REM theta activity predicts re-experiencing symptoms after exposure to a traumatic film

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Abstract

Background Extensive empirical evidence indicates that sleep plays an active role in memory consolidation. Moreover, sleep has been found to preferentially enhance emotional memories and may modulate affective reactions to previously encountered stimuli. Intriguingly, recent findings suggest that disruptions of sleep-related memory processing could be involved in posttraumatic symptom development such that sleep disturbances may accelerate symptoms of intrusive re-experiencing.

Methods Based on this emerging evidence, we investigated whether an analogue traumatic event would result in immediate impairments of sleep quality in a group of healthy, robust sleepers. In addition, we examined associations between a specific oscillatory correlate of emotional memory consolidation processes (REM theta activity) and subsequent analogue PTSD symptoms. Thirty-three healthy participants entered the study and were exposed to either "traumatic" or neutral films. Thereafter, participants were subjected to an 8.5-hour-long nocturnal sleep opportunity under standardized laboratory conditions including full-night polysomnographic recordings. Ambulatory intrusive memories and subjective symptom ratings were assessed during a period of three consecutive days.

Results and Conclusions Our results partially confirm that trauma film exposure impairs subsequent sleep quality (i.e. with regard to sleep quantity). Correlation analyses further reveal that a longer REM sleep duration after "traumatic" exposure predicts reduced analogue PTSD symptoms. Critically, REM theta activity selectively predicts lower re-experiencing symptoms. As previous findings suggest that REM theta activity is reduced in patients with posttraumatic stress disorder, our findings provide a new perspective on the functional role of REM sleep in trauma memory processing.

Keywords: REM sleep; intrusive memories; memory consolidation; emotional memory; posttraumatic stress disorder

1. Introduction

Posttraumatic stress disorder (PTSD; APA, 2013) is a mental disorder that may develop after exposure to a traumatic life event (e.g. threatened death, severe injury or sexual assault). Amongst the core characteristics of PTSD, re-experiencing symptoms are assumed to play a pivotal role in the development and course of the disorder (Ehlers, 2010; Ehlers & Clark, 2000). Specifically, the persistence of distressing, intrusive memories is found to be a strong predictor of chronic symptom trajectories (Ehlers, Hackmann, & Michael, 2004; Hackmann, Ehlers, Speckens, & Clark, 2004). Intrusive memories consist of brief, sensory fragments of the traumatic event, which are involuntarily retrieved when individuals encounter internal or external cues of the trauma (Ehlers, 2010). These intrusions are experienced as highly vivid, characterized by a strong sense of "nowness", and often lack awareness of the self in the past (i.e. autonoetic consciousness). As a result of these characteristics, intrusive memories cause high levels of distress and perpetuate perceptions of ongoing threat (Birrer, Michael, & Munsch, 2007; Michael, Ehlers, Halligan, & Clark, 2005). This ongoing sense of threat may, in turn, promote the development of hyperarousal and avoidance symptoms (Ehlers & Clark, 2000; Ehlers et al., 2002).

The mechanisms underlying intrusive memory formation are strongly tied to the implicit memory system. It is assumed that stress-induced enhancements of perceptual priming and associative learning at the time of the traumatic event promote subsequent intrusive re-experiencing (Ehlers, Michael, Chen, Payne, & Shan, 2006; Ehring & Ehlers, 2011; Lissek & van Meurs, 2015; Michael, Ehlers, & Halligan, 2005; Streb, Conway, & Michael, 2017; Wegerer, Blechert, Kerschbaum, & Wilhelm, 2013). Contemporary models further postulate that poor memory elaboration (Ehlers & Clark, 2000) and a lack of hippocampal engagement during encoding (Brewin, Gregory, Lipton, & Burgess, 2010) contribute to weaker explicit retrieval of trauma memories (Halligan, Michael, Clark, & Ehlers, 2003; Murray, Ehlers, & Mayou, 2018). Restricted voluntary access to trauma memories may further result in a lack of contextual retrieval during episodes of intrusive re-experiencing (Brewin, 2014). Despite the critical role of intrusive re-experiencing in PTSD, research shows that the frequency of early intrusive memories is only weakly associated with persistent PTSD (Michael, Ehlers, Halligan, et al., 2005; Shalev, 1992). Moreover, an early peak reaction of PTSD symptoms (within two weeks of the traumatic event) has been associated with less severe pathology after 12 weeks (Gilboa-Schechtman & Foa, 2001). Hence, it is assumed that post-encoding memory processing in the early aftermath of a trauma constitutes a critical time period during which trajectories of resilience and symptom persistence start to emerge (van Marle, 2015).

One particularly strong influential factor of post-encoding memory processes is sleep. Current frameworks propose that sleep provides a unique neurophysiological environment for reactivations of newly acquired

memory representations in the hippocampus, which are subsequently redistributed to long-term storage sites in the neocortex (Diekelmann & Born, 2010; Rasch & Born, 2013). This process of systems consolidation is assumed to support episodic memory by enabling accurate retrieval of remote events. Of particular note, sleep has been found to preferentially enhance emotional memories and may modulate affective reactions to previously encountered stimuli (Goldstein & Walker, 2014; Walker & van der Helm, 2009). After a period of sleep, individuals demonstrate higher recognition performance for emotional stimuli as compared to neutral stimuli (e.g. Groch, Wilhelm, Diekelmann, & Born, 2013; Groch, Zinke, Wilhelm, & Born, 2015; Payne & Kensinger, 2010; Payne, Stickgold, Swanberg, & Kensinger, 2008). Conversely, it has been shown that physiological responses to emotional stimuli dissipate across sleep whilst remaining preserved across wakefulness (Cunningham et al., 2014; Pace-Schott et al., 2011; but see Werner, Schabus, Blechert, Kolodyazhniy, & Wilhelm, 2015). Integrating these findings suggests that sleep strengthens explicit retrieval of emotional events and simultaneously modulates implicit memory, as for instance evident in greater between-session habituation to aversive stimuli (Pace-Schott et al., 2011).

In contrast to episodic memory for neutral events, which is assumed to be supported by slow wave sleep (SWS), emotional memory has been frequently associated with rapid eye movement (REM) sleep physiology (for reviews see Genzel, Spoormaker, Konrad, & Dresler, 2015 and Hutchison & Rathore, 2015). This association is reflected in significant correlations between REM sleep duration and microstructure (latency, REM density, and theta activity) and post-sleep emotional memory performance (Gilson et al., 2015; Nishida, Pearsall, Buckner, & Walker, 2009; Prehn-Kristensen et al., 2013). Notably, REM theta power (4-7 Hz) has also been shown to correlate with enhanced retention of location memory for emotional images (Sopp, Michael, Weess, & Mecklinger, 2017). This finding is in line with the assumption that REM theta activity reflects reactivations of emotional memory representations in the limbic system and neocortical networks, which may enable the strengthening of different qualities of these representations across disparate brain regions (Goldstein & Walker, 2014).

Based on the extensive evidence of sleep's role in emotional memory processing, it has been hypothesized that sleep disturbances, and particularly REM sleep disturbances, may accelerate memory-related symptoms after trauma (Pace-Schott, Germain, & Milad, 2015; Spoormaker & Montgomery, 2008). This assumption is supported by the high prevalence of sleep disturbances in patients with PTSD (70-91%; Maher, Rego, & Asnis, 2006) and by differences in sleep physiology between PTSD patients and trauma-exposed healthy controls. Specifically, PTSD patients show enhanced REM sleep fragmentation (Insana, Kolko, & Germain, 2012; Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002; Mellman, Pigeon, Nowell, & Nolan, 2007; Stocker et al., 2016) and reduced frontal theta activity (Cowdin, Kobayashi, & Mellman, 2014). Longitudinal studies further indicate that sleep disturbances – both before and after trauma – are a

risk factor of PTSD development (Babson & Feldner, 2010; Marcks, Weisberg, Edelen, & Keller, 2010). Hence, it is assumed that sleep disturbances may facilitate the transition from acute stress symptoms to PTSD by interfering with post-encoding memory processes (Pace-Schott et al., 2015). Maintaining restful sleep after trauma exposure may conversely provide a window for integrative processing of trauma memories, which could promote natural recovery (Kleim, Wysokowsky, Schmid, Seifritz, & Rasch, 2016).

This notion has been further explored in experimental studies using the trauma film paradigm (James et al., 2016). In a first study, Porcheret, Holmes, Goodwin, Foster, and Wulff (2015) investigated the effects of sleep deprivation as opposed to rested sleep on intrusive memories after exposure to a "traumatic" film. Participants in the sleep deprivation condition reported a reduced number of intrusions on the first two days of ambulatory assessment, contradicting the hypothesis that sleep alleviates symptoms of intrusive re-experiencing. However, these effects emerged during the phase of acute sleep deprivation and may thus be attributable to the neurophysiological impact of sleep deprivation (Forest & Godbout, 2000) rather than to sleep-related consolidation processes. In line with this assumption, Kleim et al. (2016) found that rested sleep (as compared to sleep deprivation) was associated with reduced intrusive re-experiencing when intrusions were measured over a prolonged period of time and excluded from the analysis when they had occurred during acute sleep deprivation. Further findings of Woud et al. (2018) support these results as nap sleep was found to promote reduced intrusive re-experiencing, which was evident in ambulatory assessment and on a clinical measure of intrusive re-experiencing (Horowitz, Wilner, & Alvarez, 1979).

Overall, the current state of research suggests that restful sleep may be linked to reduced intrusive re-experiencing. However, further research is required to substantiate this claim and to elucidate the processes which contribute to these effects. In particular, further studies need to investigate whether traumatic exposure directly impacts subsequent sleep quality. Although sleep disturbances are highly prevalent in PTSD patients, it has not been firmly established whether these disturbances arise directly from exposure to a traumatic event or whether they are also evident prior to traumatization. Moreover, previous analogue studies did not focus on the effects of REM sleep and REM theta oscillations on subsequent symptoms of intrusive re-experiencing. These associations may be critical as REM sleep and REM theta activity have been implicated in emotional memory processing and are found to be altered in PTSD patients.

To address these gaps in the current literature, we conducted an experimental analogue study contrasting the effects of "traumatic" and neutral films on subsequent sleep physiology. In addition, we investigated links between post-"traumatic" REM sleep physiology and subsequent intrusive re-experiencing. The study examined healthy participants, who were assigned to one of two experimental conditions exposing

them to either "traumatic" or neutral films. Films were presented in the same standardized procedure, which included the assessment of physiological and subjective stress responses. Thereafter, participants were subjected to an 8.5-hour-long nocturnal sleep opportunity under standardized laboratory conditions including full-night polysomnography (PSG) recordings. Ambulatory intrusive memories and subjective symptom ratings (Horowitz et al., 1979) were assessed during a period of three consecutive days. Based on the high prevalence of sleep disturbances in patients with PTSD, we hypothesized that exposure to the trauma film would result in reduced total sleep time and prolonged periods of wakefulness during the night. In addition, we examined whether "traumatic" exposure would delay sleep onset as evident in great sleep onset latencies. Based on accounts of REM sleep fragmentation in PTSD, we further predicted that REM sleep would be reduced in the trauma film group. Finally, we hypothesized that REM sleep physiology (duration and theta activity) would be negatively correlated to intrusive re-experiencing as assessed with intrusion diaries and subjective symptom ratings.

2. Methods

2.1. Sample characteristics

Thirty-three healthy university students participated in the present study. Study eligibility was confirmed using an online screening survey and a telephone interview. The online screening survey contained questionnaires assessing sleep quality (PSQI; Buysse et al., 1991), daytime sleepiness (ESS; Johns, 1991), diurnal preference (rMEQ; Randler, 2013), handedness (Oldfield, 1971), and depressive symptoms (PHQ-9; Kroenke, Spitzer, & Williams, 2001). Participation was restricted to individuals fulfilling the following criteria: Good sleep quality (PSQI $\leq$ 5; Buysse et al., 1991), lack of significant depressive symptoms (PHQ-9 $\leq$ 9; Kroenke et al., 2001), no extreme evening preference, habitual sleep duration $\geq$ 6 hours, and pronounced right-handedness. Participants were further required to be aged between 18 and 30 years with normal or corrected-to-normal vision, and to be in good general health (BMI in the normal range, no acute or chronic disorders except for thyroid disorders, or long-term medication except for thyroid medication). Potentially confounding effects of menstrual cycle were minimized by selecting only females using hormonal contraception. Additionally, participants were excluded if they met any of the following criteria: Insufficient German language skills, pronounced preference for splatter or horror movies, regular night shift work, and previous familiarization with study materials (i.e. participation in a trauma film study).

Potential participants who met these preliminary criteria were additionally required to undergo a semi-structured telephone screening procedure in which previous traumatic experiences, symptoms of axis I disorders, and previous psychotherapeutic treatment were assessed. Any indications of a previous history of psychiatric symptoms or traumatic exposure resulted in exclusion from further participation. Of all participants, n = 3 were excluded from further analyses as polysomnographic recordings were

discontinued during nocturnal sleep. Thus, the final sample for analyses comprised 16 participants (8 male; M_{age}= 21.69, *SD*= 2.27) in the trauma film (TF) condition and 14 participants (6 male; M_{age}= 22.14, *SD*= 2.45) in the neutral film (NF) condition. All participants gave written informed consent in accordance with the Declaration of Helsinki and were paid € 75 for study participation. The study protocol was approved by the local ethics committee.

2.2. Experimental procedure

Qualified participants were informed that they would be assigned to one of two study conditions in which they would either be exposed to aversive or neutral film clips. Group assignment was performed in a pseudorandom fashion in order to establish balanced gender ratios in both groups (Genzel et al., 2012). Participants were assigned to individual study groups (TF or NF) before their arrival at the laboratory. However, they did not receive any information regarding their assignment until the experiment was completed. Participants were instructed to maintain a regular sleep-wake schedule starting 72 hours prior to the experiment. In addition, they were asked to refrain from drinking alcohol or caffeinated beverages 24 hours before their laboratory appointment. Participants were further instructed to rise at 7:00 a.m. latest on the morning of the experiment, which was to be confirmed by emailing the experimenter upon awakening.

On the night of the experiment, participants were instructed to arrive at the laboratory by 8:00 p.m.. Upon arrival, compliance with pre-experimental instructions was confirmed and participants were familiarized with the sleep study room. They were instructed to change into a comfortable track suit and prepare as if they were going to bed shortly (e.g. by brushing their teeth). Afterwards, participants were seated in a sound-proof testing booth facing a 27" LCD monitor (60 Hz refresh rate) at a viewing distance of about 65 cm and were prepared for psychophysiological assessment (see 2.3.1.2.). Thereafter, resting state psychophysiological measures were recorded for 5 minutes (pre-baseline) while participants watched a black screen. After completion, they were asked to fill out a questionnaire assessing their state anxiety levels (STAI-S; Laux, Glanzmann, Schaffner, & Spielberger, 1981; see 2.3.1.3.). Then they received standardized instructions on subsequent film presentation, which were presented on the computer screen. They were informed that they would be exposed to different film clips via computer screen and headphones, which they should pay attention to while imagining that they were an eyewitness of what was happening. Participants were reminded that the film may contain aversive scenes and that they were free to withdraw from further study participation at any time. Instructions were identical for TF and NF participants. Following these instructions, film presentation was started and psychophysiological recording were continued. After the film had ended participants were asked to complete the STAI-S again, followed by another 5-minute recording of resting state psychophysiological measures (post-baseline).

Upon completion, participants were prepared for polysomnographic measurements during the succeeding night. During this time, they were not allowed to speak with the experimenter about the content of the film or any related topics. If preparation was completed before 10:00 p.m., participants were offered a coloring book or asked to sit and relax for the remaining time. Shortly before 10:00 p.m., participants were accompanied into the sleep study room, where they were allowed to sleep for 8.5 hours while PSG measures were continuously recorded. At 6:30 a.m. participants were woken up by the experimenter, PSG recordings were terminated and participants were asked to sit in the testing booth and complete a clinical questionnaire assessing analogue PTSD symptoms (IES-R T1; Maercker & Schützwohl, 1998; see 2.3.1.4.). Prior to leaving the laboratory, participants received an intrusion diary as well as a sleep diary (to be completed throughout days 1 to 3). On day 4, participants returned to the laboratory handed in their diaries and completed the IES-R (T2) again. Thereafter, participants underwent debriefing and received monetary compensation.

2.3. Materials and measures

2.3.1. Trauma film procedure

2.3.1.1. Trauma film and neutral film

The trauma film used in the current study consisted of different film clips taken from a variety of commercially available R-rated movies (e.g. *German Angst*, *I spit on your grave 2*). All film clips contained depictions of extreme physical and/or sexual violence. Individual clips were selected based on the results of a pilot study in which an unrelated sample of participants (N = 14) rated different preselected film clips with regard to their emotional impact and aversiveness. Clips with the highest mean ratings were compiled to form a 14-minute-long film. The neutral film consisted of film clips from commercially available films (e.g. *The Police Officer's Wife*, *Three Colors: Blue*) depicting neutral interactions between two different couples and a family. Scenes were chosen to closely match the number of actors presented in each "traumatic" film clip and were compiled to an identical length (14 minutes). To prevent order effects, individual film clips were presented counterbalanced across participants of the TF and NF conditions.

Participants were informed in the study advertisement, letter of introduction, and informed consent process that study participations may include the presentation of film clips containing graphic material that could be disturbing, and that they were free to withdraw at any time without any disadvantages.

2.3.1.2. Physiological stress measurements

Physiological recordings were collected using an ActiveTwo amplifier (BioSemi, Amsterdam, The Netherlands) at a sampling rate of 2048 Hz. For heart rate (HR) measurement, a standard lead-II electrocardiogram (ECG) with two Ag/AgCl electrodes was used to collect a raw ECG signal. R-waves

were identified automatically by ANSLAB 2.6 (Wilhelm & Peyk, 2005) and edited manually for artifacts, false positives or non-recognized R-waves and were transformed into instantaneous heart rates (HR). To measure skin conductance level (SCL), two Ag/AgCl electrodes filled with isotonic electrode gel were attached to the proximal part of the palm of the participant's non-dominant hand (with an alternating current of 1 mA synchronized with the sampling frequency passed between the electrodes). The recorded signal was downsampled to 25 Hz, edited manually for artifacts and smoothed using a 1 Hz low-pass filter. As measures of primary interest, means of HR and SCL were calculated prior to (pre), during (peri), and following (post) film presentation.

2.3.1.3. Subjective stress ratings (STAI-S; Laux et al., 1981)

The state scale of the State-Trait-Anxiety Inventory was used to measure participants' change in anxiety levels in response to film presentation. The STAI-S is a brief self-report measure which is used to ascertain momentary feelings of apprehension, nervousness, tension, and worry. The questionnaire consists of 20 items, which are rated on a 4-point-Likert scale ranging from 1 ("not at all") to 4 ("totally agree"). The total score ranges from 20 to 80, with a score of 20 indicating a very low state anxiety level and 80 indicating a very high state anxiety level. The scale has shown high internal consistency (α= .90-.94).

2.3.1.4. Impact of Events Scale (IES-R; Maercker & Schützwohl, 1998)

The IES-R is a clinical questionnaire that measures symptoms of intrusive re-experiencing, hyperarousal, and avoidance. The questionnaire consists of 22 items which are rated on a 4-point scale (1 = "not at all" to 4 = "extremely"). Item scores are converted into a non-equidistant format (0, 1, 3, 5) resulting in a maximum total score of 110. The scale has shown satisfactory to high internal consistency as well as satisfactory convergent validity with a structured interview assessing PTSD symptoms. For the purpose of the current study, participants were instructed to rate IES-R items with reference to the presented film.

2.3.1.5. Intrusion diary

Participants were asked to complete an intrusion diary starting upon awakening (day 1) for three consecutive days (days 1 to 3). They were instructed to carry the intrusion diary with them during the whole assessment period and document every intrusive memory immediately after its occurrence. Intrusions were defined as recurrent, sudden, spontaneous, and non-initiated memories of film scenes that are very vivid and consist of pictures, sounds, thoughts, words or sentences, feelings or combinations of those. Participants were carefully instructed that intrusions do not include reflective and conscious thinking or deliberate thoughts about the film scenes, which were not to be recorded in the diary. For each intrusion, participants provided a brief description of its content as well as the exact time and cause (if identifiable) of its occurrence. In addition, they were asked to rate intrusion-related distress (11-point scale

from 0 = "not at all" to 10 = "extremely"), valence (5-point scale from 1 = "happy" to 5 = "unhappy"), and arousal (5-point scale from 1 = "not aroused" to 5 = "aroused") experienced during the intrusion. As expected, the average intrusion frequency of NF participants (M = 0.43, SD = 0.65) fell below the level of one intrusion during the entire assessment period [$t(13) = 3.31$, $p = .006$] and all reported intrusions were rated with a distress level of 0. Thus, all subsequent analyses were focused on diary data of the TF group.

To reduce alpha inflation in our correlation analyses, we calculated a composite measure of intrusive re-experiencing as previously described by Wegerer, Kerschbaum, Blechert, and Wilhelm (2014). Each intrusion measure (overall frequency, mean distress, mean valence, and mean arousal) was transformed into z-scores to account for different scales. Thereafter, individual measures were summed up to form an index of intrusive re-experiencing (IR index). For significant correlations between the IR index and sleep physiology, we report correlations for single intrusion measures in corresponding footnotes.

2.3.2. Polysomnographic assessment

2.3.2.1. Polysomnographic recordings and sleep stage scoring

Polysomnographic recordings were performed according to the guidelines provided by the AASM (2007) including EEG at frontal and central sites (F3, F4, and Cz according to the international 10-20 system) and submental EMG. EOG electrodes were placed on the lower right and upper left canthi to record combined vertical and horizontal eye movements. Signals were digitized at a sampling rate of 512 Hz and amplified by a wireless SOMNOtouch amplifier system (SOMNOmedics GmbH, Randersacker, Germany). Data were filtered online with a first-order high-pass filter at 0.3 Hz, a second-order Butterworth low-pass filter at 75 Hz, and a Notch-filter at 50 Hz. All electrodes were recorded referenced to Cz and were re-referenced offline to the average of both mastoids. A 0.3-35 Hz bandpass filter was applied offline for sleep stage scoring.

Visual sleep stage scoring was performed independently by two trained raters in accordance with the criteria provided by the AASM (2007) and using the Matlab-based toolbox FASST (fMRI Artefact Rejection and Sleep Scoring Toolbox; Leclercq, Schrouff, Noirhomme, Maquet, & Phillips, 2011). Epochs (20 s) were scored visually as wake, stage N1, N2, N3 (corresponding to SWS), stage R, and stage W. This epoch length deviates from the AASM criteria and was chosen to allow for overlapping windows of 4 s in the computation of spectral power density. Total sleep time (TST), absolute sleep stage durations, sleep onset latency (SOL), and minutes of wakefulness after sleep onset (WASO) were determined for further analyses.

2.3.2.2. Spectral analysis

The spectral analysis was performed using the Matlab-based toolbox EEGLAB (http://www.sccn.ucsd.edu/eeglab/). Prior to analyses, REM sleep epochs were rejected semi-automated on the basis of automatic detection of extremely large fluctuations (> 1000 µV) and thereafter applying a threshold of 5 standard deviations followed by visual identification of muscle and eye movement artifacts. Spectral power density was computed for each epoch using the pwelch function (50% overlap, Hamming window) with a resulting frequency resolution of 0.25 Hz (see also Sopp, Michael, & Mecklinger, 2018; Sopp et al., 2017). Spectral power density was averaged for the theta band (4.0– 7.0 Hz; Nuwer et al., 1998) across both frontal electrodes (F3 and F4). The distribution of mean frontal theta power was asymmetric and was thus log-transformed to conform to a normal distribution for correlation analyses (see Ackermann, Hartmann, Papassotiropoulos, de Quervain, & Rasch, 2015 for a similar approach).

2.4. Statistical analyses

Subjective and physiological responses during film presentation were analyzed by means of separate univariate analysis of variance (ANOVAs) including the factors Time (pre vs. post for subjective responses, pre vs. peri vs. post for physiological responses) and Group (TF vs. NF). When the sphericity assumption was violated, analyses include Greenhouse-Geisser corrections for nonsphericity with corrected *p*-values and uncorrected degrees of freedom. Group differences in sleep architecture were analyzed in a multivariate analysis of variance (MANOVA) including the factor Group (TF vs. NF) and the dependent variables WASO, N1, N2, N3, and REM sleep. Differences in TST, SOL, relative sleep stage durations (% TST) were analyzed by means of independent *t*-tests. To link REM sleep physiology with subsequent analogue PTSD symptoms, non-parametric correlation coefficients (Spearman's ρ) were computed between REM sleep duration, REM theta activity, IES-R subscales, and diary data (IR index). The alpha level for all analyses was set to .05. All statistical analyses were calculated using IBM SPSS Statistics 21 (IBM Corp., Armonk, NY, USA).

3. Results

3.1. Sample characteristics

Comparisons of demographic characteristics did not reveal any significant between-group differences (see Table 1). Moreover, participants of both groups demonstrated comparable characteristics in baseline sleep quality, sleep duration, sleepiness, diurnal preference, and depressive symptoms ($p > .160$).

Table 1 Sample characteristics

	Trauma film group ***n* = 16**	Neutral film group ***n* = 14**	Group comparison
Age	21.69 (.57)	22.14 (.65)	$t(28) = 0.53$, $p = .601$
Gender	8 ♀/ 8♂	8 ♀/ 6♂	$\chi(1) = 0.15$, $p = .696$
Average sleep duration (h)	7.78 (.25)	7.66 (.28)	$t(28) = 0.32$, $p = .753$
Sleep Quality (PSQI)	3.06 (.35)	2.57 (.33)	$t(28) = 1.02$, $p = .316$
Daytime Sleepiness (ESS)	6.94 (.82)	5.86 (.77)	$t(28) = 0.95$, $p = .349$
Circadian Preference (rMEQ)	14.56 (.58)	13.07 (.86)	$t(28) = 1.45$, $p = .161$
Depressive symptoms (PHQ-9)	2.13 (.49)	1.57 (.36)	$t(28) = 0.89$, $p = .382$

Note. PSQI= Pittsburgh Sleep Quality Index, ESS= Epworth Sleepiness Index, rMEQ= reduced Morningness-Eveningness Questionnaire, PHQ-9= Patient Health Questionnaire; standard errors are given in parentheses.

3.2. Validity of the film material

3.2.1. Physiological stress measures

To examine whether trauma film presentation induced a significant change in physiological stress measures, we subjected means of HR and SCL to separate ANOVAs. An ANOVA of HR including the factors Time (pre/peri/post) and Group (TF/NF) revealed a significant main effect of Time [$F(2, 54) = 5.01$, $p = .010$, $\eta^2_p = .16$] reflecting a significant rise in HR from pre-film to peri-film assessment [$t(28) = 2.79$, $p = .009$]. The analysis also yielded a significant interaction of Time and Group [$F(2, 54) = 3.47$, $p = .038$, $\eta^2_p = .11$] in the absence of a main effect of Group [$F(1, 27) = 1.02$, $p = .323$, $\eta^2_p = .04$]. Post-hoc tests confirmed that pre-film HR was comparable between both groups [$t(27) = 1.15$, $p = .256$]. Further analyses did not verify a significant difference in HR increase between TF and NF participants during film presentation [pre-peri-difference values: $t(27) = 0.92$, $p = .366$]. As such, our results do not provide evidence for a differential HR response to the trauma film. Nevertheless, HR responses were descriptively larger in TF participants ($M = 4.34$, $SD = 8.09$) than in NF participants ($M = 2.16$, $SD = 3.73$; see Figure 1A).

An ANOVA of SCL including the factors Time (pre/peri/post) and Group (TF/NF) revealed a significant main effect of Time [$F(2, 54) = 30.94$, $p < .001$, $\eta^2_p = .53$] and a significant interaction of Time and Group [$F(2, 54) = 5.56$, $p = .015$, $\eta^2_p = .17$; see Figure 1B] in the absence of a main effect of Group [$F(1, 27) = 1.16$, $p = .291$, $\eta^2_p = .04$]. The main effect of Time reflected a significant increase in SCL from pre-film to peri-film assessment [$t(28) = 6.23$, $p= .009$], which was maintained from peri-film to post-film assessment

[$t(28) = 0.13$, $p = .896$]. Consequently, mean post-film SCL was significantly higher than mean pre-film SCL [$t(28) = 5.11$, $p < .001$]. Post-hoc analyses confirmed that pre-film SCL was comparable between both groups [$t(27) = 0.70$, $p = .493$]. Further analyses demonstrated that TF participants exhibited a stronger increase of SCL during film presentation than NF participants [pre-peri-difference values: $t(27) = 2.28$, $p = .032$].

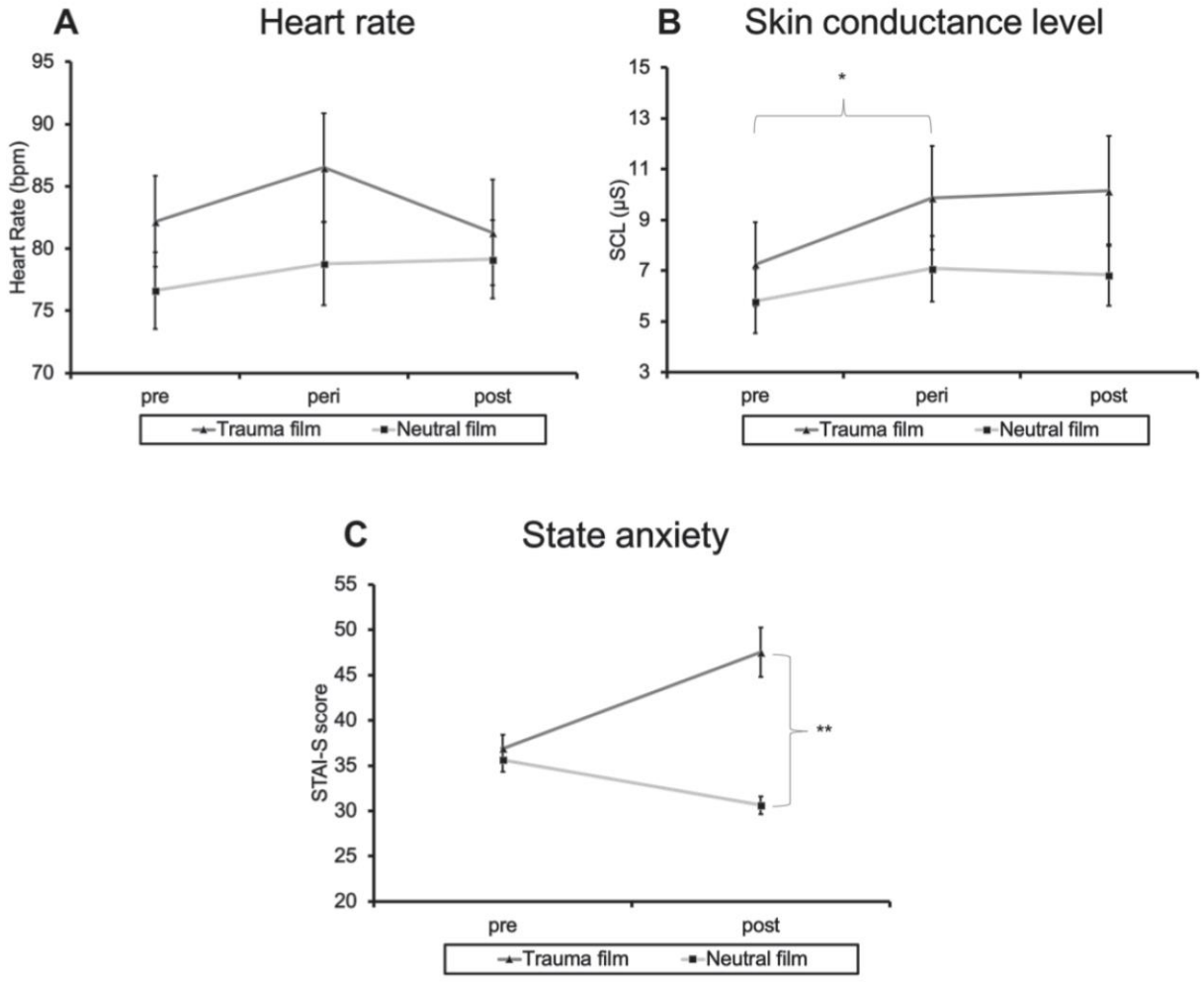

Figure 1. Group means of (A) heart rate, (B) skin conductance level and (C) state anxiety at pre, (peri,) and post film assessment. *Note:* Error bars represent standard errors, ** indicates $p < .01$, * indicates $p < .05$

3.2.2. Subjective stress ratings

To ascertain whether state anxiety levels were influenced by film presentation, we analyzed STAI-S scores in an ANOVA with the factors Time (pre/post) and Group (TF/NF) as independent variables. Analyses revealed a significant main effect of Group [$F(1, 28) = 17.45$, $p < .001$, $\eta^2_p = .38$] and a significant Time × Group interaction [$F(1, 28) = 29.34$, $p < .001$, $\eta^2_p = .51$] in the absence of a main effect of Time [$F(1, 28) = 3.80$, $p = .061$, $\eta^2_p = .12$]. Post-hoc analyses confirmed that groups did not differ at

pre-film assessment [$t(28) = 0.61$, $p = .548$]. Further analyses at post-film assessment revealed significantly enhanced STAI-S-scores in TF participants as compared to NF participants [$t(28) = 5.49$, $p < .001$; see Figure 1C]. Moreover, analyses within each group confirmed a significant increase in STAI-S scores from pre- to post-film assessment in TF participants [$t(15) = 4.35$, $p = .001$], whereas NF participants exhibited a significant decline in STAI-S scores [$t(13) = 3.86$, $p = .002$].

Overall, analyses of stress measures confirm the validity of the current trauma film material as state anxiety and SCL were found to increase differentially in TF participants. The lack of significant between-group differences in HR increase and overall modest effect sizes may be accounted for by blinded assignment. Although necessary, blinded film presentation may have provoked anticipatory responses in NF participants as they were not aware of the course of events in the film.

3.3. Comparison of post-film sleep characteristics

Table 2 Comparison of sleep stage durations

	Trauma film group ***n* = 16**	**Neutral film group** ***n* = 14**	**Group comparison**
SOL (min)	27.23 (5.71)	14.64 (2.73)	$t(28) = 1.99$, $p = .060$, $\eta^2_p = .11$
TST (min)	470.35 (6.23)	490.69 (3.42)	$t(28) = 2.86$, $p = .009$, $\eta^2_p = .21$
WASO (min)	14.38 (3.84)	5.21 (1.35)	$F(1, 28) = 4.53$, $p = .042$, $\eta^2_p = .14$
N1 (min)	39.73 (4.75)	42.10 (5.44)	$F(1, 28) = 0.11$, $p = .745$, $\eta^2_p < .01$
N2 (min)	219.25 (10.26)	263.02 (9.59)	$F(1, 28) = 9.54$, $p = .005$, $\eta^2_p = .25$
N3/ SWS (min)	122.98 (8.01)	99.21 (7.65)	$F(1, 28) = 4.53$, $p = .042$, $\eta^2_p = .14$
REM (min)	88.40 (4.75)	86.36 (6.19)	$F(1, 28) = 0.70$, $p = .793$, $\eta^2_p < .01$
N1 (% TST)	8.58 (1.10)	8.56 (1.09)	$t(28) = 0.01$, $p = .993$, $\eta^2_p < .01$
N2 (% TST)	46.50 (1.87)	53.55 (1.79)	$t(28) = 2.70$, $p = .012$, $\eta^2_p = .21$
N3/ SWS (% TST)	26.08 (1.65)	20.29 (1.63)	$t(28) = 2.49$, $p = .019$, $\eta^2_p = .18$
REM (% TST)	18.85 (1.05)	17.60 (1.25)	$t(28) = 0.77$, $p = .450$, $\eta^2_p = .02$

Note. TST = Total sleep time, WASO = Wake after sleep onset, N1 = NREM Stage 1, N2 = NREM Stage 2, N3 = NREM Stage 3 (corresponding to SWS), REM = REM sleep; standard errors are given in parentheses.

To investigate whether film presentation influenced overall sleep duration, we compared mean TST of TF and NF participants. In line with our hypothesis, TST was significantly reduced in TF participants as

compared to NF participants [$t(28) = 2.86$, $p = .009$, $\eta^2_p = .21$]. However, TF participants did not demonstrate a significantly delayed sleep onset [$t(28) = 1.99$, $p = .060$, $\eta^2_p = .11$; see Table 2]. In a subsequent step, we analyzed differences in sleep architecture in a MANOVA with sleep stage durations and WASO as dependent variables. The analysis yielded a significant main effect of Group [$F(5, 24) = 3.01$, $p = .030$, $\eta^2_p = .39$]. As expected, TF participants experienced higher WASO times when compared to NF participants [$F(1, 28) = 4.53$, $p = .042$, $\eta^2_p = .14$]. Contrary to our hypotheses, groups did not differ with respect to REM sleep duration [$F(1, 28) = 0.70$, $p = .793$, $\eta^2_p < .01$]. However, significant group differences emerged for N2 sleep [$F(1, 28) = 9.54$, $p = .005$, $\eta^2_p = .25$] and SWS [$F(1, 28) = 4.53$, $p = .042$, $\eta^2_p = .14$] indicating that TF participants experienced shorter N2 sleep and longer SWS than NF participants. Analyses of sleep stage durations relative to TST (% TST) revealed the same pattern of significant group differences (see Table 2).

3.4.1. Exploratory correlation analyses of subjective stress response and REM sleep duration

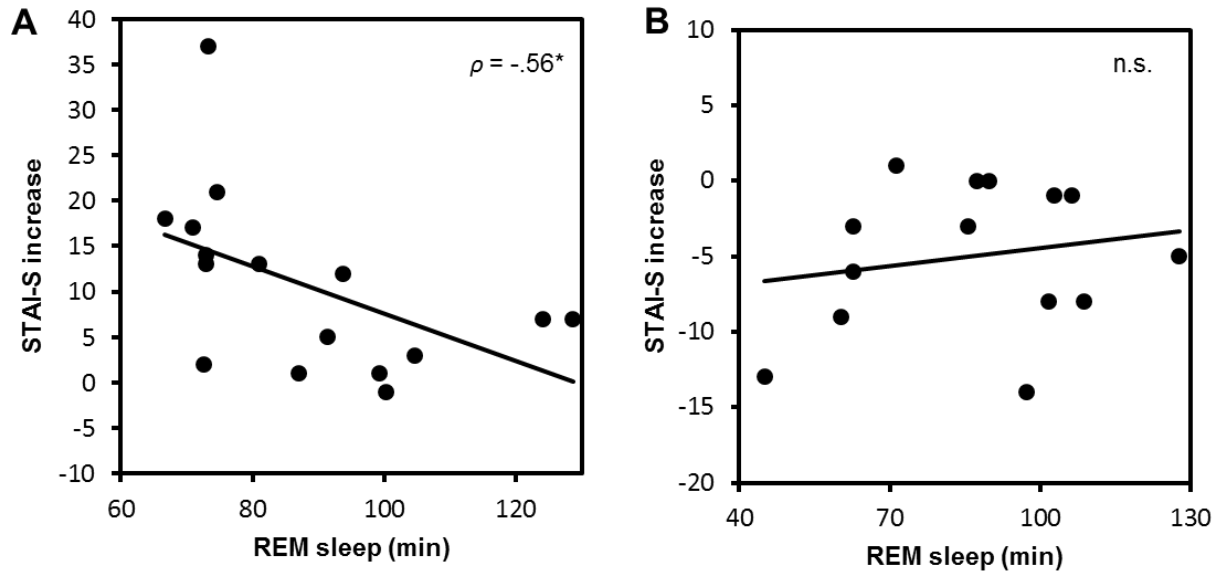

Figure 2. Correlation between STAI-S increase and REM sleep in the (A) TF and (B) NF group. *Note*: The correlation in the TF group (A) remains significant ($\rho = -.56$, $p = .029$) after exclusion of the univariate outlier (STAI-S increase = 37); ** indicates $p < .01$, * indicates $p < .05$.

As between-group analyses did not confirm a significant difference in REM sleep duration, we conducted additional analyses to examine potential associations between peri-"traumatic" stress responses and subsequent REM sleep duration in the TF group. To this end, we correlated the increase of STAI-S scores during film presentation (pre-post difference) with REM sleep duration. Analyses revealed a significant negative correlation ($\rho = -.56$, $p = .023$) indicating that higher increases in state anxiety were linked to a lower REM sleep duration (see Figure 2A). Corresponding analyses in the NF group did not reveal a

significant correlation (ρ = .14, p = .632; see Figure 2B). Moreover, STAI-S difference scores were not significantly correlated to any other sleep stage measure in the TF group (.10 > ρ > -.26, p > .330).

3.4. Correlations between REM sleep physiology and analogue PTSD symptoms

3.4.1. Clinical symptom rating

Table 3 Correlations between REM sleep physiology and IES-R scores

	IES-R T1				IES-R T2			
	Intrusive re-exp	**Hyper**	**Avoid**	**Total**	**Intrusive re-exp**	**Hyper**	**Avoid**	**Total**
REM sleep (min)	ρ = -.42 (p = .109)	ρ =-.19 (p = .472)	ρ = -.32 (p = .222)	ρ = -.31 (p= .239)	ρ = -.19 (p= .494)	ρ = -.39 (p= .137)	ρ = -.62 (p = .010)	ρ =-.66 (p = .005)
REM theta (μV²/Hz)	ρ = -.36 (p= .170)	ρ = -.44 (p = .089)	ρ = -.28 (p = .300)	ρ = -.34 (p= .196)	ρ = -.56 (p = .024)	ρ = -.21 (p= .434)	ρ =.04 (p= .892)	ρ = -.28 (p =.291)

Note. IES-R= Impact of Events Scale – revised, T1= day 1, T2= day 4, Intrusive Re-exp= Intrusive re-experiencing, Hyper= Hyperarousal, Avoid= Avoidance.

Based on the dose-dependent effects of peri-"traumatic" stress responses on REM sleep duration, we examined potential correlations between REM sleep physiology (REM sleep duration and frontal theta power) and IES-R subscale scores (see Table 3). Our analyses revealed that REM sleep physiology was not significantly correlated with symptom ratings upon immediate awakening (T1). However, both REM sleep measures demonstrated significant correlations with symptom ratings on day 4. More specifically, a significant negative correlation was found between REM sleep duration and the IES-R total scale (ρ = -.66, p = .005; see Figure 3A), which was mainly evident for subjective symptoms of avoidance (ρ = -.62, p = .010). In addition, a significant negative correlation was evident between REM theta activity and subjective symptoms of intrusive re-experiencing (ρ = -.56, p = .024; see Figure 3B), which was not paralleled in IES-R total scores (ρ = -.28, p = .291). Thus, REM sleep duration was associated with decreased overall symptom levels whereas REM theta activity was selectively linked to decreased symptoms of intrusive re-experiencing.

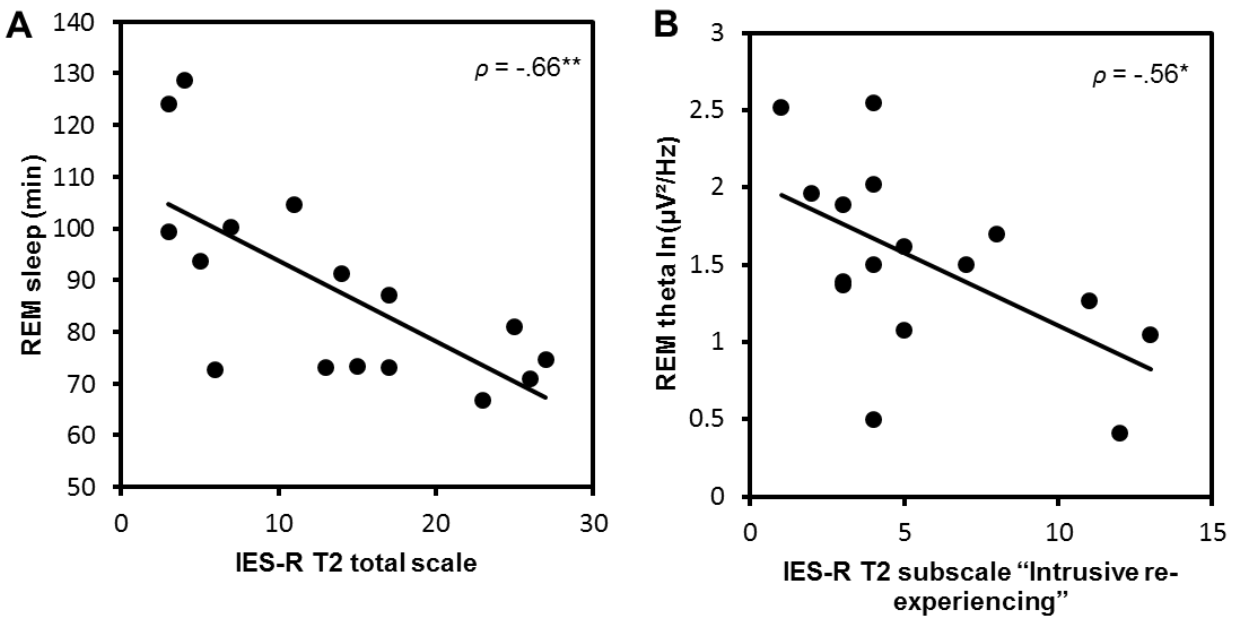

Figure 3. Correlation between (A) REM sleep and IES-R T2 total score and between (B) REM theta activity and IES-R T2 intrusion score. *Note*: IES-R= Impact of Events Scale – revised, T2= day 4; ** indicates $p < .01$, * indicates $p < .05$.

3.4.2. Intrusion frequency and distress ratings

In our final analyses, we examined correlations between REM sleep physiology and the IR index. Participants in the TF group reported 2.94 intrusions on average (*SD* = 2.49) and a mean distress rating of 3.59 (*SD* = 1.93). Mean valence and arousal ratings indicate that participants perceived intrusions as aversive ($M_{Valence}$ = 3.52, $SD_{Valence}$ = 0.71) and medium-to-low arousing ($M_{Arousal}$ = 2.18, $SD_{Arousal}$ = 0.93). Correlation analyses between IR index scores, REM sleep duration, and theta activity revealed a significant negative correlation between REM theta activity and IR index scores (ρ = -.78, p = .001[1]; see Figure 4B). The respective correlation between REM sleep duration and IR index scores failed to reach significance (ρ = -.44, p = .113; see Figure 4A). Thus, in parallel with our previous results (3.3.1.), only REM theta activity demonstrated a significant association with intrusive re-experiencing such that higher REM theta activity was linked to lower intrusive re-experiencing.

[1] Correlations for individual intrusion measures: frequency: ρ = -.31, p = .239; distress: ρ = -.69, p = .006; valence: ρ = -.32, p = .272; arousal: ρ = -.67, p = .009

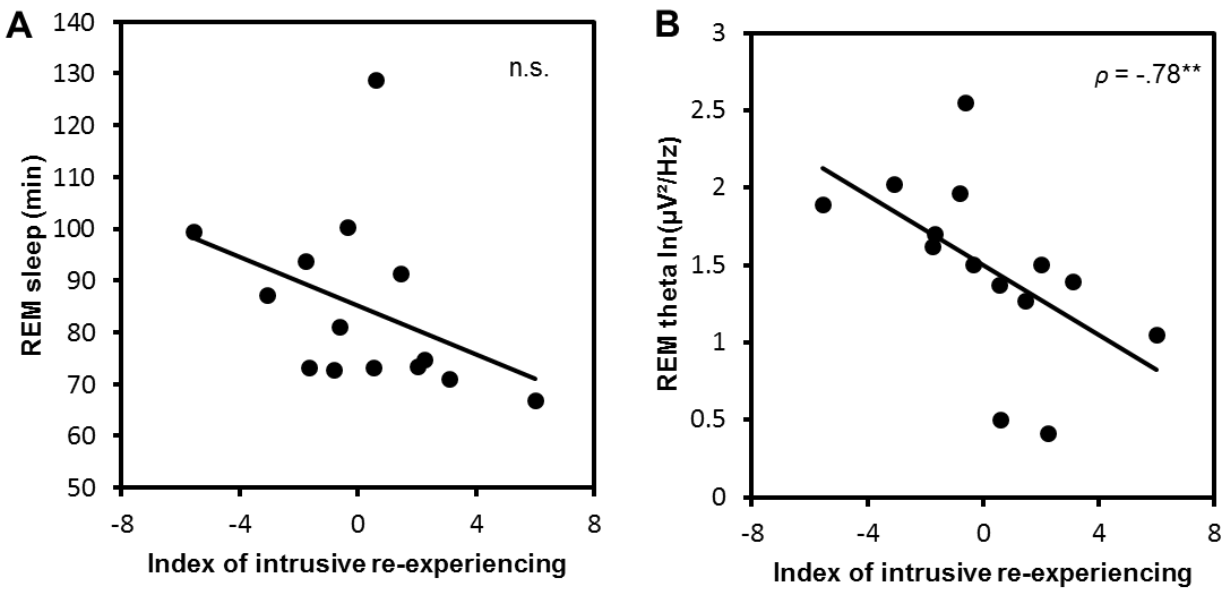

Figure 4. Correlations between (A) REM sleep and (B) theta power and IR index scores. *Note*: ** indicates $p < .01$, * indicates $p < .05$.

4. Discussion

The current study investigated whether exposure to an analogue traumatic event would impact sleep quality as well as specific aspects of sleep architecture (i.e. REM sleep). In addition, we examined whether REM sleep physiology would be linked to subsequent intrusive re-experiencing symptoms. Our results demonstrate alterations of sleep architecture in response to the trauma film that indicate partial impairments of sleep quality. However, these impairments did not include significant decrements in REM sleep duration. Correlation analyses revealed negative associations between REM sleep duration and subsequent analogue PTSD symptoms. Microstructural REM sleep analyses further delineate a selective association between REM theta activity and intrusive re-experiencing symptoms.

Regarding our first major objective which was to investigate effects of the trauma film on subsequent sleep quality, we were able to show that a lack of restful sleep may directly result from traumatic exposure. More specifically, we found that healthy individuals who were exposed to the trauma film showed reduced overall sleep time and enhanced wakefulness periods after sleep onset as compared to healthy individuals who watched a neutral film. These differences resulting from film presentation are particularly noteworthy as both variables (TST and WASO) show similar characteristic alterations in patients with chronic insomnia (Harvey, Stinson, Whitaker, Moskovitz, & Virk, 2008; Natale, Plazzi, & Martoni, 2009). Although groups did not differ in sleep onset latencies, TF participants demonstrated a delay in sleep onset well above 20 minutes which may similarly indicate insomnia-like characteristics (e.g. Lineberger, Carney, Edinger, & Means, 2006). Hence, if our results are extended to the emotional

impact of real life traumatic events, it may be assumed that clinically relevant sleep disturbances can arise as a direct result of traumatic exposure. Despite the abundance of findings linking posttraumatic sleep disturbances with psychopathological outcomes of traumatization (Babson & Feldner, 2010; Germain, 2013), it has not been sufficiently investigated to which extent these effects are driven by pretraumatic sleep disturbances and/or preexisting vulnerability factors (Breslau, Roth, Rosenthal, & Andreski, 1996; Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2010; Gehrman et al., 2013; Mellman & Hipolito, 2006; Wild et al., 2016). As such, our results make a unique contribution to the current literature by demonstrating reduced sleep quantity after "traumatic" exposure in healthy robust sleepers who lack any indication of previous sleep disturbances.

Beyond these changes in overall sleep quantity, we hypothesized that "traumatic" exposure would specifically result in decreased REM sleep duration. However, in contrast to accounts of fragmented REM sleep after trauma exposure (Mellman et al., 2002; Mellman et al., 2007; Stocker et al., 2016), our results do not establish a significant between-group difference in REM sleep duration. Although unexpected, this finding is in line with two previous studies examining changes in sleep physiology after the presentation of emotional films (Talamini, Bringmann, de Boer, & Hofman, 2013; Werner et al., 2015). Neither study reported a significant effect of film presentation on REM sleep, apart from an altered distribution of REM sleep across both night halves (Talamini et al., 2013). Further paralleling the findings of Talamini et al. (2013), we found an enhancement of SWS duration in response to the trauma film, which may reflect an increase in homeostatic sleep pressure after exposure to an experimental stressor. Moreover, this enhancement of SWS indicates that an aspect of sleep architecture was improved - rather than impaired - after "traumatic exposure". Thus, different aspects of sleep (i.e. TST and % SWS), which contribute to overall subjective quality (Keklund & Åkerstedt, 1997) may be impacted by trauma in opposite ways. This pattern of changes should be investigated further in future studies.

Although planned comparisons did not reveal any significant between-group differences in REM sleep, exploratory analyses suggest that stronger anxiety responses to the trauma film may be linked with decreased REM sleep duration. These findings indicate that effects of traumatic exposure on REM sleep may depend on peritraumatic stress responses. Interestingly, rodent studies similarly suggest that experimentally-induced stress responses provoke selective reductions in REM sleep (for a review see Pace-Schott et al., 2015). Moreover, peritraumatic stress responses have been repeatedly linked to adverse psychopathological outcomes of trauma exposure (for a review see Ozer, Best, Lipsey, & Weiss, 2003). Thus, our results reinforce the perspective that alterations in REM sleep architecture may specifically emerge in those individuals who are less efficient in adapting to environmental stressors.

The second major objective of our study was to examine links between post-"traumatic" REM sleep characteristics and subsequent analogue PTSD symptoms. In line with our hypotheses and previous work of others (Kleim et al., 2016; Woud et al., 2018), we found that sleep was correlated with reduced overall symptoms and symptoms of intrusive re-experiencing, which further challenges the proposal that sleep deprivation may be a useful intervention in the early aftermath of trauma (Porcheret et al., 2015). Moreover, in agreement with the study by Kleim et al. (2016), we found that associations between sleep and decreased re-experiencing symptoms only emerge across time (IES-R T2) and are not evident immediately upon awakening (IES-R T1). This pattern of results may suggest that sleep in the early aftermath of trauma initiates processes with a prolonged time course. Kleim et al. (2016) argue that these processes may be related to systems consolidation during NREM sleep as evidenced by negative correlations between intrusion frequency and N2 sleep physiology in their study. Here, we found complementary indications of an involvement of REM-sleep-dependent processes as reflected in negative correlations between REM theta power and intrusive re-experiencing. These findings are in line with theoretical frameworks proposing a specific role of REM theta activity in emotional memory processing (Genzel et al., 2015; Hutchison & Rathore, 2015; Walker & van der Helm, 2009). The outcomes of this processing are assumed to be twofold; resulting in the strengthening of explicit memory retrieval and a downregulation of the affective charge associated with these memories (Walker & van der Helm, 2009). Based on this assumption and on the finding that PTSD patients show reduced REM theta activity (Cowdin et al., 2014), it has been proposed that REM sleep may be involved in integrative processing of trauma memories (Goldstein & Walker, 2014), ultimately resulting in natural recovery from trauma (Stickgold & Manoach, 2017). Our results for the first time offer support for this hypothesis by demonstrating that REM theta activity is related to reduced intrusive re-experiencing.

Integrating these novel indications with the findings of Kleim et al. (2016) suggests that both REM- and NREM-sleep-related processes make unique contributions to trauma-related memory processing. An important emerging agenda resulting from these accounts is to examine how basic sleep-related processes tie in with natural recovery from trauma. According to cognitive models of PTSD, natural recovery from trauma may entail spontaneous updating of trauma memory traces during early episodes of intrusive re-experiencing (Brewin et al., 2010; Ehlers & Clark, 2000; Foa, Huppert, & Cahill, 2006). Further extending this view, it could be hypothesized that sleep facilitates consolidation of spontaneously updated trauma memories (Deliens et al., 2013) such that corrective information (e.g. absence of anticipated harm) is incorporated more efficiently into trauma memory representations (Foa et al., 2006; McLean & Foa, 2011). In addition, sleep could promote natural recovery by enabling integration of the traumatic experience into pre-existing memory networks (Stickgold & Manoach, 2017). In line with this hypothesis, sleep has been found to facilitate qualitative changes of newly encoded memory traces, e.g. by creating

new links between overlapping features of events (for a review see Stickgold & Walker, 2013). These qualitative changes of trauma memory representations could result in an attenuation of negative emotional components which may, in turn, reduce the likelihood and distress of intrusive memories (Kleim et al., 2016). Alternatively, sleep-related processes may result in a downregulation of autonomic reactions to trauma-related stimuli (Pace-Schott et al., 2011), which could reduce trauma-associated fear responses and distress levels associated with intrusive memories (Pace-Schott et al., 2015; Pace-Schott et al., 2009; Stickgold & Manoach, 2017). These different hypotheses should be tested in future analogue studies by integrating different trauma memory measures (e.g. explicit memory tests, psychophysiological measurements during re-exposure to trauma cues). Moreover, future studies are required to investigate the extent to which sleep-related effects on analogue PTSD symptoms are modulated by trait factors. Importantly, the current pattern of delayed correlations between REM sleep physiology and symptom levels opposes the conception that high REM theta power reflects the presence of resilience traits without any causal link to sleep-related processes. On the basis of such a trait account, one would predict a temporally stable pattern of correlations between REM sleep physiology and symptom levels. By contrast, our findings suggest that post-"traumatic" REM theta activity indexes the onset of continuing sleep-related processes that alleviate intrusive re-experiencing symptoms over time.

Although our results reveal a remarkably defined pattern of correlations between REM theta power and analogue re-experiencing symptoms, it is important to address certain limitations. First of all, it is not possible to directly link the current effects to consolidation processes as we did not assess objective indicators of memory. Nevertheless, selective correlations between REM theta power and intrusive re-experiencing strongly suggest an involvement of memory processes. To substantiate these indications, future studies should adopt experimental procedures which are suited to probe subsequent memory performance for trauma-related stimuli (Ehlers et al., 2006; Holz, Lass-Hennemann, Streb, Pfaltz, & Michael, 2014; Michael & Ehlers, 2007). Secondly, it is important to note that correlations between REM sleep physiology and IES-R T2 subscales were not consistent for different parameters (REM theta power and REM sleep duration). Hence, we were not able to demonstrate any selective associations between REM sleep duration and re-experiencing symptoms. Previous accounts have argued that microstructural sleep features are more precise markers of consolidation processes than sleep stage durations (Cox, Hofman, & Talamini, 2012), which may account for a broader, less circumscribed association between REM sleep duration and analogue PTSD symptoms. Nevertheless, future studies are required to replicate the current correlational patterns in a confirmatory manner, especially given that the current analyses did not control for multiple comparisons. Thirdly, our results are limited by the restricted number of participants (N = 30) that entered our analyses. In contrast to previous studies (Kleim et al., 2016; Porcheret et al., 2015), we examined sleep-related effects in a highly controlled setting, which included

psychophysiological recordings during film presentation and nocturnal, laboratory-based PSG assessment. These design considerations placed restrictions on our overall sample size which we aimed to account for by means of non-parametric correlation analyses. Despite these efforts, our findings warrant replication in a larger sample of participants. Finally, it is important to consider that the current study used an analogue procedure which limits the application of our findings to clinical populations. The trauma film paradigm has been shown to reliably induce short-term symptoms of intrusive re-experiencing allowing the investigation of research question that require prospective study designs (Holmes & Bourne, 2008; Holz, Lass-Hennemann, & Michael, 2017; James et al., 2016; Lass-Hennemann, Peyk, Streb, Holz, & Michael, 2014; Streb, Mecklinger, Anderson, Lass-Hennemann, & Michael, 2016). Nevertheless, further studies are needed to examine whether the current associations between REM sleep physiology and posttraumatic stress symptoms are also evident in clinical samples.

Despite these limitations, our results add to the current literature in providing further evidence that posttraumatic sleep disturbances may accelerate subsequent posttraumatic stress symptoms (Kleim et al., 2016; Woud et al., 2018). Firstly, our findings suggest that trauma can directly impact sleep duration. Secondly, our correlational results indicate that processes occurring during REM sleep reduce subsequent re-experiencing symptoms. Thus, it may be assumed that trauma-associated sleep disturbances restrict sleep-related processes which contribute to the alleviation of re-experiencing symptoms. These considerations are in line with clinical research (Babson & Feldner, 2010) and raise the importance of delivering early interventions aimed at promoting restful sleep. Additionally, our findings highlight several important directions for future research. In particular, further studies are required to examine the intermediary processes by which sleep helps to alleviate symptoms of intrusive re-experiencing. Moreover, the time course and dynamics of sleep-related trauma memory processing call for further investigation. Although theoretical frameworks propose a dual role of sleep disturbances in promoting the onset and maintenance of PTSD (Pace-Schott et al., 2015), this longitudinal perspective remains largely unexplored at present. Thus, future experiments should investigate the effects of sleep disturbances on maintaining processes of posttraumatic stress symptoms (e.g. deficits in extinction learning) as this line of research may provide important indications for the improvement of exposure-based treatments.

Declaration of Interests

None

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