ORIGINAL ARTICLE

Sleep-Directed Hypnosis Improves Subjective Sleep Quality but not Extinction Memory After Exposure to Analog Trauma

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Abstract

Background Evidence-based treatments of posttraumatic stress disorder (PTSD) aim to promote fear extinction learning. Post-learning sleep, particularly slow wave sleep (SWS), promotes memory consolidation and recall. Thus, boosting SWS might strengthen extinction recall. The current study investigated whether sleep-directed hypnosis designed to increase SWS and sleep quality improves extinction recall and reduces analog PTSD symptoms.

Method In two subsamples (remote/laboratory), 211 healthy individuals underwent fear conditioning with a traumatic film clip. On the next evening, they underwent extinction training. Thereafter, the experimental group received sleep-directed hypnosis, whereas the control group listened to a control text. Extinction recall and generalization and film-related intrusions and rumination were assessed on the following morning.

Results Subjective sleep quality declined following exposure to an aversive film. No group differences were found in SWS though exploratory analyses indicated less rapid eye movement sleep after hypnosis. After hypnosis, the experimental group reported improved sleep quality, whereas the control group showed a further deterioration. Hypnosis had no effects on extinction retention and generalization nor on analog intrusions and rumination.

Conclusion The current results indicate that sleep-directed hypnosis may be beneficial for improving subjective sleep quality after trauma but not for enhancing extinction memory and reducing analog PTSD symptoms.

Keywords Sleep · SWS · Hypnosis · Fear conditioning · Extinction · PTSD · Intrusions

Introduction

The psychological therapies for posttraumatic stress disorder (PTSD) with the strongest evidence of effect are those in which the memories of the traumatic event are actively processed like in trauma-focused cognitive behavioral therapies or in eye movement desensitization and reprocessing (Hamblen et al., 2019). These therapies are commonly referred to as trauma-focused psychotherapy (TF-PT). While TF-PT works for many patients, unfortunately, a significant number continues to have residual symptoms following therapy (Schnurr & Lunney, 2019; Schottenbauer et al., 2008). Thus, it has been argued that research investigating interventions with the potential to boost the effectiveness of TF-PT is crucial (Michael et al., 2019). PTSD is characterized by disturbed emotional learning and memory resulting in re-experiencing of the traumatic event in the present, avoidance of trauma reminders associated with re-experiencing of the traumatic event, and a persistent sense of current threat (Ehlers et al., 2004; WHO, 2019). It is assumed that a central mechanism in the pathogenesis of PTSD is associative learning that leads to the formation of strong aversive memories (Craske et al., 2018). During the traumatic event, individuals are assumed to acquire fear associations between neutral stimuli that are present in the environment (conditioned stimuli, CS) and the existential threat of trauma (unconditioned stimulus, US). Whenever individuals are subsequently faced with stimuli that resemble the CS, they experience a complex conditioned reaction in the

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form of unwanted, distressing (intrusive) trauma memories and perceptions of ongoing threat. The occurrence of intrusive memories is strengthened by abstract, repetitive thinking about the trauma (i.e., rumination), which often arises in an effort to control intrusions (Laposa & Rector, 2012). During TF-PT, fear associations are reactivated via various methods, thereby exposing patients to the trauma memory. Since this reactivation takes place in a safe setting (i.e., in the absence of the US), it is assumed to initiate fear extinction, resulting in a dissipation of the conditioned fear response. The memory trace acquired during extinction competes against, but does not erase, the original traumatic memory (de Quervain et al., 2017). Thus, it is of particular importance that the extinction memory is well consolidated so that later the extinction memory and not the traumatic memory will be recalled.

Recent accounts propose that the consolidation of extinction memory can be enhanced by manipulating post-extinction sleep. Research of the past two decades has firmly documented a strengthening effect of post-encoding sleep on subsequent memory performance (Rasch & Born, 2013). These effects are assumed to emerge due to offline consolidation processes occurring during slow wave sleep (SWS). Specifically, models propose that slow oscillations during SWS drive reactivation and subsequent redistribution of hippocampal memory traces (Diekelmann & Born, 2010). Correspondingly, first studies indicate that SWS may play a crucial role for reactivation and reprocessing of fear memory traces (Hauner et al., 2013; He et al., 2015) though this assumption is challenged by research suggesting rapid eye movement (REM) sleep to be critical for the consolidation of extinction memory (e.g., Menz et al., 2016; Spoormaker et al., 2012). Moreover, preliminary findings suggest that sleep facilitates the generalization of extinction to unextinguished stimuli (Pace-Schott et al., 2009).

Although manipulating post-extinction sleep appears to be a promising approach to enhance extinction – thereby potentially boosting treatment effects of TF-PT – no study to date has investigated the impact of sleep-enhancing interventions on the retention and generalization of extinction. An intervention that is easily administered and has been shown to directly affect objective and subjective sleep quality is sleep-directed hypnosis. Across several experiments, Cordi and colleagues investigated the impact of a hypnotic suggestion that aims to increase sleep depth on daytime and nighttime sleep (Cordi et al., 2014; 2015; 2020). They found that the suggestion increased subjective sleep quality (Cordi et al., 2020) and SWS duration (Cordi et al., 2014; 2015; 2020). The latter finding is particularly interesting since SWS has been implicated in memory consolidation (Hu et al., 2020) and facilitation of fear extinction (Hauner et al.,

2013; He et al., 2015; but see Ai et al., 2015, for contrasting evidence).

Based on these findings, the current study sought out to investigate the impact of sleep-directed hypnosis on subsequent sleep quality as well as retention and generalization of extinction. Healthy participants were subjected to a trauma-adapted fear conditioning paradigm, resulting in the acquisition of a conditioned fear response to neutral stimuli that predicted the occurrence of a traumatic film clip. After extinction training, participants were either subjected to sleep-directed hypnosis or a control condition prior to a full night of sleep. On the following day, participants completed an extinction retention and fear renewal test for previously extinguished and unextinguished conditioned stimuli. Sleep quality, intrusive memories, and rumination were assessed repeatedly throughout the experiment. We predicted to find higher SWS duration and sleep quality following sleep-directed hypnosis as compared to the control condition. Moreover, we expected that participants receiving sleep-directed hypnosis would show stronger retention and generalization of extinction. Finally, we expected that these effects would transfer to intrusive memories and ruminative thoughts such that participants would report fewer intrusive memories and ruminative thoughts about the traumatic clip after sleep-directed hypnosis.

Methods

Sample

The sample consists of two subsamples: The first subsample completed the experiment under remote conditions (using online stimulus presentation) without psychophysiological assessment, whereas the second subsample completed the experiment at the laboratory and underwent the hypnosis intervention at home. Psychophysiological assessment was carried out for the second subsample during the experiment and during sleep. Criteria for study eligibility were: age between 18 and 35 years; normal or corrected-to-normal vision; sufficient German language skills; no current or chronic mental disorder; no acute physical illness; no lifetime trauma exposure. Participants were requested to refrain from alcohol and drug consumption during the experimental days. In the laboratory subsample, participants were additionally required to be highly hypnotizable since previous research has shown that sleep-directed hypnosis is not effective in low hypnotizable individuals (Cordi et al., 2014; 2015; 2020). Participants received financial compensation or student credits for participation (Online Experiment: € 20; Lab Experiment: € 75).

Remote Subsample

One hundred twenty-two undergraduate university students took part remotely. Eleven participants were excluded due to technical problems. Four additional participants were removed from analyses based on their performance during acquisition training.[1] The resulting sample comprised 107 participants: hypnosis group: $n=55$, 46 females, 9 males; control group: $n=52$, 41 females, 11 males. Please note that non-related analyses based on this subsample are reported in another publication (Friesen et al., 2022).

Laboratory Subsample

Eighty-nine undergraduate university students took part at the laboratory. Nine datasets had to be excluded due to drop-out ($n=5$) or technical problems ($n=4$). Additional four datasets were removed from analyses due to failed contingency learning during acquisition training.[1] Two participants were excluded due to an insufficient amount of sleep (i.e., 2:55 h:m, more than five SDs below the group mean) or high levels of distress during study participation due to a breakup in between study sessions. The final sample comprised 74 participants: hypnosis group: $n=38$, 23 females, 15 males, $M_{age}=23.32$ ($SD=3.09$); control group: $n=36$, 27 females, 9 males, $M_{age}=23.11$ ($SD=3.12$).

Descriptive statistics of hypnotizablity (HGSHS-A; Shor & Orne, 1963), baseline sleep quality (SQS; Snyder et al., 2018) and trait anxiety (STAI-T; Laux et al., 1981) are provided in Table 1. Groups did not differ in baseline characteristics (p's $>=0.280$). However, the laboratory subsample was more hypnotizable, $t_w(165.83)=5.18$, $p<.001$ (due to deviations in inclusion criteria; see also below), and reported higher sleep quality, $t_w(169.73)=2.18$, $p=.030$, than the remote subsample.
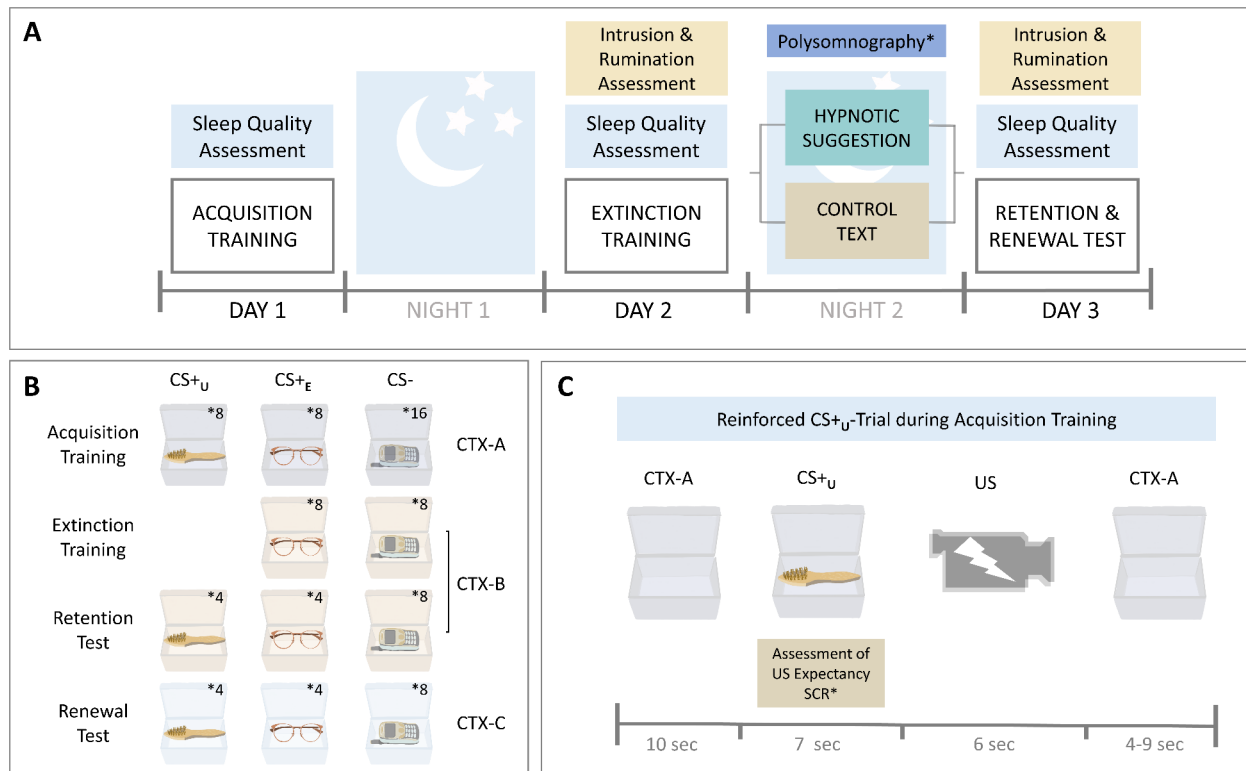
Experimental Procedure

Screening

Participant recruitment and screening was conducted online via SoSci Survey (Leiner, 2019) and telephone. Hypnotizability was determined by means of the HGSHS-A (Shor & Orne, 1963; German version by Bongartz, 1985; details are provided in the Supplementary Material). Based on their scores, participants were divided into low-to-medium hypnotizability (HGSHS-A $<=6$) and medium-to-high

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[1] Participants showing no indication for differential contingency learning, i.e., negative US expectancy difference scores for mean CSs + and CS- at the final acquisition trial, were excluded from analyses.

Table 1 *Sample characteristics according to Group and Subsample*

Subsample	Variable	Hypnosis group (n=93)			Control group (n=88)			Test statistics		
		M	SD	%	M	SD	%	X^2/t_w	df	p
Remote (n=107)	HGSH-A	7.33	2.42	-	7.20	2.09	-	-0.28	98.85	.777
	% HGSH-A≥7	-	-	61.82	-	-	61.54	<0.01	1	>.999
	% Females	-	-	83.64	-	-	78.85	0.15	1	.699
	SQS	7.06	1.29	-	7.04	1.41	-	-0.06	100.87	.951
	STAI-T	38.55	9.75	-	37.67	9.74	-	-0.46	104.68	.644
Laboratory (n=74)	HGSH-A	8.58	1.40	-	8.81	1.26	-	0.71	65.99	.480
	% HGSH-A≥7	-	-	100	-	-	100	-	1	-
	% Females	-	-	60.53	-	-	75.00	1.17	1	.280
	SQS	7.51	1.06	-	7.40	1.28	-	-0.40	67.94	.688
	STAI-T	36.89	6.90	-	36.33	9.81	-	-0.28	62.71	.780
Total (n=181)	HGSH-A[a]	7.84	2.15	-	7.83	1.97	-	-0.04	167.95	.971
	% HGSH-A≥7[a]	-	-	77.42	-	-	77.27	<0.01	1	>.999
	% Female[b]	-	-	74.19	-	-	77.27	0.10	1	.757
	SQS[c]	7.24	1.22	-	7.19	1.36	-	-0.28	172.04	.777
	STAI-T[b]	37.88	8.71	-	37.13	9.73	-	-0.55	173.85	.585

Note. HGSH-A=hypnotizability; SQS=baseline sleep quality, STAI-T=trait anxiety. [a] Difference between subsamples significant (Remote < Laboratory; $p<.001$). [b] No differences between subsamples (p's > .05). [c] Difference between subsamples significant (Remote < Laboratory; $p=.030$).

Fig. 1 Experimental procedure. *Note.***1 A**: Task procedure across experimental days. In both subsamples, participants filled out a sleep quality questionnaire at the beginning of each experimental day. After acquisition and extinction training, participants slept at home during night-time while the experimental groups received either a hypnotic suggestion or a control text before sleep in night 2. (*) Polysomnographic assessment was carried out only in the laboratory subsample. After sleep manipulation, participants underwent retention and renewal test on Day 3. Film-related intrusions and rumination from the acquisition training were assessed by questionnaires (Day 2 & 3) and an intrusion provocation task (Day 3). **1B**: Conditioned stimuli and contexts across the conditioning phases. During the conditioning phases (except for extinction training), three objects were presented in one of three boxes. (*) The number of trials for each conditioned stimulus are indicated by asterisks. One of the two aversive conditioned stimuli (CS+) was presented during extinction training (CS $+_E$) while the other was not (CS $+_U$). The boxes differed in type and color as well as different flooring in the background, serving as conditioning contexts specific to the phases. Retention was tested in the same context in which extinction training was carried out and thereafter in a new (renewal) context. **1 C**: Stimulus presentation in a reinforced CS $+_U$ trial during acquisition training. An empty box (context) was presented for 10 s. Then, the conditioned stimulus appeared in the box for 7 s together with the US expectancy scale. (*) SCR assessment was carried out only in the laboratory subsample. The traumatic film clip (US) was presented immediately after stimulus offset. During the inter-trial interval, the context was presented for 4 to 9 s. CTX = Conditioning context, CS $+_U$ = aversive conditioned stimulus – unextinguished, CS $+_E$ = aversive conditioned stimulus – extinguished, CS- = safety stimulus, US = unconditioned stimulus, SCR = skin conductance response

hypnotizability (HGSHS-A> = 7; Bongartz, 1985; Cordi et al., 2014). Group assignment was conducted pseudo-randomly to control for effects of baseline sleep quality and hypnotizability (see Supplementary Material).

General Procedure

The experiment was carried out on three consecutive days (see Fig. 1 A). During this period, participants were instructed to keep a regular sleep schedule at home and to restrict their sleep time to eight hours. The remote subsample completed all experimental sessions web-based via *Labvanced* (Finger et al., 2017); the laboratory subsample completed all

experimental sessions using *E-Prime 2.0* (Psychology Software Tools Inc., USA). On Day 1 (between 2 and 6 PM for the remote subsample and at 10 AM for the laboratory subsample), participants went through the first experimental session including assessment of sleep quality of the preceding night and fear acquisition training. On Day 2 (between 6 and 10 PM for the remote subsample and at 8.30 PM for the laboratory subsample), participants rated their sleep quality again and then, filled out a questionnaire assessing intrusions and rumination related to the aversive film clip from acquisition training (Intrusive Memories Questionnaire, IMQ). Thereafter, they underwent fear extinction training. The laboratory subsample was subsequently prepared for polysomnography (PSG) in accordance with the AASM

guidelines (Berry et al., 2012) and went home (for details, see Supplementary Material). Participants received an audio file comprising either a hypnotic trance induction and a suggestion to sleep deeper (hypnosis group) or a control text (control group; see Cordi et al., 2014). They were instructed to listen to the audio file after preparing to go to sleep. On Day 3 (between 8 AM and midday for the remote subsample and at 8.30 AM for the laboratory subsample), sleep quality and film-related intrusions and rumination were re-assessed as described above. Afterwards, participants completed retention and renewal tests, and, finally, an intrusion provocation task (IPT).

Fear Conditioning Phases

Prior to acquisition training, participants viewed an aversive film clip (US) about a female chef, who sustains severe burns during a kitchen accident accompanied by a piercing scream. The film clip was taken from a social marketing campaign by Ontario's Workplace Safety and Insurance Board (https://www.youtube.com/watch?v=tN2gpRcFKAQ) and was successfully used in previous fear conditioning experiments (Landkroon et al., 2019). Subsequently, participants were informed that they would see different objects of which some would be followed by an excerpt of the film clip. Three everyday objects (i.e., a brush, a cellphone and glasses), serving as to-be conditioned stimuli (CS), were presented in a box (acquisition context; see Fig. 1B). During CS presentation, participants were asked to rate their expectation to see the aversive film clip afterwards (very low expectancy [0] – very high expectancy [100]). In the laboratory subsample, skin conductance was recorded simultaneously (for details, see Supplementary Material). The presentation of the two CSs + was followed by a short version of the clip. The CS- was never followed by the clip. Acquisition training was divided into two sequential blocks, each consisting of eight CS+ (one of the two CSs+) and eight CS- trials (see Fig. 1 C, for trial procedure) in randomized order. US presentations followed the CSs + with a reinforcement ratio of 75%.

During extinction training, one of the two CSs+ (extinguished CS+ [CS + $_E$]) and the CS- were presented eight times each in random order in a new box (extinction context). None of the CS presentations was followed by the US. As in acquisition training, retention test and renewal test were divided into two sequential blocks, during which one of the two CSs+ (CS + $_E$ or the unextinguished CS+ [CS + $_U$]) and CS- were presented four times each. The US was not presented in any of the trials. During the retention test, CSs were shown in the extinction context. During renewal test, CSs were presented in a new box (renewal context). The

order of CSs + blocks during acquisition training and retention and renewal test as well as the contexts were balanced across subjects. This design was adapted from Milad et al. (2007) to assess generalization of extinction (to the CS + $_U$) during sleep (for further information, see Supplementary Material). Secondary subjective fear indices (i.e., arousal, fear and valence ratings) were measured in the remote subsample but will not be reported in the context of the current analyses.

Measurement of Film-Related Intrusions and Rumination

Intrusions and ruminative thoughts associated with the aversive film scene from acquisition training were assessed using a variant of the Intrusive Memory Questionnaire (IMQ; Michael & Ehlers, 2007). The questionnaire was adapted to assess frequency, duration (in seconds) and distress (not at all [0] – extremely [100]) of intrusions and rumination. Intrusion frequency was additionally assessed using an IPT (James et al., 2015; Michael et al., 2005). During the task, participants were re-exposed to the first four seconds of the film clip from acquisition training. After viewing the clip, they were instructed to close their eyes for two minutes. Finally, they were asked to rate intrusion frequency, duration and distress using the IMQ rating scales. Intrusion and rumination indices (sum scores of Z-standardized frequency, duration, and distress items) were calculated for further analyses.

Data Preparation and Analyses

Based on sleep stage scoring and reports on time in bed during Night 2 in the laboratory subsample, the amount of time spent in sleep stages (minutes and % of total sleep time [TST]), sleep efficiency, sleep onset latency (SOL), wake after sleep onset (WASO), and sleep stage latencies were calculated (for details, see Supplementary Materials). In accordance with Cordi et al. (2020), spectral power of slow wave activity (SWA; 0.5-4 Hz) relative to total power (0.5–50 Hz) during non-rapid eye movement sleep stages 2 and 3 (N2 and N3 [i.e., SWS]) was extracted and calculated for the entire sleep phase and the first hour of sleep (for details, see Supplementary Materials). Two datasets were additionally excluded for all whole night analyses due to missing data in PSG recordings at the end of the sleep period.

In terms of manipulation checks, we first tested whether both CSs + induced comparable fear responses at the end of acquisition training. Moreover, we tested whether acquisition and extinction were successful. For analyses of the

retention and renewal test, differential fear indices for CS +$_E$ and CS +$_U$ were calculated by subtracting CS- from CS + responses from sequentially corresponding trials (see Pace-Schott et al., 2015). Afterwards, differential scores were averaged across trials.

Skin conductance responses (SCRs) were quantified as peak amplitudes by means of (non-model-based) trough-to-peak scoring (further details on SCR quantification are provided in the Supplementary Materials). SCR outlier detection (Z >= ±3) was based on raw magnitudes within-subjects. Statistical outliers were winsorized to the lowest/highest score within Z =< ±3 of individual scores. Afterwards, SCRs were square-root transformed. For all other measures, outlier detection was performed across subjects for each dependent variable and univariate outliers were removed from analyses. Any deviations of results due to outlier exclusion are reported in corresponding footnotes.

Statistical analyses were conducted in *R* (R Core Team, 2020). Data of both subsamples were analyzed using linear mixed-effects models (LMMs) with individuals (level 1) being nested in subsamples (level 2). For repeated measures, measurements (level 1) were nested in individuals (level 2) which were nested in subsamples (level 3). All LMMs included (nested) random intercepts. (Nested) random slopes were included whenever they significantly improved model fit. In addition to LMM analyses with subsample as level, the main analyses were repeated including subsample and its interactions as fixed effects (results provided in the Supplementary Materials). In all linear models, fixed effects were centered within subsamples. Dichotomous predictors were dummy-coded (e.g., Group: control group [-0.5], hypnosis group [0.5]). LMM analyses were performed using the packages *nlme* (Pinheiro et al., 2022) and *reghelper* (Hughes, 2021). Robust linear regression analyses were conducted with *robustbase* (Maechler et al., 2022). Plots were built with *ggplot2* (Wickham, 2016). All other hypotheses were tested using X^2-tests or Welch's *t*-tests. Effects were considered significant at *p* < .05. Degrees of freedom varied due to missing data. Model parameters and coefficient tables are provided in the Supplementary Material.

Results

Effect of Sleep-Directed Hypnosis on Sleep Characteristics

Subjective Sleep Quality

Manipulation check: To investigate whether presentation of the aversive film clip affected sleep quality, we investigated changes in sleep quality from Day 1 to Day 2 across both subsamples. Both groups showed a decline in sleep quality from Day 1 to Day 2. That is, separate LMM analysis in each group including the fixed effect Day (1, 2) revealed significant negative slopes: hypnosis group: *b* = -0.21, 95% CI [-0.38, -0.05], *se* = 0.08, *t*(90) = -2.60, *p* = .011; control group: *b* = -0.18, 95% CI [-0.34, -0.02], *se* = 0.08, *t*(87) = -2.20, *p* = .031.

Intervention effects: To examine the effect of sleep-directed hypnosis on sleep quality, we ran a LMM analysis including the fixed effects Day (2, 3) and Group (hypnosis, control) across both subsamples. Sleep Quality on Day 1 was included as covariate to control for potential baseline effects. The analysis revealed a Day*Group interaction effect: *b* = 0.52, 95% CI [0.26, 0.79], *se* = 0.14, *t*(172) = 3.86, *p* < .001. On Day 2, sleep quality did not differ by Group (*p* = .160). From Day 2 to Day 3, sleep quality changed inversely depending on the experimental group: While the hypnosis group showed an increase in sleep quality, *t*(172) = 2.42, *p* = .017, sleep quality in the control group declined, *t*(172) = -3.04, *p* = .003. Sleep quality on Day 3 differed significantly between groups with higher scores in the hypnosis group compared to the control group, *t*(177) = 3.47, *p* < .001 (see Fig. 2). These findings are in line with the hypothesis that sleep-directed hypnosis influenced subjective sleep quality. The LMM analysis further revealed a positive relationship between Sleep Quality at Day 1 and sleep quality at later time points, *b* = 0.38, 95% CI [0.27, 0.49], *se* = 0.06, *t*(177) = 6.79, *p* < .001.

Sleep Architecture and Slow Wave Activity

Sleep characteristics and group comparisons in the laboratory subsample are presented in Table 2. Analyses of N3 duration (minutes and % of TST) during the whole night and during the first hour of sleep revealed no significant differences between groups (*p*'s >= .931). Furthermore, group comparisons on relative SWA power during N2 and N3 did not reveal any significant differences between the hypnosis and control condition across the whole night and the first hour of sleep (*p*'s >= .593). Exploratory analyses of other sleep stage amounts across the whole night revealed significantly lower REM sleep duration (minutes and % of TST) in the hypnosis group compared to the control group (*p*'s <= .034). Further exploratory analyses on objective indices of sleep quality, i.e., sleep efficiency, SOL and WASO, or on N3 and REM sleep latency did not reveal any group effects (*p*'s >= .225). These findings do not support our hypothesis of increased amounts of N3 and SWA as a result of sleep-directed hypnosis.

To explore whether sleep physiology could explain the change in subjective sleep quality from Day 2 to Day 3 in the laboratory subsample, linear regression models with Group

Fig. 2 Change in sleep quality across experimental days._Note._ Means and standard errors of sleep quality change (Day [x] – Day 1). From Day 1 to Day 2, both groups showed decreased sleep quality. From Day 2 to Day 3, experimental groups showed inverse changes: sleep quality significantly improved in the hypnosis group to baseline (Day 1) level (turquoise asterisk), whereas sleep quality in the control group decreased further (yellow asterisk), leading to significantly different sleep quality scores at Day 3 by Group (black asterisk). Note that results are based on a linear-mixed effects model (LMM) analysis including Day 1 as covariate. The graph does not represent all components of the LMM that was built by the data; plot is shown for illustration.
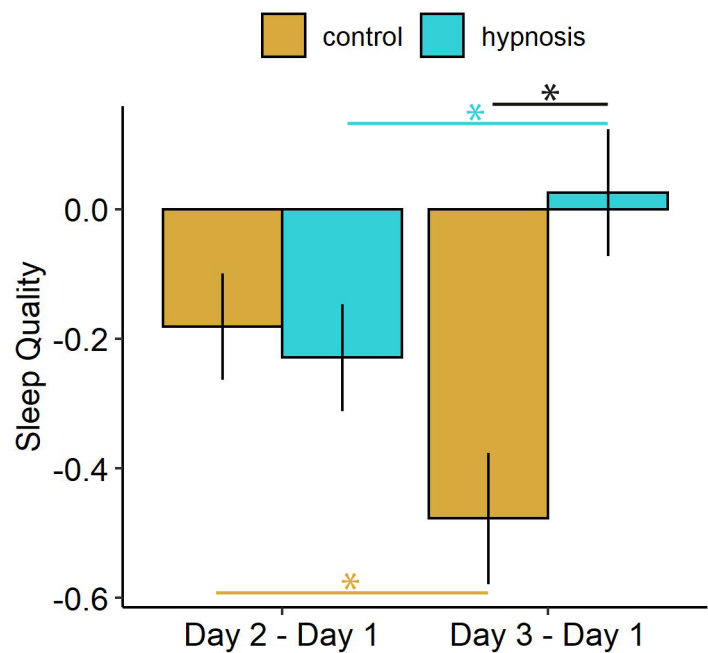

Table 2 _Sleep parameters of the whole night and first hour of sleep (laboratory subsample)_

Measure	Sleep Stage	Hypnosis Group		Control Group		Test Statistics		
		M	SD	M	SD	t_W	df	p
Whole night sleep								
Duration	N1	39.98	14.57	35.60	14.94	-1.25	68.80	.215
(Min)[a]	N2	205.14	38.18	207.53	30.81	0.29	68.34	.770
	N3	101.18	32.47	100.97	27.78	-0.03	67.92	.977
	REM	99.10	21.90	109.08	16.49	2.17	64.98	.034
	TST	456.25	23.57	453.18	26.80	-0.51	66.91	.613
Duration (% TST)[a]	N1	9.03	3.82	7.86	3.31	-1.38	68.11	.172
	N2	45.87	7.37	45.73	5.57	-0.09	66.83	.932
	N3	22.48	6.41	22.35	6.23	-0.09	69.00	.931
	REM	21.83	4.19	24.06	3.27	2.50	65.88	.015
Latency (Min)	N3	14.77	7.27	14.65	5.96	-0.08	69.05	.935
	REM	104.95	47.66	92.71	37.11	-1.23	67.78	.225
Sleep efficiency (%)	/	94.11	4.55	93.00	4.27	-1.03	66.00	.305
WASO (Min)	/	9.40	10.40	12.65	14.22	1.08	62.30	.282
SOL (Min)	/	22.61	16.16	18.63	13.24	-1.12	65.59	.268
SWA power (% TP)	N2 + N3	86.67	4.19	86.58	3.54	-0.11	70.00	.915
First hour of sleep								
Duration (Min)	N3	35.06	11.64	35.41	8.73	0.14	66.72	.887
Duration (% TST)	N3	59.15	18.61	59.60	14.31	0.12	67.43	.907
SWA power (% TP)	N2 + N3	89.06	5.20	88.48	3.98	-0.54	67.26	.593

Note. N1–3 = non-rapid eye movement sleep stage 1–3; REM = rapid eye movement sleep; TST = total sleep time; WASO = wake after sleep onset; SOL = Sleep onset latency; SWA = slow wave activity; TP = total power. [a] Note that outlier exclusion was performed for each dependent variable. Therefore, descriptive statistics of sleep stage amounts does not correspond to TST amounts

as predictor and PSG-based sleep variables as additional predictors were examined. Goodness-of-fit tests did not reveal a significant increase in model fit by including any of the sleep variables. Further explorations on model parameters, however, indicated multivariate outliers and influential

data points. Therefore, robust linear regression models were computed in addition. Goodness-of-fit tests on robust regression models indicated that the amount of time spent in N3, $F(2,65) = 16.24$, $p < .001$, and WASO, $F(2,63) = 7.53$, $p = .023$, significantly improved estimation of the change in
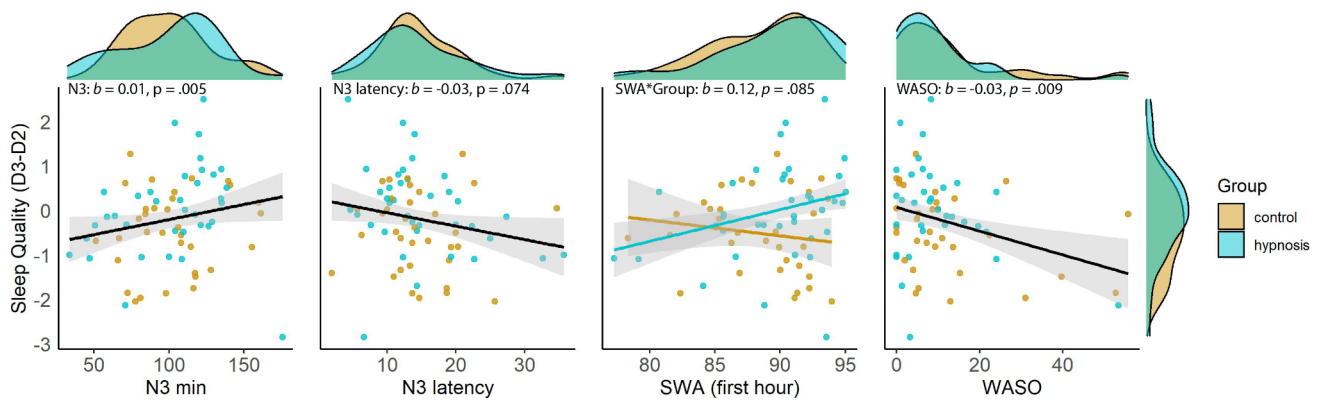
Fig. 3 Relationship between subjective sleep quality and objective sleep parameters of the sleep period in Night 2 in the laboratory subsample. *Note.* Density plots showing the distribution of sleep variables in the control and hypnosis group. Scatter plots illustrating the linear relationships between the change in sleep quality from Day 2 to Day 3 (difference scores) and PSG-based sleep variables across/within the experimental groups. Note that the scatterplots present raw data, whereas the linear slopes were build on robust regression modeling. N3 = non-rapid eye movement sleep stage 3, SWA = slow wave activity, WASO = wake after sleep onset

sleep quality from Day 2 to Day 3 (see Fig. 3).[2] Goodness-of-fit tests further indicated that N3 latency and relative SWA power during the first hour of sleep improved model fit (p's = < .007). The regression outputs, however, did not show any significant effects of sleep variables (p's > = .074). An exploratory robust regression analysis including Group and N3 duration revealed a main effect of Group, $b = 0.54$, 9% CI [0.10, 0.98], $se = 0.22$, $t(65) = 2.45$, $p = .017$, and a main effect of N3, $b = 0.01$, 9% CI [0.003, 0.01], $se = 0.003$, $t(65) = 2.90$, $p = .005$. No interaction effect was found ($p = .128$). Improvement in sleep quality in the laboratory subsample was associated with hypnosis compared to the control condition and with higher amounts of N3. Another exploratory robust regression analysis including Group and WASO revealed a main effect of WASO, $b = -0.03$, 9% CI [-0.05, -0.01], $se = 0.01$, $t(63) = -2.70$, $p = .009$. The Group effect did not reach significance ($p = .051$) and no interaction effect was found ($p = .494$). Less time spent awake during the sleep period was associated with an improvement in subjective sleep quality from Day 2 to Day 3 in the laboratory subsample.

## Effects of Sleep-Directed Hypnosis on Fear Extinction Recall and Film-Related Intrusions and Rumination

Fear Conditioning Phases

Manipulation check - Acquisition: Analyses of CSs + responses during acquisition training revealed no differences between CS + $_E$ and CS + $_U$ on the final trial of acquisition training for US expectancy across both subsamples ($p = .667$) and SCR in the laboratory subsample ($p = .804$). Averaged CSs+ (CS + $_E$, CS + $_U$) and CS- (across blocks) responses were therefore calculated for each trial in the following analysis. LMM analyses of US expectancy including Trial, CS type (CS+, CS-) and Group across both subsamples showed differential elevation of fear expressions for CS + but not for CS- during acquisition training, indicated by a Trial*CS type interaction effect, $b = 7.16$, 95% CI [6.66, 7.66], $se = 0.26$, $t(2709) = 27.97$, $p < .001$. SCR analyses in the laboratory subsample revealed higher SCRs for the CS + compared to CS-, main effect of CS type: $b = 0.09$, 95% CI [0.06, 0.13], $se = 0.02$, $t(1070) = 5.63$, $p < .001$. No Group effects were found (p's > = 0.252).

Manipulation check - Extinction: LMM analyses of US expectancy including Trial, CS type (CS + $_E$, CS-) and Group across both subsamples indicated successful fear extinction, reflected in a significant Trial*CS type interaction effect, $b = -3.14$, 95% CI [-3.54, -2.74], $se = 0.20$, $t(2686) = -15.44$, $p < .001$. Successful extinction was also evident in SCR responses in the laboratory subsample, as reflected in a main effect of CS type, $b = 0.03$, 95% CI [0.002, 0.06], $se = 0.02$, $t(1067) = 2.11$, $p = .035$, and post-hoc tests, showing that SCRs for CS + $_E$ were higher than for CS- at the first trial of extinction training, $t(1067) = 2.30$, $p = .022$, whereas no

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[2] When outliers are included, N3 no longer significantly predicted the change in sleep quality ($p = .530$). A regression analysis including Group and N3 latency revealed significant main effects of Group ($p = .015$) and N3 latency ($p = .040$) when including outliers. Another regression analysis including outliers with Group and relative SWA power during the first hour of sleep as predictors revealed a significant Group*SWA interaction effect ($p = .005$). Finally, a regression analysis including Group and WASO revealed significant main effects of Group ($p = .020$) and WASO ($p = .034$) when including outliers.
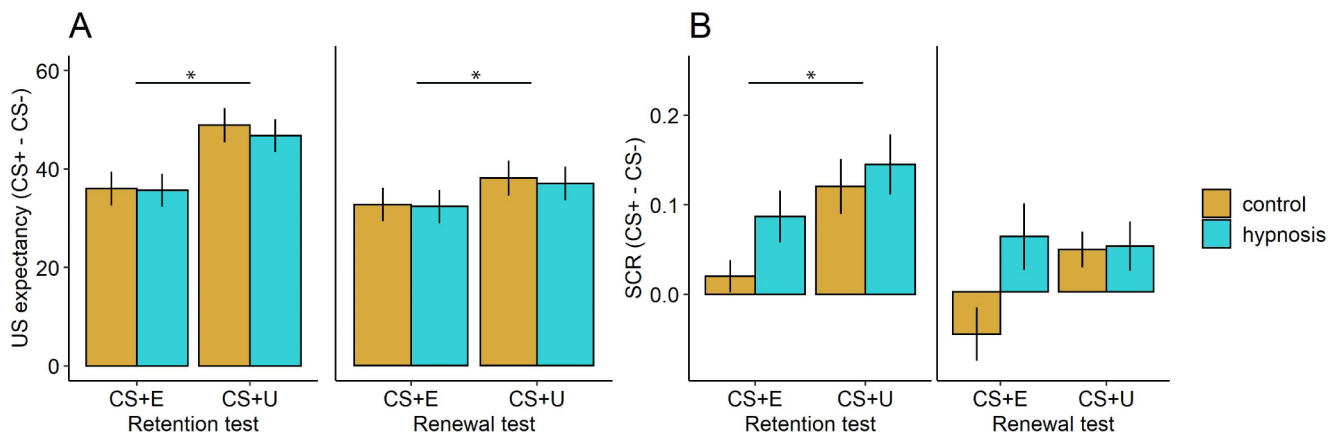
Fig. 4 Fear expressions during retention test and renewal test. *Note.* Means and standard errors of differential US expectancy scores (**4A**) and skin conductance responses (SCR in microSiemens, square-root transformed; **4B**) in the laboratory subsample averaged across the respective test phase. Asterisks indicate significant main effects of CS + type based on linear mixed-effects model (LMM) analyses (*$p < .05$). No effects of Group were found in any analysis. Note that means and standard errors do not represent all components of the LMMs that were built by the data; plots are shown for illustration.

difference was found at the final extinction trial ($p = .689$). No Group effects were found (p's > = .288).

Retention test LMM analyses of US expectancy averaged across retention test trials including CS + type (difference scores for $CS +_E$ and $CS +_U$) and Group across both subsamples revealed higher US expectancy for $CS +_U$ compared to $CS +_E$, $b = -11.98$, 95% CI [-15.86, -8.11], $se = 1.97$, $t(178) = -6.07$, $p < .001$. This pattern confirms the intended manipulation, i.e., differential extinction of the $CS +_E$. No effects of Group were found ($p > = 0.656$). For SCR, a LMM analysis including CS + type and Group in the laboratory subsample similarly showed higher SCRs for $CS +_U$ compared to $CS +_E$, $b = -0.08$, 95% CI [-0.11, -0.04], $se = 0.02$, $t(67) = -4.28$, $p < .001$. No effects of Group were found ($p > = 0.233$). These findings suggest no effect of sleep manipulation on extinction recall. Moreover, no significant differences between groups were found when comparing differential $CS +_E$ and $CS +_U$ responses, which does not support the hypothesis of extinction generalization promoted by sleep-directed hypnosis.

We further explored whether interindividual differences in SWS amount (minutes) could have influenced extinction recall during retention test in the laboratory subsample. Goodness-of-fit tests did not reveal significant improvements in model fit by introducing SWS amount into the LMMs including US expectancy or SCRs as dependent variable (p's > .05).

Renewal test LMM analyses of averaged differential US expectancy across the renewal test including CS + type and Group across both subsamples revealed a significant effect of CS + type with higher US expectancy for $CS +_U$ compared to $CS +_E$, $b = -4.75$, 95% Ci [-8.22, -1.27], $se = 1.77$, $t(177) = -2.68$, $p = .008$. No effects of Group were found

(p's > = .878). SCR analyses including CS + type and Group revealed no significant effects in the laboratory subsample (p's > = .061; see Fig. 4 for an overview of retention and renewal test performance). These results similarly do not support any impact of sleep-directed hypnosis on extinction recall. Further exploratory tests on whether interindividual differences in SWS amount (minutes) could have influenced performance during renewal test in the laboratory subsample were carried out. Goodness-of-fit tests did not indicate significant improvements in model fit by introducing SWS amount into the LMMs including US expectancy or SCRs as outcomes (p's > .05).

Intrusions and Rumination

To examine the effect of sleep-directed hypnosis on film-related intrusion and rumination, LMM analyses including Group and Day (Day 2, Day 3) were performed across both subsamples. For both measures, the analyses revealed a significant decline across days (main effect), intrusion index: $b = -0.81$, 95% CI [-1.15, -0.48], $se = 0.17$, $t(170) = -4.80$, $p < .001$, rumination index: $b = -1.24$, 95% CI [-1.90, -0.58], $se = 0.34$, $t(168) = -3.69$, $p < .001$. No significant effects of Group were found for either measure (p's > = .199). Finally, we examined whether Group effects were evident in intrusions during the IPT on Day 3 across both subsamples. LMM analyses including Group revealed no main effect of Group ($p = .577$). In contrast to our hypothesis, these findings do not support the assumption that sleep-directed hypnosis has a beneficial impact on intrusions or rumination.

Discussion

The current study aimed to investigate potential effects of sleep-directed hypnosis on extinction of trauma-associated fear memories. Analyses revealed a beneficial impact of hypnosis on subjective sleep quality in both subsamples. Although subjective sleep quality was significantly correlated with the amount of N3 sleep and WASO in the laboratory subsample, we found no direct effects of hypnosis on SWS or SWA. Surprisingly, exploratory findings indicate that hypnosis decreased the amount of REM sleep. Neither analyses of extinction retention and generalization nor of trauma-associated intrusions and rumination revealed any effects of hypnosis. As such, the current results indicate that sleep-directed hypnosis is useful for improving subjective sleep quality but not for enhancing extinction memory and reducing analog PTSD symptoms.

Our finding that sleep-directed hypnosis improves subjective sleep quality aligns with previous findings from Cordi et al. (2020) and extends these in important ways: First of all, we were able to show that this enhancing effect is also evident when sleep-directed hypnosis and subsequent sleep take place under ecologically valid conditions, i.e., in the participants' home. Secondly, we were able to demonstrate convergent effects in and across two subsamples with high statistical power. Finally, we found this effect in the context of exposure to a traumatic film clip that may have affected sleep quality beforehand. That is, both groups showed a decline in sleep quality following acquisition training, suggesting a detrimental impact of the traumatic film clip on sleep (see also Richardson et al., 2015; Sopp et al., 2019; Talamini et al., 2013). This effect dissipated after the hypnosis intervention but not after control condition, indicating that sleep-directed hypnosis may have counteracted the effect of exposure to the aversive material on sleep quality. These findings suggest that the effects of sleep-directed hypnosis may also emerge in the context of trauma and other distressing life events, forming an important basis for the application of sleep-directed hypnosis in clinical settings.

In contrast to our assumptions, we did not find any significant impact of hypnosis on SWS parameters. Groups only differed in REM sleep duration, which may be related to numerically higher REM sleep latency in the hypnosis group. This effect may have been driven by increased REM sleep amounts due to the presentation of aversive film clips (Delannoy et al., 2015). This increase may have been normalized by subsequent hypnosis, hence only emerging in the control group. However, since our design did not comprise an assessment of sleep physiology during the first experimental night after acquisition training, this hypothesis requires further testing in subsequent studies.

In line with earlier findings showing that SWS and SWA were positively associated with subjective sleep quality (Cordi et al., 2014; 2015), exploratory analyses pointed towards a role of SWS in determining the change in sleep quality from pre- to post-intervention. However, it is important to note that only the amount of SWS and not other SWS parameters predicted sleep quality and that this effect did not interact with hypnosis. Inconsistencies between the current results and the findings from Cordi et al. may be related to the fact that we assessed sleep physiology under potentially noisy (ambulatory) conditions in a subsample of participants. Moreover, previous research used within-subjects designs, whereas we used a between-subjects design to accommodate the fear conditioning procedure. Nevertheless, the fact that we did not find direct evidence for increased SWS by hypnosis limits the interpretation of our findings with respect to potential effects on extinction memory.

Our analyses of extinction retention and generalization did not reveal any significant between-group differences, suggesting that hypnosis did not affect the consolidation of extinction memory. Consonantly, we did not observe any group differences in intrusive memories or ruminative thoughts. Hence, our study suggests that while hypnosis enhanced subjective sleep quality, it did not facilitate sleep-related memory reprocessing. Several explanations may account for these results: First, the lack of significant effects of hypnosis on SWS features may have prevented finding a beneficial effect of hypnosis via SWS-induced strengthening of extinction memory. Secondly, sleep-directed hypnosis may not be suited to target memory processes during SWS. Correspondingly, the hypnotic suggestion used in the current study has recently been shown to increase SWS parameters but decrease slow wave-spindle coupling (Beck et al., 2021), which is assumed to underlie memory consolidation during SWS. Finally, another explanation could be that extinction memory is consolidated via a process, which is functionally independent of SWS. This is also supported by additional exploratory analyses on retention and renewal test performance in the laboratory subsample, which did not indicate that SWS amount significantly contributed to the prediction of extinction retrieval. Indeed, previous studies have suggested an involvement of REM sleep rather than SWS in the consolidation of extinction memory (e.g., Menz et al., 2016; Spoormaker et al., 2012). However, this assumption is challenged by our findings that REM sleep amounts varied between groups without having any impact on extinction recall. Moreover, a recent meta-analysis did not show a robust relationship between REM sleep and subsequent extinction recall (Schenker et al., 2021). Nevertheless, future studies should explore the impact of sleep interventions targeting REM sleep on extinction retention

and generalization as well as the additive benefits of different interventions targeting subjective and objective sleep quality.

Beyond the limitations mentioned above, several others should be considered. First of all, it is important to note that our design lacked a wake control group. Hence, it is not possible to draw any conclusions regarding the effects of sleep per se but only regarding the effects of sleep-directed hypnosis. Second, previous studies have shown that effects of sleep-directed hypnosis rely on the hypnotizability of participants (Cordi et al., 2014; 2015; 2020). However, additional analyses in the remote subsample, which comprised individuals with different levels of hypnotizability, did not reveal any significant effects of hypnotizability. This contrasts previous reports and could be related to procedural differences between studies (e.g., remote assessment of hypnotizability). Relatedly, it is important to note that the majority of participants of the remote subsample was strongly hypnotizable, which may have prevented us from finding effects of hypnotizability. Third, differences between remote and laboratory assessment may have brought about additional variance in the current analyses. We based this approach on previous research (Kleim et al., 2016) in an effort to further enhance statistical power. Moreover, we used LMM analyses to account for dissimilarities between subsamples. In addition, we tested whether our results differed between the two subsamples. These analyses revealed mostly consistent results between the remote and laboratory subsample (see Supplementary Materials). Fourth, in order to quantify SCRs, we used a non-model-based approach (i.e., trough-to-peak scoring). There has been significant debate on the appropriateness of such approaches (see e.g., Bach & Melinscak, 2020). However, recent research confirms that through-to-peak scoring produces satisfactory effects in terms of CS discrimination (Kuhn et al., 2022). Hence, we believe that, while more research on the issue of SCR quantification is needed, our approach can be considered sufficient for the scope of our research questions. Finally, it is important to note that we used an analog procedure to investigate processes during TF-PT. Although this approach aligns with previous research aiming to shed light on the underlying processes of PTSD symptom development and treatment (Ney et al., 2022), further research is needed to establish whether results replicate in clinical settings. Relatedly, pre-experimental differences in psychological vulnerability may have influenced memory processing during the experimental tasks as well as the impact of hypnosis. Though participants in our two subsamples did not report a history of traumatic events or current mental disorders, we cannot rule out possible effects of (sub)clinical characteristics. To keep assessment as brief as possible, we did not assess such characteristics except for trait anxiety.

Additional exploratory analyses including trait anxiety as covariate did not change the direction of our results nor did trait anxiety interact with Group in any of our analyses.

Despite these limitations, our study provides important insights: Although sleep-directed hypnosis did not affect extinction memory and objective sleep quality, it robustly improved subjective sleep quality, which was significantly reduced after fear conditioning including exposure to a traumatic film clip. These findings indicate that sleep-directed hypnosis could be beneficial for improving subjective sleep quality in traumatized individuals with PTSD symptoms. This is noteworthy since the majority of PTSD patients (i.e., 70–91%) experience difficulties falling and staying asleep (Maher et al., 2006) and sleep problems are a particularly common residual symptom after treatment (Schnurr & Lunney, 2019). Moreover, trauma-induced insomnia severely affects quality of life and overall well-being (Werner et al., 2021). For instance, recent findings indicate that subjective sleep quality moderates the association between trauma exposure and suicide attempts (King et al., 2021). At the same time, standard approaches for treating sleep disturbances are not effective in all PTSD patients (see e.g., full remission rate of 41% in Talbot et al., 2014). Sleep-directed hypnosis might thus be a useful addition to treat sleep disturbances in PTSD (Arditte Hall et al., 2021; Galovski et al., 2016). Moreover, due to easy, self-guided implementation, sleep-directed hypnosis could be used as an intervention strategy for trauma survivors, experiencing sleep disturbances in the early aftermath of trauma.

Declarations

The study procedures were approved by the local ethics committee of Saarland University (A15-3). The study protocol was registered at the German Clinical Trials Register (DRKS00022369) prior assessment of the laboratory subsample. All participants provided written informed consent in accordance with the Declaration of Helsinki. Data underlying our analyses are accessible at OSF: https://doi.org/10.17605/OSF.IO/X4T25.

CRediT author statement E.F.: Conceptualization, Methodology, In-

vestigation, Writing – original draft, Formal analysis, Visualization, Project administration. **M.R.S.**: Conceptualization, Methodology, Investigation, Writing – original draft, Formal analysis, Visualization, Project administration, Funding acquisition. **M.J.C.**: Conceptualization, Methodology, Resources, Writing - review & editing. **B.R.**: Conceptualization, Methodology, Resources, Writing - review & editing. **T.M.**: Conceptualization, Methodology, Resources, Writing – review & editing, Funding acquisition, Supervision.

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