2.33$, $P = 0.13$) was comparable between the stress and the relaxation condition. We did not observe a ME of group for the number of slow and fast spindles (slow: $F_{1,65} = 1.83$, $P = 0.18$; fast: $F_{1,65} = 0.04$, $P > 0.80$), density of fast spindles ($F_{1,65} = 0.06$, $P > 0.80$) and the duration of slow and fast spindles (slow: $F_{1,65} = 1.47$, $P > 0.20$; fast: $F_{1,65} = 0.49$, $P > 0.40$). ME of group yielded that subjects anticipating a task in the postsleep group showed an increased density of slow spindles ($F_{1,65} = 5.34$, $P = 0.024$, $\eta_p = 0.08$), a decreased amplitude of slow and fast spindles (slow: $F_{1,65} = 5.86$, $P = 0.018$, $\eta_p = 0.08$; fast: $F_{1,65} = 3.43$, $P = 0.07$, $\eta_p = 0.05$), an increased frequency of slow spindles ($F_{1,65} = 4.65$, $P = 0.035$, $\eta_p = 0.07$) and a decreased frequency of fast spindles ($F_{1,65} = 3.10$, $P = 0.08$, $\eta_p = 0.05$) compared with the presleep group. In addition, the frequency of fast spindles tended to be increased in the stress compared with the relaxation condition only in the postsleep group ($t_{32} = 1.85$, $P = 0.07$, $d = 0.32$), while conditions where comparable in the presleep group ($t_{33} = 0.23$, $P > 0.80$; interaction condition × group: $F_{1,65} = 3.05$, $P = 0.09$, $\eta_p = 0.04$). We did not observe an interaction between condition and the group factor (i.e. the timing of the intervention) for any other slow and fast spindle parameter (all $P > 0.20$). Therefore, the anticipation of stress and real presleep stress appear to affect overall slow-wave, slow spindle, and fast spindle parameters in a comparable manner.

Dynamic changes of SWA/beta power ratio, slow-waves, and sleep spindles

Our previous analyses have almost uniformly shown that anticipating a stressful task after sleep and performing the stressful task before sleep have comparable effects on objective sleep parameters. However, direct stress before sleep increases subjective somatic arousal and dynamically affects heart rate mainly during the first half of sleep (Fig. 1D), whereas anticipation of postsleep stress does not change heart rate and subjective ratings of somatic arousal. Thus, we examined whether the effect of stress on sleep could show different dynamics of sleep parameters in the postsleep stress group (only cognitive arousal) compared with the presleep stress group (somatic and cognitive arousal).

We explored the progression of SWA/beta power ratio, SWS, SWA, beta power, slow-wave parameters and the number of slow spindles and fast spindles over the course of the nap, focusing on differences between the postsleep and presleep group. Over all these parameters, we observed a consistent pattern: Performing the stressful task before sleep resulted in a deterioration of sleep parameters in the first half of the nap, while anticipating the stressful task after sleep resulted in a deterioration of sleep parameters during the second half of the nap compared with the relaxation condition. This pattern was most pronounced for slow-wave parameters (Fig. 4A–D) and spindles (Fig. 3B and C) and supported by significant interactions between the factors condition (stress vs. relaxation), group (postsleep vs. presleep) and time (0, 5, 10, …, 80 min) for the number of slow-waves ($F_{16,1040} = 2.78$, $P = 0.029$, $\eta_p = 0.04$), slow-wave density ($F_{16,1040} = 2.83$, $P = 0.025$, $\eta_p = 0.04$), amplitude of slow-waves ($F_{16,1040} = 4.12$, $P < 0.001$, $\eta_p = 0.06$), down-slope of slow-waves ($F_{16,1040} = 3.69$, $P = 0.001$, $\eta_p = 0.05$), up-slope of slow-waves ($F_{16,1040} = 3.03$, $P = 0.003$, $\eta_p = 0.04$), number of frontal slow spindles ($F_{16,1040} = 3.82$, $P = 0.001$, $\eta_p = 0.06$) and number of parietal fast spindles ($F_{16,1040} = 3.75$, $P = 0.001$, $\eta_p = 0.05$). The same interaction did not reach significance for SWA/beta power ratio ($F_{16,1040} = 1.73$, $P = 0.15$, see Fig. 3A), SWA ($F_{16,1040} = 2.10$, $P = 0.06$, $\eta_p = 0.03$) and SWS ($F_{16,1040} = 3.75$, $P = 0.07$, $\eta_p = 0.05$), however, the progression pattern remained the same: Anticipating stress after sleep reduced SWA/beta power ratio, SWA and SWS at the end of the nap (postsleep group), while performing the tasks before sleep resulted in decreased SWA/beta power ratio, SWA and SWS in the first half of the nap (presleep group). Interestingly, beta power was comparable between the stress and relaxation condition during late sleep in the postsleep group (see Supplementary Fig. S1B) and even decreased during early sleep in the stress compared with the relaxation condition in the presleep group (see Supplementary Fig. S1C).

Discussion

The current study confirms previous findings that anticipation of psychosocial stress negatively affects objective sleep parameters. In addition, to our knowledge this study is the first study directly assessing systematic differences between the effect of anticipated vs. presleep stress on both cognitive and physiological arousal as well as on sleep. Anticipation of a psychosocial stressor increased cognitive arousal without a concomitant increase in subjective and objective somatic arousal. Moreover, the anticipation of stress reduced the SWA/beta power ratio, SWS, number of spindles and slow-wave parameters during late sleep. In contrast, presleep psychosocial stress increased subjective and objective somatic arousal in addition to subjective cognitive arousal. This increase in somatic arousal mainly occurred during early sleep and in conjunction with a deterioration of sleep parameters.

Our study suggests that anticipating a psychosocial stressor after sleep overall deteriorates objective measures of sleep such as the SWA/beta power ratio, SWS, the number of slow-waves, slow and fast spindles, and SOL. These results are in line with previous research showing that anticipation of stress negatively affects SWS (Kecklund and Åkerstedt 2004), decreases low/high EEG power (Wuyts et al. 2012b) and tends to decrease sleep efficiency and TST and increase WASO (Elder et al. 2018). In addition, subjectively as well as objectively measured physiological arousal was comparable between the

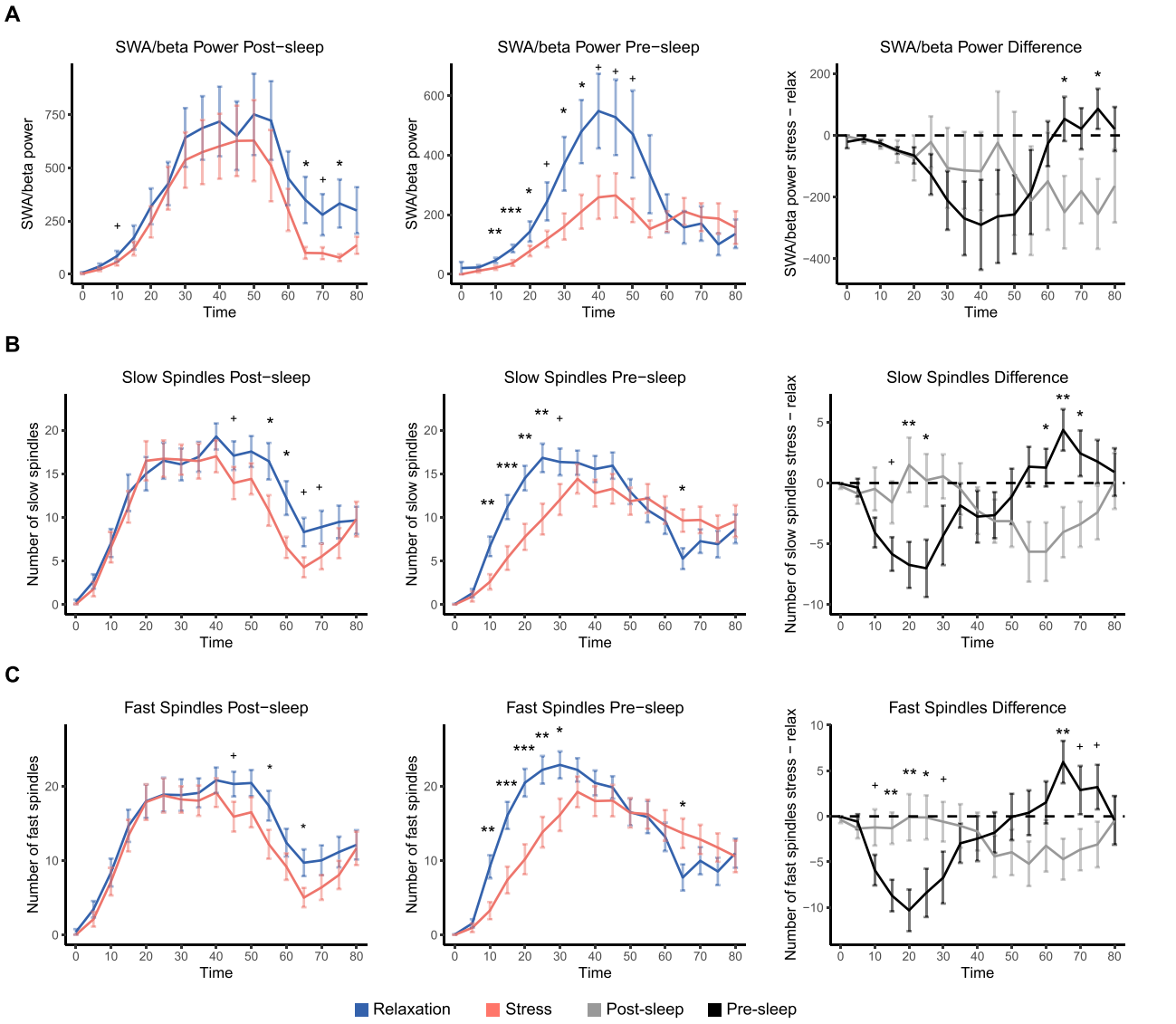

Fig. 3. Dynamic changes of the ratio between SWA and beta power and of sleep spindles across the nap. Data are shown as mean ± SEM averaged over 5-min time segments for the relaxation condition (blue line) and the stress condition (red line). The left column displays the data from the postsleep group anticipating the tasks after sleep. The middle column displays the data from the presleep group conducting the tasks before sleep. Differences between the stress and the relaxation condition are shown separately for the postsleep (gray line) and the presleep group (black line) in the right column. Performing the stressful task before sleep resulted in a decrease in the ratio between SWA and beta power, number of slow-spindles and number of fast spindles in the first half of the nap in the presleep group (A–C, middle and right column), while anticipating the stressful task after sleep resulted in a decrease of these parameters during the second half of the nap (A–C, left and right column). Dynamic differences between groups were supported by significant interactions between the factors condition (stress vs. relaxation), group (postsleep vs. presleep) and time (0, 5, 10, . . . , 80 min) for the number of frontal slow spindles ($F_{16,1040} = 3.82$, $P = 0.001$, $\eta_p = 0.06$) and number of parietal fast spindles ($F_{16,1040} = 3.75$, $P = 0.001$, $\eta_p = 0.05$). $^{***}P < 0.001$, $^{**}P < 0.01$, $^{*}P < 0.05$, $^{+}P < 0.10$.

stress and the relaxation conditions in the postsleep anticipation group, while cognitive arousal was increased. These results show that cognitive arousal without a concomitant physiological arousal is sufficient to disturb sleep and thereby suggests a direct effect of anticipated psychosocial stress on sleep physiology. This is in line with results showing an effect of anticipating a psychosocial stressor on sleep physiology, while physiological arousal measures could not account for these changes (Gross and Borkovec 1982). In addition, the overall effect on sleep parameters in the postsleep group were similar in comparison with the group conducting the same stressful task directly before sleep, which highlights the importance of future stressors for the effect of stress on sleep.

Such detrimental effects of the anticipation of a stressor on sleep are in line with findings in insomnia literature. The anticipation of a stressor is closely linked to the construct of worry, and more generally to cognitive arousal, which was increased in both groups of our study. Cognitive arousal is a decisive factor in the development and maintenance of sleep disturbances (Ballesio et al. 2020; Clancy et al. 2020; Kalmbach et al. 2020; Lemyre et al. 2020) and increased in insomnia

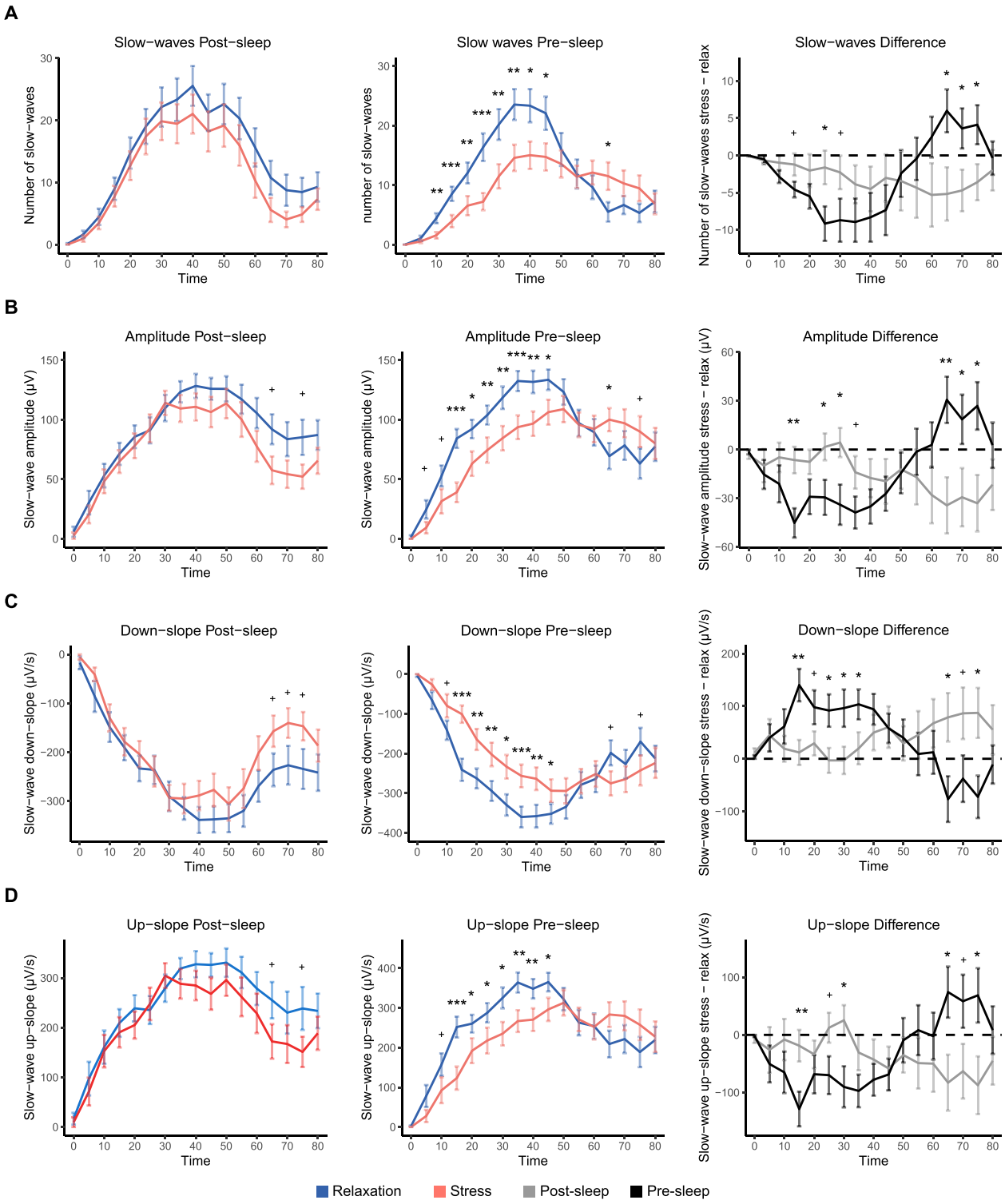

Fig. 4. Dynamic changes of slow-wave parameters across the nap. Data are shown as mean ± SEM averaged over 5-min time segments for the relaxation condition (blue line) and the stress condition (red line). The left column displays the data from the postsleep group anticipating the tasks after sleep. The middle column displays the data from the presleep group conducting the tasks before sleep. Differences between the stress and the relaxation condition are shown separately for the postsleep (gray line) and the presleep group (black line) in the right column. Performing the stressful task before sleep resulted in a decreased number of slow-waves, amplitude, down-slope, and up-slope in the first half of the nap in the presleep group (A–D, middle and right column). Anticipating the stressful task after sleep (postsleep group) resulted in a decrease of these parameters during the second half of the nap (A–D, left and right column). Dynamic differences between groups were supported by significant interactions between the factors condition (stress vs. relaxation), group (postsleep vs. presleep) and time (0, 5, 10, …, 80 min) for all four slow-wave parameters (all $P < 0.029$). $^{***}P < 0.001$, $^{**}P < 0.01$, $^{*}P < 0.05$, $^{+}P < 0.10$.

patients compared with healthy controls (Spiegelhalder et al. 2012), especially while falling asleep (Harvey 2000; Jansson-Fröjmark and Norell-Clarke 2012; Hantsoo et al. 2013; Kalmbach et al. 2020). It has been shown to mediate the effect of stress on sleep (Tousignant et al. 2019) and is a key component in insomnia models (Espie 2002; Harvey 2002; Riemann et al. 2010; Ong et al. 2012). Cognitive arousal has been associated with subjective sleep quality and difficulties falling asleep (Zoccola et al. 2009; Takano et al. 2012; Pillai et al. 2014), with objective sleep disturbances as well as physiological hyperarousal during the day and night (Kalmbach et al. 2020). In line with these results, reducing cognitive arousal with cognitive behavioral therapy for insomnia (CBT-I) has been linked to decreased post-treatment depression and anxiety symptoms (Ballesio et al. 2020). In addition, subjective data suggests that cognitive presleep arousal predicts the effect of stress on sleep to a greater extent as compared to somatic arousal (Tousignant et al. 2019).

In contrast to our findings, several previous studies which experimentally induced anticipated stress before sleep, failed to show effects of anticipated stress on sleep depth (Gross and Borkovec 1982; Germain et al. 2003; Hall et al. 2004; Elder et al. 2018). This might be explained by a failure to induce sufficient stress (Elder et al. 2018) or featuring an underpowered design to detect effects on overall sleep ($n = 15$, within subjects comparison (Born et al. 1999); $n = 40$, between subjects (Elder et al. 2018); $n = 63$, between subjects comparison (Germain et al. 2003); $n = 59$, between subjects comparison (Hall et al. 2004)). Moreover, a between subjects control group together with the lack of an adaptation session (Gross and Borkovec 1982; Germain et al. 2003; Hall et al. 2004) could have resulted in a generally elevated stress level and changes in sleep physiology in both groups (e.g. a decreased amount of SWS, increased WASO and SOL) due to the first-night effect (Agnew et al. 1966; Le Bon et al. 2000; Lee et al. 2016). Thus, the negative effect of anticipatory stress in a laboratory setting on sleep might have been superimposed by this effect. In addition, none of the studies conducted an in-depth sleep analysis on slow-waves and sleep spindles and analyzed the progression of sleep parameters across sleep. Thereby, these studies could have missed dynamic changes during sleep, which might have been too small to affect overall sleep parameters.

Strikingly, we found differences in the course of various sleep parameters across the nap between the group anticipating the stressor after sleep and the group experiencing the stressor before sleep. Differences between groups were observed for SWA/beta power ratio, SWS, slow and fast spindles and various slow-wave parameters such as the number, amplitude, up- and down-slope of slow-waves. The presleep group displayed a result pattern similar to previous studies, with presleep stress mainly affecting early sleep (Vandekerckhove et al. 2011; Wuyts et al. 2012a; Ackermann et al. 2019). This was accompanied by an increase in subjective as well as objective physiological arousal in our study. Therefore, effects of presleep stress on sleep could be caused by an increased physiological arousal level in response to the stressor, which would be incompatible with sleep. In this scenario, cognitive arousal, which was also increased in the presleep group, could have further fueled increases in physiological arousal and thereby affect sleep. This would be in line with early insomnia models assuming that the effect of psychosocial stress and cognitive arousal is mediated by an increase in physiological arousal (Spielman et al. 1987; Morin 1993; Perlis et al. 1997). Such an explanation would also fit with later insomnia models, which highlight an interaction between both types of arousal in their effect on sleep (Espie 2002; Harvey 2002; Espie et al. 2006; Riemann et al. 2010). The idea of an interaction between both arousal types is also in line with previous research showing that later bedtimes in response to a social rejection task occur most strongly for high trait ruminators and are associated with physiological arousal (Gordon et al. 2019). In addition, this approach could also explain, why the effect of presleep stress fades after about 45–50 min, when the physiological stress response to the TSST also starts to decline (Kirschbaum et al. 1993; Janson and Rohleder 2017; Yamanaka et al. 2019) and vanishes during late sleep. The decline of physiological arousal could have been enhanced by the extensive debriefing of the subjects after the stressful task to not expect any further stressful events within the study. This might also explain why a study applying an additional memory task in between the TSST and sleep did not find any effect of the stressful task on overall sleep parameters (Kim et al. 2019). The physiological stress response might have already declined or been too small to affect sleep at all.

In the postsleep anticipation group, we only found minor effects on early sleep with an increased SOL. Strikingly, after about 30–50 min asleep, anticipating a stressor after the nap started to gradually deteriorate sleep with the largest differences in the second half of the nap that means closer to the stressor. These results are in line with previous research showing that anticipation of a stressor after sleep affects sleep physiology during late sleep (Born et al. 1999; Germain et al. 2003; Hall et al. 2004). However, these studies only reported changes of specific physiological measures such as high frequency ECG power, blood ACTH concentration, and changes in the number of rapid eye-movements. In addition, they did not report any changes in sleep parameters and also lacked an in-depth analysis of the progression of sleep parameters across sleep. Previously mentioned methodological issues and the lack of in-depth sleep analysis might be responsible for missing effects of anticipated stress on sleep parameters in these studies. In contrast, based on ECG measures we did not find an effect of anticipated stress on physiological arousal throughout sleep in our study. Therefore, our results suggest a direct

effect of anticipatory stress on sleep physiology without a mediation by physiological arousal.

Given the changes in the progression of sleep parameters across the nap, the question of how the anticipation of a stressor can affect sleep over an hour after having fallen asleep arises. In recent stress models, Brosschot and colleagues assume that a large part of the detrimental and prolonged effect of stressors on health and sleep is caused by unconscious perseverative cognition, for example unconsciously worrying about a future stressor (Brosschot et al. 2007; Brosschot et al. 2010, 2018). They assume that a mental representation of the stressful event is created and continuously activated by unconscious cognitive processes and thereby induces a prolonged physiological response to an anticipated event. This notion could account for the physiological changes observed during sleep in previous studies (Born et al. 1999; Hall et al. 2004), but cannot explain our results as we did observe changes on sleep parameters but not on physiological arousal during late sleep. Thus, concerning early insomnia models, an increased physiological arousal cannot explain our results (Spielman et al. 1987; Morin 1993; Perlis et al. 1997). However, an assumed general interaction between physiological and cognitive arousal does not contradict our findings, but the models fail to provide an underlying mechanism, how such an interaction could look like and affect sleep (Espie 2002; Harvey 2002; Espie et al. 2006). The neurocognitive and the hyperarousal model of insomnia provide a link between cognitive and physiological arousal by introducing cortical arousal as a physiological measure that is high frequency EEG activity (beta and gamma power, >14 Hz) of cognitive functions (Perlis et al. 1997; Riemann et al. 2010). Such cortical arousal during NREM and possibly REM sleep is assumed to directly affect sleep via enhanced sensory processing, information processing, and memory formation, leading for example to a facilitated disruption of sleep by ambient noise. Such a mechanism could explain how cognitive processes directly affect sleep and might have contributed to our findings on overall SWA/beta power ratio, sleep latency, WASO, and TST. Though, this explanation is unlikely based on comparable amounts of beta power over the whole nap and during the second half of sleep in the postsleep group (see Supplementary Fig. S1B), where changes of other sleep parameters were most pronounced. Moreover, this approach cannot explain dynamic changes in the progression of various sleep parameters across the nap and the differences in this dynamic between the pre- and the post-sleep group.

We recently proposed that mental concepts that are active during sleep are able to affect sleep physiology including sleep-depth regulatory systems and the subjective evaluation of sleep (Beck, Lorentz, et al. 2021). As an underlying mechanism, we assume that mental concepts related to sleep or wake are closely linked to somatosensory bodily functions. In support of this notion, several studies showed that semantic processing of words leads to an activation of related somatosensory brain areas (Boulenger et al. 2012; Moseley et al. 2012; Dreyer and Pulvermüller 2018). The second assumption of our theory is based on memory consolidation research (Oudiette and Paller 2013; Rasch and Born 2013) and assumes that the presleep activation of mental concepts associated to sleep or wake increases the likelihood that such concepts are reactivated during subsequent sleep. This is also in line with previously mentioned stress models assuming that unconscious cognitive representations of a stressor are active during sleep (Brosschot 2010; Brosschot et al. 2018). Lastly, we provided evidence for the core mechanism of the framework meaning that the activation of sleep- or wake-related semantic concepts during sleep is capable of affecting sleep itself (Beck, Lorentz, et al. 2021).

Within this framework, a generally prolonged effect of presleep stress on sleep originates from a repeated reactivation of mental concepts associated with this stressor during sleep. Due to their close link to somatosensory brain functions including wake, stress, and sleep regulatory systems, sleep depth is thought to be directly reduced. The model does not exclude that physiological measures such as heart rate, cortisol, or ACTH are also increased, but it does not require physiological arousal as a mediator to affect sleep depth. In addition to this general effect of stress on sleep physiology, an explanation for the differences in dynamic changes between the presleep and postsleep group could be that the frequency of reactivations of mental concepts associated with the stressor increases with temporal proximity to the stressor and decreases with temporal distance to the stressor. Such a mechanism might explain the decreasing effect of stress on sleep in the presleep group performing the stress-inducing task before sleep and the increasing effect of stress on late sleep in the postsleep group anticipating the stressor to happen after sleep. This could also explain, why studies including a temporal delay between a stressor and sleep do not report an effect of stress on sleep physiology (Kim et al. 2019).

So far, our results are limited to a daytime nap setting and should be further investigated in future studies assessing sleep over a full night. Such studies should include an analysis of dynamic changes of sleep parameters and oscillations as well as measures of cognitive and physiological arousal to further elucidate the effects of anticipated future vs. past stressors on sleep physiology. We would assume that over a whole night of sleep, cognitive arousal might contribute even more strongly to the detrimental effect of stress on sleep. Physiological arousal in response to a presleep stressor would decline and affect sleep similarly as in our nap study, however reactivations of mental concepts and their associated bodily functions might affect night time sleep over a longer time period compared with a nap design. In addition to a nighttime design, a more direct measures of the HPA-axis activity such as ACTH or cortisol concentrations (Dickerson and Kemeny 2004) could provide

additional information about the association between cognitive and physiological arousal and their progression across sleep. However, choosing a measure of autonomic nervous system activity such as heart rate has the advantage to be less affected by habituation to the TSST (Allen et al. 2017). As the TSST is a well-established and frequently used task, subjects who have already conducted the TSST in previous studies would need to be excluded when cortisol or ACTH measures are administered. In our study, a virtual reality version of the TSST comprised a video of a panel, while one real and nonvisible experimenter was located behind the subject. This setup differs from the standard TSST procedure and could have affected the stress response, also in comparison with previous virtual reality applications utilizing fully animated and interacting experimenters (Zimmer et al. 2019). Yet, such effects would have only affected the presleep group, as the postsleep anticipation group did not receive information that the task will be conducted in a virtual reality environment after sleep. In addition, our results suggest that conducting the virtual reality tasks before sleep affected subsequent sleep by generally increasing higher EEG frequency power in the beta range, which might not occur using a real-life version of both tasks. Moreover, a limitation of the current study is the comparison of the stress condition to a relaxation instead of a neutral control condition. Thus, the effect of the stress condition on sleep might have been overestimated in this study as sleep in the relaxation condition could be improved compared to sleep without a manipulation. Furthermore, this study is restricted to the effects of a psychosocial stressor on sleep. Future experiments could study different types of cognitive arousal, such as cognitive demanding tasks, which have shown to affect sleep without a concomitant emotional arousal (Wuyts et al. 2012a) or the effect of positive emotional arousal such as an anticipated birthday or concert one is looking forward to attend to. While our experimentally induced differences in arousal between groups elicited large effect sizes in our sample of healthy subjects, the question of whether these effects are functionally relevant to people with stress-related disorders warrants further investigation.

Moreover, subjective evaluations of sleep were not strongly affected by our manipulation. Despite similar ME on EEG parameters in both study groups, presleep stress seemed to affect subjective sleep quality more strongly than anticipated future stress. This effect might be related to the subjectively rated time to fall asleep, which is included in the assessment of subjective sleep quality and tended to be more elevated in response to stress in the presleep group. This, in turn, might be attributed to the fact that the falling asleep period is temporally closer to the stressor in the presleep group. Subjective sleep quality is considered more important in the assessment of clinical sleep disorders compared with objectively measured sleep and the diagnosis of insomnia is based solely on the subjective evaluation of sleep (Schutte-Rodin et al. 2008; Riemann et al. 2020). As we found no effects on subjective sleep quality in the postsleep group and only trends in the presleep group, future studies need to further examine the relevance of stress-related dynamic changes in objective sleep parameters for subjective sleep complaints in healthy subjects and insomnia patients.

In conclusion, our results show that our presleep cognitions have a direct effect on sleep, which is not mediated by physiological arousal. We suggest that the mental representations with links to somatosensory bodily functions are repeatedly activated during sleep and thereby directly affect sleep physiology during early, but also during later parts of the sleep period. In addition, our results suggest that the effect of cognitions on sleep is enhanced with temporal proximity to the stressor. This proposed mechanism might be especially relevant for early awakening insomnia, where the stressful event comprises an early awakening and its daytime consequences. However, future studies first need to compare the effects of anticipated stress on sleep upon awakening, with the effects of stress anticipated later during the subsequent day, as well as anticipated several days in advance. To further elucidate the relationship between stress, cognition and sleep, futures studies in healthy individuals as well as in insomnia patients should include a systematic assessment of the progression of arousal and sleep parameters across the sleep period. This would provide a basis for the development of nonpharmacological preventive measures and therapy options to contrast stress-related sleep disturbances.

Acknowledgments

We would like to thank Adrian Quartenoud, Ajinthan Muthusamy, Carla Preuss, Emil Nissen, Leonie Schmidlin, Louisa Clarke, Maximilian Spottke, Michel Bosshard, Sandrine Baselgia, Sarah Tanner, and Simon Janz for assistance in data collection, Louisa Clarke for helpful comments on an earlier version of this article, and all subjects for their participation. J.B. and B.R designed the research; J.B. performed the experiments; J.B. and E.L. analyzed the data; J.B. and B.R. interpreted the results, J.B. and B.R. wrote the manuscript.

Supplementary material

Supplementary material can be found at *Cerebral Cortex* online.

Funding

This work was supported by a grant of the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation program (grant agreement number 667875). This study was conducted at the University of Fribourg, Department of Psychology, Division of Cognitive Biopsychology and Methods.

Conflict of interest statement. E.L. is an employee of The Siesta Group Schlafanalyse GmbH. J.B. and B.R. have nothing to disclose.

References

Ackermann S, Hartmann F, Papassotiropoulos A, de Quervain DJ-F, Rasch B. No associations between interindividual differences in sleep parameters and episodic memory consolidation. *Sleep*. 2015:38(6):951–959.

Ackermann S, Cordi MJ, La Marca R, Seifritz E, Rasch B. Psychosocial stress before a nap increases sleep latency and decreases early slow-wave activity. *Front Psychol*. 2019:10:20.

Agnew HW, Webb WB, Williams RL. The first night effect: an EEG study of sleep. *Psychophysiology*. 1966:2(3):263–266.

Allen AP, Kennedy PJ, Dockray S, Cryan JF, Dinan TG, Clarke G. The trier social stress test: principles and practice. *Neurobiol Stress*. 2017:6:113–126.

Anderer P, Moreau A, Woertz M, Ross M, Gruber G, Parapatics S, Loretz E, Heller E, Schmidt A, Boeck M, et al. Computer-assisted sleep classification according to the standard of the American Academy of Sleep Medicine: validation study of the AASM version of the Somnolyzer 24 × 7. *Neuropsychobiology*. 2010:62(4):250–264.

Ballesio A, Bacaro V, Vacca M, Chirico A, Lucidi F, Riemann D, Baglioni C, Lombardo C. Does cognitive behaviour therapy for insomnia reduce repetitive negative thinking and sleep-related worry beliefs? A systematic review and meta-analysis. *Sleep Med Rev*. 2020:55:101378.

Beck J, Cordi MJ, Rasch B. Hypnotic suggestions increase slow-wave parameters but decrease slow-wave spindle coupling. *Nat Sci Sleep*. 2021:13:1383–1393.

Beck J, Loretz E, Rasch B. Exposure to relaxing words during sleep promotes slow-wave sleep and subjective sleep quality. *Sleep*. 2021:44(11):1–14.

Born J, Hansen K, Marshall L, Mölle M, Fehm HL. Timing the end of nocturnal sleep. *Nature*. 1999:397(6714):29–30.

Boulenger V, Shtyrov Y, Pulvermüller F. When do you grasp the idea? MEG evidence for instantaneous idiom understanding. *NeuroImage*. 2012:59(4):3502–3513.

Brosschot JF. Markers of chronic stress: prolonged physiological activation and (un)conscious perseverative cognition. *Neurosci Biobehav Rev*. 2010:35(1):46–50.

Brosschot JF, Gerin W, Thayer JF. The perseverative cognition hypothesis: a review of worry, prolonged stress-related physiological activation, and health. *J Psychosom Res*. 2006:60(2):113–124.

Brosschot JF, van Dijk E, Thayer JF. Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. *Int J Psychophysiol*. 2007:63(1):39–47.

Brosschot JF, Verkuil B, Thayer JF. Conscious and unconscious perseverative cognition: is a large part of prolonged physiological activity due to unconscious stress? *J Psychosom Res*. 2010:69(4):407–416.

Brosschot JF, Verkuil B, Thayer JF. Generalized unsafety theory of stress: unsafe environments and conditions, and the default stress response. *IJERPH*. 2018:15(3):464.

Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989:28(2):193–213.

Clancy F, Prestwich A, Caperon L, Tsipa A, O'Connor DB. The association between worry and rumination with sleep in non-clinical populations: a systematic review and meta-analysis. *Health Psychol Rev*. 2020:14(4):427–448.

Cordi MJ, Ackermann S, Rasch B. Effects of relaxing music on healthy sleep. *Sci Rep*. 2019:9(1):9079.

Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol Bull*. 2004:130(3):355–391.

Drake CL, Cheng P, Almeida DM, Roth T. Familial risk for insomnia is associated with abnormal cortisol response to stress. *Sleep*. 2017:40(10):1–10.

Dreyer FR, Pulvermüller F. Abstract semantics in the motor system? An event-related fMRI study on passive reading of semantic word categories carrying abstract emotional and mental meaning. *Cortex*. 2018:100:52–70.

Elder GJ, Barclay NL, Wetherell MA, Ellis JG. Anticipated next-day demand affects the magnitude of the cortisol awakening response, but not subjective or objective sleep. *J Sleep Res*. 2018:27(1):47–55.

Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annu Rev Psychol*. 2002:53:215–243.

Espie CA, Broomfield NM, MacMahon KMA, Macphee LM, Taylor LM. The attention-intention-effort pathway in the development of psychophysiologic insomnia: a theoretical review. *Sleep Med Rev*. 2006:10(4):215–245.

Fan M, Sun D, Zhou T, Heianza Y, Lv J, Li L, Qi L. Sleep patterns, genetic susceptibility, and incident cardiovascular disease: a prospective study of 385 292 UK biobank participants. *Eur Heart J*. 2020:41(11):1182–1189.

Friborg O, Hjemdal O, Rosenvinge JH, Martinussen M. A new rating scale for adult resilience: what are the central protective resources behind healthy adjustment? *Int J Methods Psychiatr Res*. 2003:12(2):65–76.

Germain A, Buysse DJ, Ombao H, Kupfer DJ, Hall M. Psychophysiological reactivity and coping styles influence the effects of acute stress exposure on rapid eye movement sleep. *Psychosom Med*. 2003:65(5):857–864.

Gordon AM, Del Rosario K, Flores AJ, Mendes WB, Prather AA. Bidirectional links between social rejection and sleep. *Psychosom Med*. 2019:81(8):739–748.

Görtelmeyer R. *SF-A/R und SF-B/R: Schlaffragebogen a und B*. Hogrefe; 2011

Griefahn B, Kunemund C, Brode P, Mehnert P. Zur Validitat der deutschen Ubersetzung des Morningness-Eveningness-questionnaires von Horne und Ostberg. The validity of a German version of the Morningness-Eveningness-questionnaire developed by Horne and Ostberg. *Somnologie*. 2001:5(2):71–80.

Gross RT, Borkovec TD. Effects of a cognitive intrusion manipulation on the sleep-onset latency of good sleepers. *Behav Ther*. 1982:13(1):112–116.

Hale L, Troxel W, Buysse DJ. Sleep health: an opportunity for public health to address health equity. *Annu Rev Public Health*. 2020:41:81–99.

Hall M, Vasko R, Buysse D, Ombao H, Chen Q, Cashmere JD, Kupfer DJ, Thayer JF. Acute stress affects heart rate variability during sleep. *Psychosom Med*. 2004:66(1):56–62.

Hall M, Thayer JF, Germain A, Moul DE, Vasko R, Puhl M, Miewald J, Buysse DJ. Psychological stress is associated with heightened physiological arousal during NREM sleep in primary insomnia. *Behav Sleep Med*. 2007:5(3):178–193.

Hantsoo L, Khou CS, White CN, Ong JC. Gender and cognitive-emotional factors as predictors of pre-sleep arousal and trait hyperarousal in insomnia. *J Psychosom Res*. 2013:74(4):283–289.

Harvey AG. Pre-sleep cognitive activity: a comparison of sleep-onset insomniacs and good sleepers. *Br J Clin Psychol*. 2000:39(3):275–286.

Harvey AG. A cognitive model of insomnia. *Behav Res Ther*. 2002:40(8): 869–893.

Hogan SE, Delgado GM, Hall M, Nimgaonkar VL, Germain A, Buysse DJ, Wilckens KA. Slow-oscillation activity is reduced and high frequency activity is elevated in older adults with insomnia. *J Clin Sleep Med*. 2020:16(9):1445–1454.

Iber C, Ancoli-Israel S, Chesson AL, Quan SF. *The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications*. Vol. 1. Westchester, IL: American Academy of Sleep Medicine; 2007

Janson J, Rohleder N. Distraction coping predicts better cortisol recovery after acute psychosocial stress. *Biol Psychol*. 2017:128: 117–124.

Jansson-Fröjmark M, Norell-Clarke A. Psychometric properties of the pre-sleep arousal scale in a large community sample. *J Psychosom Res*. 2012:72(2):103–110.

Kalmbach DA, Buysse DJ, Cheng P, Roth T, Yang A, Drake CL. Nocturnal cognitive arousal is associated with objective sleep disturbance and indicators of physiologic hyperarousal in good sleepers and individuals with insomnia disorder. *Sleep Med*. 2020:71: 151–160.

Kecklund G, Åkerstedt T. Apprehension of the subsequent working day is associated with a low amount of slow wave sleep. *Biol Psychol*. 2004:66(2):169–176.

Kecklund G, Åkerstedt T, Lowden A. Morning work: effects of early rising on sleep and alertness. *Sleep*. 1997:20(3):215–223.

Kennedy RS, Lane NE, Berbaum KS, Lilienthal MG. Simulator sickness questionnaire: an enhanced method for quantifying simulator sickness. *Int J Aviat Psychol*. 1993:3(3):203–220.

Kim SY, Kark SM, Daley RT, Alger SE, Rebouças D, Kensinger EA, Payne JD. Interactive effects of stress reactivity and rapid eye movement sleep theta activity on emotional memory formation. *Hippocampus*. 2019:30(8):829–841.

Kirschbaum C, Pirke KM, Hellhammer DH. The 'Trier social stress Test'–a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*. 1993:28(1–2): 76–81.

Kogler L, Müller VI, Chang A, Eickhoff SB, Fox PT, Gur RC, Derntl B. Psychosocial versus physiological stress - meta-analyses on deactivations and activations of the neural correlates of stress reactions. *NeuroImage*. 2015:119:235–251.

Lattova Z, Keckeis M, Maurovich-Horvat E, Wetter TC, Wilde-Frenz J, Schuld A, Pollmächer T. The stress hormone system in various sleep disorders. *J Psychiatr Res*. 2011:45(9):1223–1228.

Le Bon O, Hoffmann G, Tecco J, Staner L, Noseda A, Pelc I, Linkowski P. Mild to moderate sleep respiratory events: one negative night may not be enough. *Chest*. 2000:118(2):353–359.

Lee D-H, Cho C-H, Han C, Bok K-N, Moon JH, Lee E, Lee H-J, Kim L. Sleep irregularity in the previous week influences the first-night effect in polysomnographic studies. *Psychiatry Investig*. 2016:13(2): 203–209.

Lemyre A, Belzile F, Landry M, Bastien CH, Beaudoin LP. Pre-sleep cognitive activity in adults: a systematic review. *Sleep Med Rev*. 2020:50:101253.

Liszio S, Graf L, Masuch M. The relaxing effect of virtual nature: immersive technology provides relief in acute stress situations. *Annu Rev CyberTherapy Telemed*. 2018:16:87–93.

Maes J, Verbraecken J, Willemen M, Volder I D, van Gastel A, Michiels N, Verbeek I, Vandekerckhove M, Wuyts J, Haex B, et al. Sleep misperception, EEG characteristics and autonomic nervous system activity in primary insomnia: a retrospective study on polysomnographic data. *Int J Psychophysiol*. 2014:91(3):163–171.

Mölle M, Marshall L, Gais S, Born J. Grouping of spindle activity during slow oscillations in human non-rapid eye movement sleep. *J Neurosci*. 2002:22(24):10941–10947.

Mölle M, Bergmann TO, Marshall L, Born J. Fast and slow spindles during the sleep slow oscillation: disparate coalescence and engagement in memory processing. *Sleep*. 2011:34(10):1411–1421.

Morin CM. *Insomnia: psychological assessment and management*. Guilford Press; 1993

Moseley R, Carota F, Hauk O, Mohr B, Pulvermüller F. A role for the motor system in binding abstract emotional meaning. *Cereb Cortex*. 2012:22(7):1634–1647.

Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behav Res Ther*. 1985:23(3):263–271.

Ong JC, Ulmer CS, Manber R. Improving sleep with mindfulness and acceptance: a metacognitive model of insomnia. *Behav Res Ther*. 2012:50(11):651–660.

Oostenveld R, Fries P, Maris E, Schoffelen J-M. FieldTrip: open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. *Comput Intell Neurosci*. 2011:2011:156869.

Oudiette D, Paller KA. Upgrading the sleeping brain with targeted memory reactivation. *Trends Cogn Sci*. 2013:17(3):142–149.

Perlis ML, Giles DE, Mendelson WB, Bootzin RR, Wyatt JK. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *J Sleep Res*. 1997:6(3):179–188.

Pillai V, Steenburg LA, Ciesla JA, Roth T, Drake CL. A seven day actigraphy-based study of rumination and sleep disturbance among young adults with depressive symptoms. *J Psychosom Res*. 2014:77(1):70–75.

R Core Team. 2018. *R: a language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing, Vienna, Austria. Available from: https://www.R-project.org/.

Rasch B, Born J. About sleep's role in memory. *Physiol Rev*. 2013:93(2): 681–766.

van Reeth O, Weibel L, Spiegel K, Leproult R, Dugovic C, Maccari S. Physiology of sleep (review)–interactions between stress and sleep: from basic research to clinical situations. *Sleep Med Rev*. 2000:4(2):201–219.

Riemann D, Spiegelhalder K, Feige B, Voderholzer U, Berger M, Perlis ML, Nissen C. The hyperarousal model of insomnia: a review of the concept and its evidence. *Sleep Med Rev*. 2010:14(1):19–31.

Riemann D, Krone LB, Wulff K, Nissen C. Sleep, insomnia, and depression. *Neuropsychopharmacology*. 2020:45(1):74–89.

Schubert T, Friedmann F, Regenbrecht H. The experience of presence: factor analytic insights. *Presence: Teleoperators Virtual Environ*. 2001:10(3):266–281.

Schutte-Rodin S, Broch L, Buysse D, Dorsey C, Sateia M. Clinical guideline for the evaluation and management of chronic insomnia in adults. *J Clin Sleep Med*. 2008:4(5):487–504.

Spiegelhalder K, Regen W, Feige B, Hirscher V, Unbehaun T, Nissen C, Riemann D, Baglioni C. Sleep-related arousal versus general cognitive arousal in primary insomnia. *J Clin Sleep Med*. 2012:8(4): 431–437.

Spielberger CD. Manual for the state-trait anxiety, inventory. *Consulting Psychologist*. 1970:1.

Spielman AJ, Caruso LS, Glovinsky PB. A behavioral perspective on insomnia treatment. *Psychiatr Clin North Am*. 1987:10(4):541–553.

Steyer R, Schwenkmezger P, Notz P, Eid M. *Der Mehrdimensionale Befindlichkeitsfragebogen MDBF [multidimensional mood questionnaire]*. Göttingen, Germany: Hogrefe; 1997

Takano K, Iijima Y, Tanno Y. Repetitive thought and self-reported sleep disturbance. *Behav Ther*. 2012:43(4):779–789.

Tellegen A, Atkinson G. Openness to absorbing and self-altering experiences ("absorption"), a trait related to hypnotic susceptibility. *J Abnorm Psychol*. 1974:83(3):268–277.

Tousignant OH, Taylor ND, Suvak MK, Fireman GD. Effects of rumination and worry on sleep. *Behav Ther*. 2019:50(3):558–570.

Trapnell PD, Campbell JD. Private self-consciousness and the five-factor model of personality: distinguishing rumination from reflection. *J Pers Soc Psychol*. 1999:76(2):284–304.

Vandekerckhove M, Weiss R, Schotte C, Exadaktylos V, Haex B, Verbraecken J, Cluydts R. The role of presleep negative emotion in sleep physiology. *Psychophysiology*. 2011:48(12):1738–1744.

Vochem J, Strobel C, Maier L, Spiegelhalder K, Hertenstein E, Riemann D, Feige B. Pre-sleep arousal scale (PSAS) and the time monitoring Behavior-10 scale (TMB-10) in good sleepers and patients with insomnia. *Sleep Med*. 2019:56:98–103.

Watkins ER, Moulds M, Mackintosh B. Comparisons between rumination and worry in a non-clinical population. *Behav Res Ther*. 2005:43(12):1577–1585.

Wuyts J, Valck E D, Vandekerckhove M, Pattyn N, Bulckaert A, Berckmans D, Haex B, Verbraecken J, Cluydts R. The influence of pre-sleep cognitive arousal on sleep onset processes. *Int J Psychophysiol*. 2012a:83(1):8–15.

Wuyts J, Valck E D, Vandekerckhove M, Pattyn N, Exadaktylos V, Haex B, Maes J, Verbraecken J, Cluydts R. Effects of pre-sleep simulated on-call instructions on subsequent sleep. *Biol Psychol*. 2012b:91(3):383–388.

Xie L, Kang H, Xu Q, Chen MJ, Liao Y, Thiyagarajan M, O'Donnell J, Christensen DJ, Nicholson C, Iliff JJ, et al. Sleep drives metabolite clearance from the adult brain. *Science*. 2013:342(6156):373–377.

Yamanaka Y, Motoshima H, Uchida K. Hypothalamic-pituitary-adrenal axis differentially responses to morning and evening psychological stress in healthy subjects. *Neuropsychopharmacol Rep*. 2019:39(1):41–47.

Zimmer P, Buttlar B, Halbeisen G, Walther E, Domes G. Virtually stressed? A refined virtual reality adaptation of the trier social stress test (TSST) induces robust endocrine responses. *Psychoneuroendocrinology*. 2019:101:186–192.

Zoccola PM, Dickerson SS, Lam S. Rumination predicts longer sleep onset latency after an acute psychosocial stressor. *Psychosom Med*. 2009:71(7):771–775.