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Reactivating a Relaxation Exercise During Sleep to Influence Cortical Hyperarousal in Individuals With Frequent Nightmares

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ABSTRACT

High-frequency EEG activity during sleep, reflecting cortical hyperarousal, has recently been incorporated into aetiology models of nightmares. However, its precise role remains insufficiently understood because experimental approaches to manipulate hyperarousal during sleep are limited. In the present study, we investigated whether re-presenting a relaxation-associated odour cue during sleep to reactivate a previously practiced deep-breathing relaxation exercise reduces cortical hyperarousal as indexed by beta (16.25–31 Hz) and gamma (31.25–45 Hz) activity and, consequently, nightmare symptoms. Twenty-five participants (21 female, mean age (SD) = 23.12 (3.70) years) experiencing at least one nightmare per week completed a 1-week deep breathing relaxation training paired with an odour cue. In a randomised, blinded, crossover design, either the relaxation-associated odour or a control odour was presented across two laboratory nights. Odour-based reactivation did not influence beta or gamma power but was unexpectedly associated with a significantly reduced spindle count and density. Spindle count during reactivation nights correlated positively with subjective wake-after-sleep-onset. Nightmare symptoms were unaffected. These findings suggest that odour-cued reactivation of a relaxation exercise did not modulate cortical hyperarousal or nightmare symptoms, despite altering spindle activity. The functional significance of this spindle effect remains to be determined. Future studies should employ longer reactivation periods and alternative interventions targeting cortical hyperarousal to further clarify its causal role in nightmare aetiology and the therapeutic potential of sleep-based interventions.

1 | Introduction

Recent studies have indicated an association between cortical hyperarousal and frequent nightmares (Blaskovich et al. 2020; Sayk et al. 2024). Cortical hyperarousal can be defined as increased high-frequency or ‘wake-like’ electroencephalographic (EEG) activity during sleep, especially in the beta and gamma bands (Blaskovich et al. 2020). Apart from beta and gamma

activity, several other forms of hyperarousal, including increased microarousals before REM sleep (Blaskovich et al. 2020), altered autonomic arousal (Paul et al. 2019), or cyclic alternating patterns (Simor et al. 2013), have been reported in individuals with frequent nightmares or nightmare disorder. Some studies suggest that hyperarousal occurs primarily during nightmare episodes (Paul et al. 2019) or in pre-REM periods (Blaskovich et al. 2020), whereas others indicate a more persistent elevation

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of beta and gamma activity in individuals with frequent nightmares compared with healthy controls (Marquis et al. 2017; Sayk et al. 2024). Hyperarousal has been associated with deficits in fear extinction that are thought to stem from amygdala hyperactivation or insufficient prefrontal inhibition (Nielsen 2017). Consequently, cortical hyperarousal is implicated in several aetiology models of nightmares, including the integrative model of Giesemann et al. (2019) and the Affect Network Dysfunction Model proposed by Nielsen and Levin (2007).

Despite the prominent role attributed to hyperarousal in nightmare aetiology, the available evidence remains largely correlational (Blaskovich et al. 2020), leaving it unclear whether hyperarousal is a consequence of fragmented REM sleep, a mere epiphenomenon, or a causal factor. This question has gained further relevance from recent intervention studies. For example, we recently found that successful imagery rehearsal therapy (IRT), the first-line treatment for nightmares (Thünker and Pietrowsky 2012), was accompanied by reduced gamma activity alongside marked reductions in nightmare symptoms (Sayk et al. 2024). The present study therefore sought to establish an experimental approach for modulating cortical hyperarousal during sleep. Such an approach would provide an important step towards clarifying its causal role in nightmare aetiology and evaluating its potential as a therapeutic target.

Relaxation-based approaches such as deep breathing have been shown to reduce high-frequency EEG activity both during the actual practice (Jacobs and Friedman 2004) as well as beyond the immediate practice period (Cheng et al. 2018), making them a promising approach for experimentally targeting cortical hyperarousal. However, testing the causal role of cortical hyperarousal requires transferring these effects from wakefulness into sleep. One possible approach is targeted memory reactivation (TMR), a technique that reactivates specific memory content during sleep by presenting an associated cue, most commonly an auditory or olfactory stimulus (Oudiette and Paller 2013; Born and Wilhelm 2012). When an odour is paired with newly acquired learning material, it becomes associated with that material, enabling its reactivation when the odour is re-presented during subsequent sleep. Although TMR has primarily been used to strengthen semantic and procedural memories, recent evidence suggests a broader potential. Carbone and Diekelmann (2024) emphasised that TMR can be applied to reactivate psychotherapeutic interventions (Borghese et al. 2022; Schwartz et al. 2022), modify dream content (Salvesen et al. 2024), and alter spectral activity by engaging relaxation-related concepts (Beck et al. 2021).

In sum, cortical hyperarousal appears to be an important factor in nightmare aetiology. To clarify its causal role, experimental manipulation is required. Deep-breathing relaxation exercises have been shown to reduce high-frequency EEG activity, and TMR may constitute a viable approach for transferring these effects into the sleep period. The primary aim of this study was to determine whether reactivating a previously learned relaxation exercise during sleep via olfactory cues reduces cortical hyperarousal in individuals with frequent nightmares. We hypothesised that beta and gamma activity would be lower during presentation of the relaxation-associated odour. Furthermore, we hypothesised that successful modulation of

cortical hyperarousal would be accompanied by reductions in nightmare frequency, nightmare distress, and impaired sleep quality.

2 | Methods and Materials

2.1 | Participants

Participants were mainly recruited from the student body at University of Luebeck, Germany through mailing lists and posts on campus. Participants were between 18 and 35 years of age, experienced at least one nightmare per week, and reported no psychiatric disorder (including lifetime PTSD), somatic illness, or other sleep disorder. Participants were also not taking medication known to affect sleep. Eligibility criteria were determined in a telephone interview, with special emphasis on ensuring that nightmare symptoms were present according to this definition given to the potential participants: 'Dreams with very negative emotional experiences which are usually so upsetting/frightening that you wake up from them'. No structured clinical interviews were conducted to establish formal diagnoses, such as nightmare disorder. The study was approved by the university's ethics committee. All participants provided written informed consent after they were given a complete description of the study protocol. Participants were compensated financially (four participants received part of the compensation in the form of course credits) for study participation.

The initial sample consisted of 29 participants. Four participants dropped out of the study after adaptation night due to inability to sleep in the sleep laboratory. The majority of the final sample ($N=25$) was female (84%), which is to be expected due to nightmares being more frequent in women (Schredl 2010), mean age was 23.12 ($SD=3.70$) years (Table 1). On a 5-point Likert scale, 20 participants rated their degree of suffering from nightmares as 'moderately' and 5 participants reported a 'strong' degree of suffering. Six participants reported recurrent nightmares with identical or nearly identical content, and nine participants reported nightmares related to autobiographical experiences. Participants on average had slight sleep problems according to the Pittsburgh Sleep Quality Index (PSQI; Table 1) but average sleep reactivity according to the Ford Insomnia Response to Stress Test (FIRST; Table 1). Since suggestibility and absorption have been implicated in previous research on relaxation (Cordi and Rasch 2021), we assessed all participants' suggestibility (tendency to react to hypnotic suggestions) on the Harvard Group Scale of Hypnotic Susceptibility 5-Item Short-Version (HGSHS-5:G; Table 1) and their absorption levels (imaginative involvement and tendency to become mentally absorbed) on the Tellegen Absorption Scale (TAS) (see also Table 1). Suggestibility levels in our sample can be rated as medium (Riegel et al. 2021) whereas levels of absorption were comparatively low (Angiulo and Kihlstrom 1993). Sample size was determined using a medium to large effect size (cf. Cheng et al. 2018 for relaxation exercise; Cordi and Rasch 2021 and Beck et al. 2021 for the effects of (reactivating) relaxation on sleep), $1-\beta=0.85$, $\alpha=0.05$ and correlation among repeated measures=0.5, which resulted in $N=21$ to which we added a 20% allowance for dropouts, resulting in a desired final sample size of $N=25$.

TABLE 1 | Demographic and psychometric characteristics of final sample at baseline.

	M (SD)
Demographics	
Age (years)	23.12 (3.7)
Sex (% female)	84%
Sleep quality and reactivity	
PSQI ^a	6.12 (1.67)
FIRST ^b	2.6 (0.5)
Suggestibility	
TAS ^c	2.92 (0.57)
HGHS:5G ^d	2.17 (1.66)
Nightmare characteristics	
Frequency (per month)	8.48 (3.72)
Frequency (per week)	2.00 (0.76)
Nightmare onset ≥ 10 years (% of sample)	60%
Memory of nightmares: very often/always (% of sample)	78%
Identical repetition (% yes)	20%
Degree of suffering ^e	3.00 (0.58)

^aPSQI = Pittsburgh Sleep Quality Index.^bFIRST = Ford Insomnia Response to Stress Test.^cTAS = Tellegen Absorption Scale.^dHGHS:5G = the Harvard Group Scale of Hypnotic Susceptibility 5-Item Short-Version.^eDegree of Suffering measured with the question 'How much do you suffer from your nightmares or the sleep disturbance caused by nightmares?' on a 5-point Likert scale from 'not at all' to 'very strongly'.

2.2 | Procedure and Design

If all inclusion criteria were met in the telephone interview, three nights in the sleep laboratory were scheduled. The first set of questionnaires was completed via an online link before the adaptation night. Upon arrival for the adaptation night, participants rated four odours in terms of their pleasantness, familiarity, and intensity after having been presented with each odour directly from the container for 10 s each from a distance of 20 cm. The odour labels were concealed from the participants. The two odours rated as least familiar while being highly pleasant and intense were selected as the relaxation-associated (reactivation) odour and the control odour, respectively, to maximise compliance and ensure sufficient olfactory perceptibility. Afterwards, they were fitted with EEG, EOG, EMG, ECG, and SCR electrodes before completing the relaxation exercise and listening to a control text, without any odour being present. The audio recordings were presented on a computer using PsychoPy 3.1 (Peirce et al. 2022) and this procedure was repeated on the evening of the first experimental night. Levels of relaxation as well as positive and negative affect were measured at baseline before the audio recordings and after each audio recording. Level of relaxation was measured using a 5-point Likert scale, while positive and negative affect was evaluated using a short version of the Positive and

Negative Affect Schedule (PANAS). Relaxation exercise and control text were presented in randomised order across participants and nights (adaptation night and first experimental night). Participants then went to bed at their usual bedtime and an adaptation odour was presented with a diffuser for the whole night. In the morning after the adaptation night, participants downloaded the audio recording of the relaxation exercise on their phones and received a diffuser filled with their individual relaxation-associated odour. They were instructed to practice the relaxation exercise daily for the next 7 days, activating the diffuser each time.

At the end of the week of practice, participants completed another set of online questionnaires and returned to the sleep laboratory for the first of two experimental nights. On the evening of the first experimental night, they were exposed to the relaxation exercise and a control text again. Adherence to practicing the relaxation exercise in the last week was assessed as well at this point. This time, the olfactometer (Amores 2016) was activated when the participants first reached stable slow wave sleep. The reactivation odour was presented in one night and the control odour in the other, with the order of odour presentation randomised between the first and second experimental night. Only the relaxation-associated odour was paired with the relaxation exercise during home practice. The control odour was not associated with any task and was used exclusively as a control stimulus during the experimental sleep nights. Participants were blind to the intervention. The last experimental night with odour presentation took place another 2–7 days after the first experimental night. (see Figure 1 for complete overview of the experimental procedure).

2.3 | Materials

2.3.1 | Questionnaires

Nightmare symptoms were assessed with a customised questionnaire (published in Sayk et al. 2024) at baseline and after the intervention week including nightmare frequency, time of nightmare onset, nightmare content, ability to remember nightmares, and distress caused by nightmares. Subjective sleep quality was measured with the *Schlaffragebogen A*, SF-A/R (Görtelmeyer 2011) during sleep lab nights, and retrospectively using the PSQI (Buysse et al. 1988) in order to accurately assess changes in sleep quality at home. Additionally, at baseline, sleep reactivity was assessed with the FIRST (Drake et al. 2004), and suggestibility and absorption were measured with the HGHS-5:G (Riegel et al. 2021) and TAS (Tellegen and Atkinson 1974), respectively. Directly after the relaxation exercise and control text, participants repeatedly rated their current affective state with a short version of the PANAS (Breyer and Bluemke 2016).

2.3.2 | Polysomnography

Participants were fitted with 64 electrode EEG caps (ActiChamp, Brain Products, Gilching, Germany) referred to the left mastoid (M1) and ground electrode. We used electromyography (EMG) placed on the chin, as well as electrooculography (EOG)

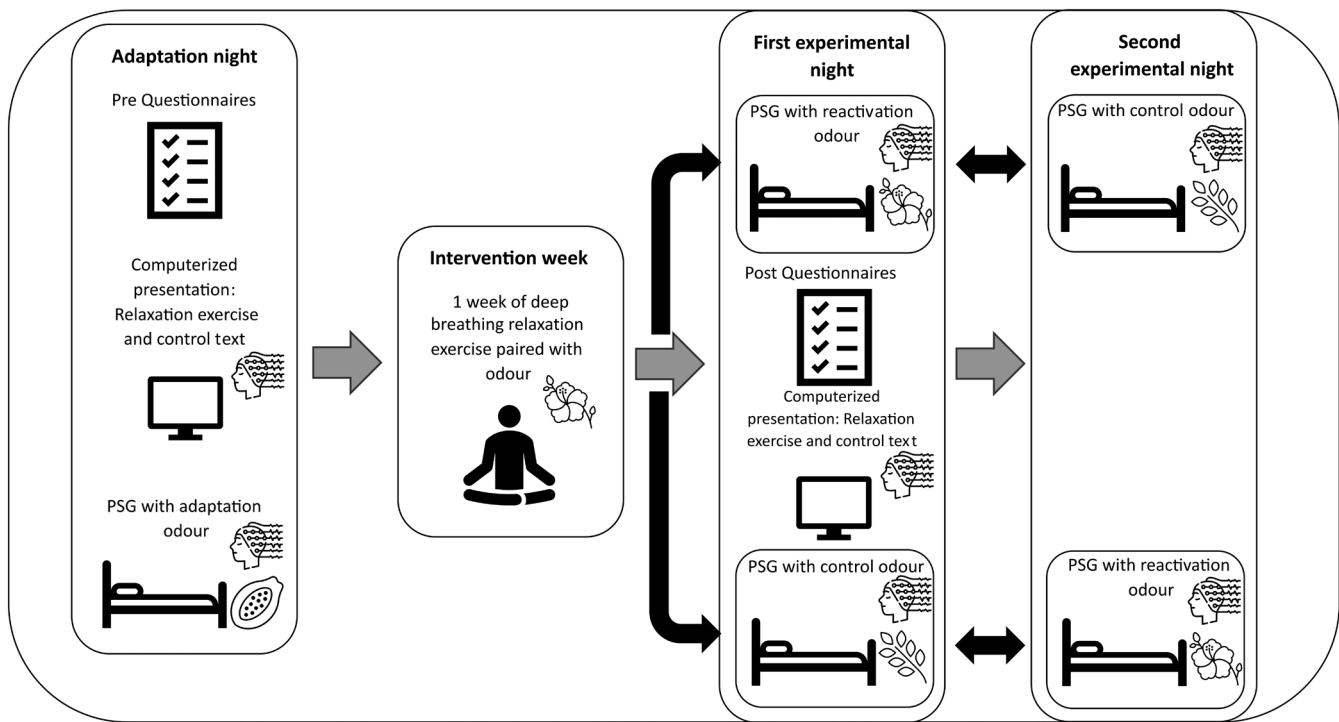


FIGURE 1 | Experimental procedure. Schematic overview of the study design and odour presentation protocol. During the adaptation night, participants completed the baseline questionnaires, listen to the relaxation exercise and a control text in randomised order while EEG was recorded and rated a set of odours to select the individual reactivation and control odour. To familiarise participants with sleeping in the presence of an odour, a separate adaptation odour (cardamom) was presented during the adaptation night. Participants then practice the deep breathing relaxation exercise at home for 1 week while the selected reactivation odour (e.g., rose geranium) was presented. During the two subsequent experimental nights either the reactivation (e.g., rose geranium) or a control odour (e.g., tea tree) were presented in randomised order (possibilities for order of presentation are indicated by black arrows). On the first experimental night, participants also completed post-intervention questionnaires and again listened to the relaxation exercise and control text.

and electrocardiography (ECG) measurement. Data were recorded with Brain Vision Recorder (Brain Products, Gilching, Germany). Impedances were below 10 k Ω . Sampling rate was 500 Hz.

2.3.3 | Sleep Scoring and Spectral Power Analysis

Sleep stages were scored manually according to AASM criteria (Berry et al. 2012) by two experienced sleep lab technicians. Recordings were visually inspected on a 30-s basis and channels with continued artefacts were discarded. Additionally, for wake EEG data, eye movement artefacts were removed with independent component analysis. EEG data was re-referenced to linked mastoids and then filtered with a 0.3 Hz high pass filter before artefact-free, 50% overlapping, 8.192-s epochs were Hanning-tapered and Fast Fourier Transformed in order to calculate absolute power of spectral densities (in μV^2) for each frequency bin between 1.25 and 45 Hz for NREM (Stage 2 and Stage 3) and REM sleep periods, separately. Spectral power was calculated for wake EEG, the whole night and for odour-on and odour-off periods. Band-wise spectral power was extracted by summing up bin-wise values into ranges of beta (16.25–31 Hz) and gamma (31.25–45 Hz) bands for sleep EEG data and alpha (8.25–13 Hz), beta (16.25–31 Hz) and gamma (31.25–45 Hz) bands for wake EEG

data gathered during the computerised presentation of the relaxation exercise and control text. Power spectra across the spectrum (1.25–45 Hz) in NREM and REM sleep can be found in Figure S1. Additionally, discrete sleep spindles (fast spindles: 12–16 Hz; slow spindles: 9–12 Hz) were identified for stages N2 and N3 of NREM sleep as described in (Ngo et al. 2020). In brief, the EEG signals were band-pass filtered between 12 and 16 Hz and the root mean square signal (RMS) was calculated based on a 200-ms window followed by an additional smoothing using a moving average with the same window length. A spindle event was identified whenever the smoothed RMS-signal exceeded a threshold, defined by the mean plus 1.2816 times the standard deviation (i.e., corresponding to the 90th percentile) of the RMS-signal across all artefact-free NREM data points, for at least 0.4 s but not longer than 3 s. The upward and downward threshold crossings represent the beginning and end of a spindle event. The spindle amplitude was determined by averaging the smoothed RMS-signal for the duration of each detected spindle event. Finally, spindle density was defined as the number of detected sleep spindles divided by the total duration of NREM sleep. For exploratory purposes, slow oscillations (0.7–1.5 Hz), delta (1.25–4 Hz), theta (4.25–8 Hz), alpha (8.25–13 Hz) and sigma (13.25–16 Hz) activity was analysed for sleep. Frequency boundaries for the spectral analyses were adopted from Blaskovich et al. (2020) to facilitate comparison with previous nightmare EEG studies.

2.3.4 | Olfactometer

During experimental nights, odours were presented using an olfactometer 'Essence' (Amores 2016) placed in a holder directed at the participant's face at 50 cm distance. Odour presentation was started by the experimenter when the participants first reached slow wave sleep, to reduce the possibility of them waking up when odour presentation began and stopped by the experimenter 30 min before their scheduled wake-up time or when they showed signs of waking up before that on the PSG, thus encompassing both NREM and REM sleep. Odours were presented in 20-millisecond bursts every 55 s arranged in four-minute loops of odour-on and odour-off periods. These parameters were chosen so that potential changes in spectral activity associated with the odour bursts could be reliably detected and habituation effects were reduced via the intermittent odour presentation. For home practice, participants received a commercially available diffuser (Aroma Diffuser Aupolo, 300 mL), which released a burst of odour every 10 s. They were instructed to place the diffuser approximately 1 m from their nose, activate it only during the relaxation exercise, ventilate the room immediately afterwards, and perform the exercise at the same time each day, at least 30 min before going to bed. Odours were obtained from Gya Labs (Los Angeles, CA, United States) and consisted of 100% essential oils derived from plants (rose geranium, tea tree, lemon grass, frankincense). Each odour served equally often as relaxation associated and control odours across participants. A constant amount of odour of 0.1 mL was used with both the 'Essence' device and the commercially available diffuser and was dispersed in a mist of distilled water.

2.3.5 | Relaxation Exercise and Control Text

The relaxation exercise used in this study was a custom-made 15-min deep breathing exercise that enabled the participants to practice deep, diaphragmatic breathing and relax. On average, participants rated the ability to follow the instructions for the relaxation exercise to be high ($M=4.04$, $SD=1.03$) as well as their ability to engage with the exercise at first trial ($M=4.01$, $SD=0.97$) on a 5-point Likert scale from 1 = 'do not agree at all' to 5 = 'completely agree'. For the week of relaxation exercise, most participants practiced every day (72% 'Yes' to 'I have completed the relaxation exercise every day for the last week.'), 12% of participants practiced for 6 days, while the remainder practiced for 5 days or less. Mean ratings of understanding of and engagement with the exercise were 4.37 ($SD=0.71$) and 4.16 ($SD=0.7$), respectively. Missed practice days did not influence results when included as a covariate.

As a control, participants listened to a text about polar research. This control text and the relaxation exercise were presented on a computer in the evening of the adaptation night and of the first experimental night. The audio recordings of the relaxation exercise and the control text were narrated by a member of the research team who was otherwise not involved in any interactions with the participants. Participants listened to the recordings through headphones at a fixed volume while sitting with their eyes closed.

2.4 | Statistical Analyses

Statistical analyses were carried out with SPSS and Matlab toolbox FieldTrip (Oostenveld et al. 2011). Differences in sleep architecture were evaluated by dependent samples *t*-tests. False discovery rate (FDR) was used to correct for multiple comparisons in analyses of behavioural data and sleep architecture. Differences in spatial patterns of band-wise spectral power for the computerised presentation of the relaxation exercise and control text, the whole night and odour-on vs. odour-off periods were examined using a non-parametric cluster-based permutation statistic (Maris and Oostenveld 2007). To this end, electrodes were selected that showed significant differences in power between conditions (two-tailed paired-samples *t*-tests, sample-level $\alpha=0.05$). In the resulting statistical map, adjacent samples were grouped into positive and negative clusters for which cluster-level statistics were calculated by summing up the *t*-values within each cluster. These were tested against a reference distribution (cluster-level $\alpha=0.05$), generated by shuffling the association of data and condition (5000 permutations) and, for each permutation, taking the maximum statistics among all clusters. In case of significant clusters, Hedge's *g* was calculated. The same tests were used for sleep spindle characteristics and all correlation analyses between neural and behavioural measures. For the computerised presentation of relaxation exercise and control text, there were subsequent 2×2 condition (relaxation, control) and time of measurement (pre: adaptation night and post: first experimental night) analysis for the beginning (2nd to 6th minute) and end (8th to 12th minute). We chose to analyse EEG activity at the beginning and the end of the exercise separately based on previous studies demonstrating that EEG patterns during relaxation exercise differ between these two phases (Jacobs and Friedman 2004, Lee et al. 2012). In line with these studies, we calculated mean EEG activity from the second to sixth minute defining the 'beginning' and the final four minutes, excluding the last minute, defining the 'end' (Jacobs and Friedman 2004). The first and last minute were excluded to avoid transition artefacts at the very beginning and very end of the exercise. Changes in questionnaires before and after the intervention week were calculated with repeated-measure ANOVAs with effect sizes in η_p^2 and correlation of questionnaire data and physiological data were calculated with cluster-based correlation analysis.

3 | Results

3.1 | Spectral Activity during the Relaxation Exercise

When comparing spectral activity during listening to the relaxation exercise and the control text, a significant difference was observed at the beginning of the audio recordings (2nd to 6th minute) with significantly less beta activity (Hedge's $g=-0.408$) in the relaxation exercise than in the control text (Figure 2). These effects did not persist to the end (8th to 12th minute) of the audio recordings. Time of measurement (pre: adaptation night and post: first experimental night) or interactions between the factors 'condition' (relaxation exercise and control text) and 'time of measurement' did not yield any significant effects.

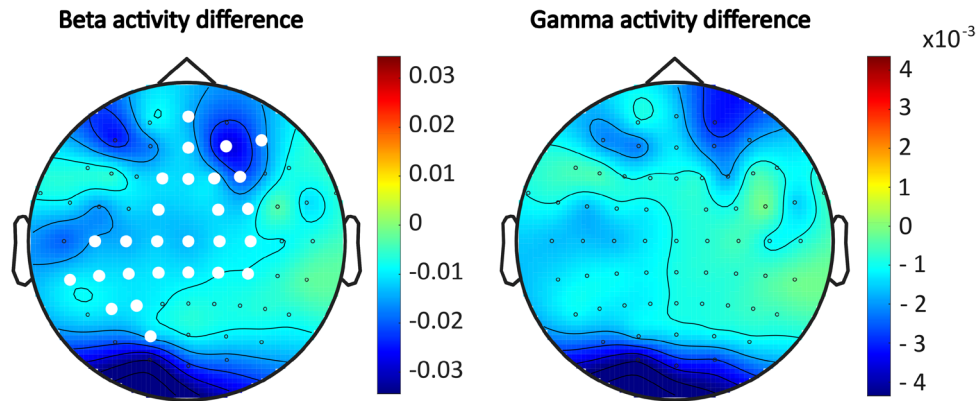


FIGURE 2 | Difference in EEG activity between relaxation exercise and control text. Topographical maps showing differences in beta (16.25–31 Hz, left) and gamma (31.25–45 Hz, right) power between the relaxation exercise and the control text at the beginning of the recording (2nd to 6th minute). Colour coding depicts spectral power measured in μV^2 . Significant electrode clusters ($p < 0.05$) are marked in white. A significant reduction in beta activity was observed during the relaxation exercise relative to the control text in a large centro-parietal electrode cluster, whereas no significant differences were found for gamma activity.

3.2 | Effects of Relaxation Exercise on Subjective Ratings

Subjectively rated relaxation rating relative to baseline relaxation ratings before listening to any of the audio recordings was significantly higher after the relaxation exercise than after the control text (main effect condition $F(24,1) = 13.206$, $p = 0.001$, $\eta_p^2 = 0.355$). There were no significant effects when comparing pre and post intervention week measurements (main effect of ‘time of measurement’) nor significant interactions between ‘condition’ (relaxation exercise and control text) and ‘time of measurement’ (pre: adaptation night and post: first experimental night). For the PANAS ratings, there was a significant effect of ‘time’ on both positive and negative affect ($F(21,1) = 18.479$, $p = 0.001$, $\eta_p^2 = 0.468$) with affect ratings generally being lower at the post than at pre-measurement. There was neither a significant effect of ‘condition’ (relaxation exercise and control text) nor a significant interaction of ‘condition’ (relaxation exercise and control text) $\times$ ‘time of measurement’ (pre vs. post) on the PANAS ratings.

3.3 | Effects of Reactivation on Sleep Architecture, Spectral Activity, and Spindle Characteristics

The comparison of sleep architecture between nights with the reactivation odour and the control odour yielded no significant results (Table 2). When comparing spectral activity in reactivation and control nights for the entire night (for NREM and REM sleep separately), there were no significant differences in the beta and gamma bands. Comparing odour on and off periods within the reactivation nights did not reveal any differences in the beta and gamma bands during NREM nor REM sleep (Figure 3).

Fast spindle count (Hedge’s $g = -0.481$) (Figure 4C) and density (Hedge’s $g = -0.511$) (Figure 4B) were significantly lower in reactivation nights than in control nights. There were no significant differences for spindle amplitude (Figure 4A) and spindle threshold (Figure 4D). When comparing odour-on and odour-off periods with respect to spindle activity, no significant

TABLE 2 | Sleep architecture during nights with the reactivation and the control odour.

	Reactivation odour	Control odour	<i>p</i>
	<i>M</i> (SD)	<i>M</i> (SD)	
Total sleep time (minutes)	435.46 (62.47)	447 (61.7)	0.285
Sleep efficiency (%)	91.35 (6.61)	90.96 (9.64)	0.776
N1 duration (%)	4.73 (2.42)	5.09 (2.57)	0.357
N2 duration (%)	52.02 (7.31)	52.63 (8.96)	0.622
N3 duration (%)	16.88 (5.29)	17.46 (5.8)	0.371
REM duration (%)	21.16 (3.85)	20.75 (5.52)	0.978
REM density	5.21 (3.16)	4.23 (2.15)	0.126
Microarousals	49.24 (23.73)	49.52 (29.46)	0.964

Note: Differences calculated by dependent samples *t*-test without the use of covariates. No differences were significant using a 0.05 false-discovery-rate (critical values 0.007–0.05).

differences emerged, as determined by cluster-based statistics with the cluster-level significance threshold set to $p \leq 0.05$. For slow spindles, there were no significant effects on any parameters (see Figure S2). Exploratory analyses revealed that delta activity during NREM sleep was significantly higher when the reactivation odour was present than when it was absent (Hedge’s $g = 0.494$), whereas this pattern was not observed for the control odour. However, there was no significant interaction between odour on and off periods and type of odour (relaxation odour vs. control odour). The observed effect was localised in a cluster of frontal and central electrodes (see Figure S3). A similar pattern emerged in alpha activity during REM sleep, with alpha activity being higher during odour on phases of the reactivation odour compared to odour off phases (Hedge’s $g = 0.136$). This was again not observable for the control odour and there was no significant interaction between odour on and off periods and type of odour.

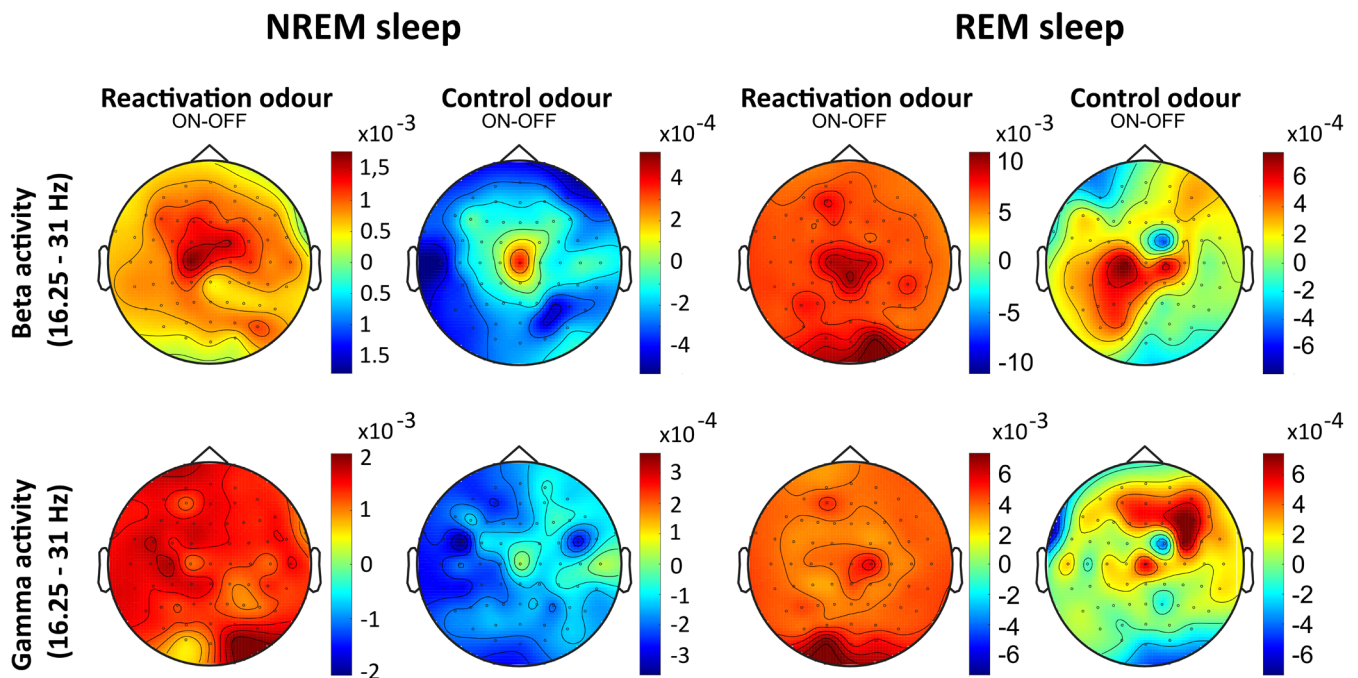


FIGURE 3 | Beta and Gamma activity in NREM and REM sleep during odour cueing. Topographical maps showing differences between odour on and odour off periods in the beta (16.25–31 Hz, top) and gamma spectral power (31.25–45 Hz, bottom) for the relaxation-associated odour (left) and the control odour (right), shown separately for NREM and REM sleep. No significant differences in beta or gamma activity were observed between odour-on and odour-off periods for either the relaxation-associated odour or the control odour during NREM or REM sleep. Colour coding indicates differences in spectral power measured in μV^2 .

The significant electrode cluster for this effect was located in a parieto-occipital cluster of electrodes (see Figure S4). No other significant effects were observed for any other comparison or frequency band during sleep, including slow oscillations.

3.4 | Effects of Relaxation Exercise on Nightmare Symptoms and Sleep Quality

One week of practicing the relaxation exercise at home did not affect retrospectively reported sleep quality as measured by the PSQI, nor did it influence nightmare frequency or the level of nightmare-related distress (all $ps \geq 0.417$ for the comparison of the measurement before and after the week of practice; Table 3).

In addition to the retrospective assessment of nightmare characteristics and sleep quality during the week of relaxation exercise, we examined direct effects of reactivation in the sleep laboratory on these parameters. A comparison of nightmare occurrence, sleep quality, and both negative and positive affect between nights with the reactivation odour and nights with the control odour revealed no significant differences. (all $ps \geq 0.249$) (Table 4).

3.5 | Correlations between Neural and Behavioural Data

For all significant differences in neural measures (i.e., beta activity during the relaxation exercise, delta and alpha activity and spindle count and density during sleep reactivation),

we examined an association with relevant behavioural data, including subjective relaxation and affect ratings during the exercise, as well as perceived sleep quality on reactivation and control nights. For the relaxation exercise, there were no significant correlations between beta activity and behavioural data (i.e., subjective relaxation ratings and PANAS ratings) all $ps \geq 0.05$ for the cluster-based permutation test statistics. However, spindle count across all nights (relaxation and control nights combined) was positively correlated with trouble staying asleep so that a higher spindle count was associated with longer subjectively reported wake after sleep onset (Figure 5).

4 | Discussion

The primary aim of this randomised, controlled, exploratory study was to determine whether reactivating a previously learned deep-breathing relaxation exercise during sleep via odour cues reduces cortical hyperarousal, as indexed by beta and gamma activity, and consequently nightmare symptoms. Although the relaxation exercise reduced beta activity during wakefulness, this effect did not translate to sleep: odour-cued reactivation did not alter beta or gamma activity, nor did it affect nightmare symptoms. Exploratory analyses, however, revealed reduced fast spindle count and density during reactivation nights, with higher spindle count being associated with greater subjectively reported wake after sleep onset. In addition, delta activity during NREM sleep and alpha activity during REM sleep were higher during odour-on than odour-off periods in the relaxation-associated odour condition.

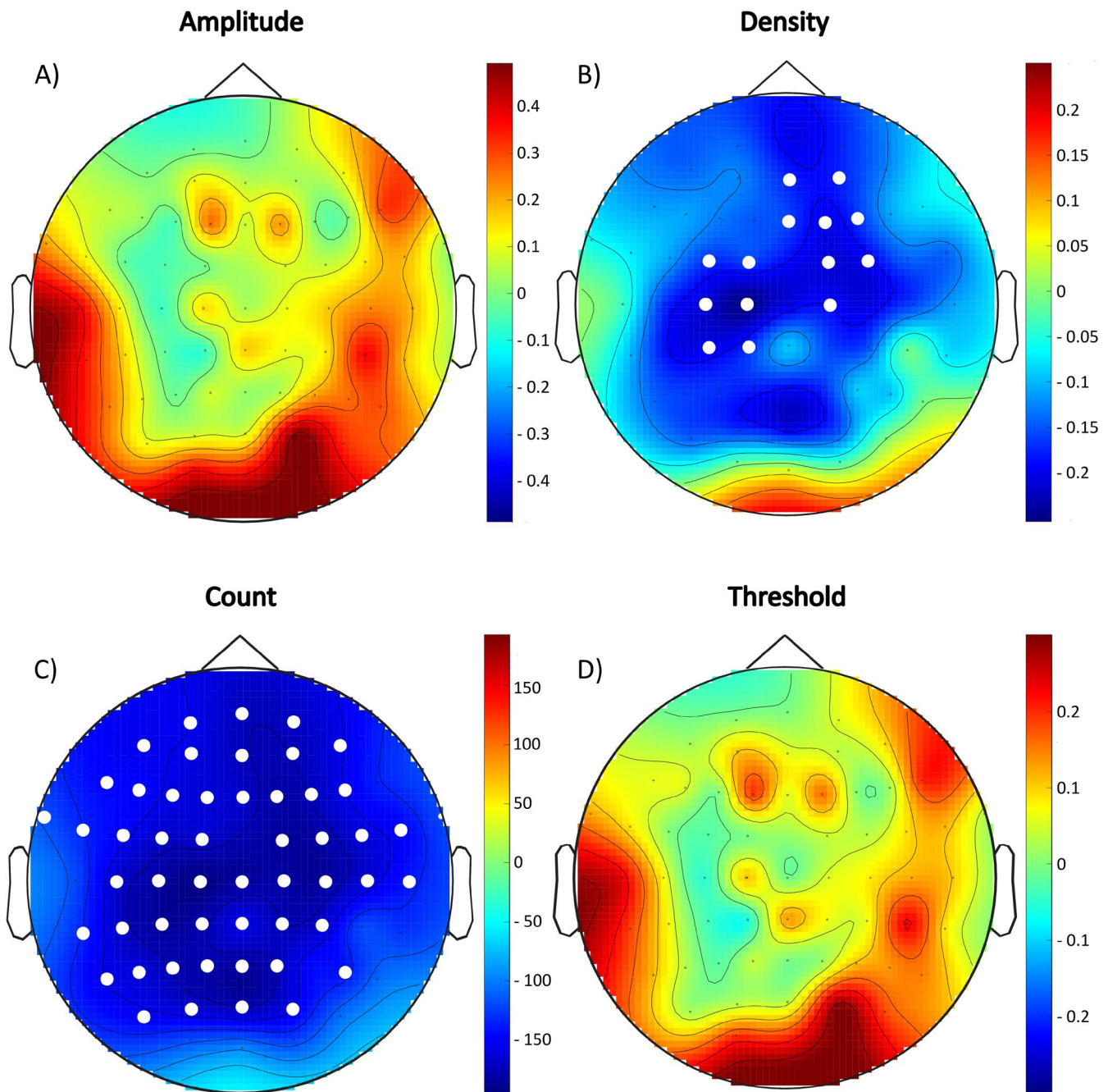


FIGURE 4 | Spindle Activity (12–16 Hz) between reactivation and control nights. Topographical maps showing differences in fast spindle parameters (from top left to bottom right: Amplitude (A), density (B), count (C), and threshold (D)) in the 12–16 Hz range between reactivation nights and control nights. Significant electrode clusters marked in white (p -alpha < 0.05). Spindle count (C) and density (B) were lower in the reactivation as compared to the control night, with significant electrode clusters in fronto-parieto-central locations.

In line with previous studies (Cheng et al. 2018), decreases in cortical hyperarousal and increases in subjective relaxation during the exercise suggest that the deep-breathing protocol functioned as intended. However, reactivating the relaxation exercise during the night did not lead to a corresponding reduction in this frequency band. One possible explanation is that cortical hyperarousal was less pronounced in the present sample than in previous studies, making further reductions more difficult to detect. Compared with our previous study, which included an older sample of patients with a confirmed nightmare diagnosis, participants in the present study were

younger and experienced less severe nightmare symptoms. For example, pre-intervention nightmare distress was lower in the present sample ($M = 3.00$, $SD = 0.57$) than in our previous study ($M = 4.04$, $SD = 0.57$), in which reductions in gamma activity were observed following imagery rehearsal therapy (Sayk et al. 2024). Consequently, the potential for further reductions in cortical hyperarousal may have been more limited. Another possibility is that the intervention itself was not optimally suited for targeted memory reactivation. Unlike cognitive or declarative contents that are able to become reactivated using TMR (Oudiette and Paller 2013;

TABLE 3 | Sleep quality and nightmare symptoms before and after 1 week of relaxation intervention.

	Pre	Post	<i>p</i>
	<i>M</i> (SD)	<i>M</i> (SD)	
PSQI ^a (sleep quality)	6.13 (1.68)	6.94 (1.89)	0.846
Nightmares/week	2.00 (0.79)	2.13 (1.33)	0.589
Degree of suffering	3.00 (0.57)	2.88 (0.97)	0.417

Abbreviation: PSQI = Pittsburgh Sleep Quality Index.

^aDifferences calculated by dependent samples *t*-test without the use of covariates. No differences were significant using a 0.05 false-discovery rate (critical values 0.016–0.05).

TABLE 4 | Comparison between nights with reactivation and control odour.

	Reactivation odour	Control odour	
	<i>M</i> (SD)	<i>M</i> (SD)	<i>p</i>
Subjective sleep quality (SF-A/R) ^a			
Sleep quality	2.59 (0.66)	2.47 (0.69)	0.484
Trouble falling asleep	3.68 (0.98)	3.72 (0.98)	0.870
Trouble staying asleep	3.24 (1.25)	3.5 (1.11)	0.249
Sleep depth	3.47 (0.58)	3.32 (0.84)	0.460
Relaxation after sleep	3.38 (0.45)	3.31 (0.67)	0.642
Nightmare occurrence (%)	25	41	0.459

Abbreviation: SF-A/R = *Schlaffragebogen A*.

^aDifferences in subjective sleep quality calculated by dependent samples *t*-test without the use of covariates. Differences in nightmare occurrence calculated by χ^2 -test. No differences were significant using a false discovery rate of 0.05 (critical values 0.008–0.05).

Born and Wilhelm 2012), deep-breathing relaxation primarily induces a physiological state, and it remains unclear to what extent such states can be successfully reactivated during sleep. Therefore, a more indirect approach, such as presenting relaxation-associated cues (Beck et al. 2021) or reactivating IRT (Schwartz et al. 2022) might yield better results on cortical hyperarousal, as further discussed below. Finally, the type of intervention could be changed entirely, so that it has greater potential to influence cortical hyperarousal. Cortical stimulation, such as transcranial direct current stimulation, can modulate high-frequency spectral activity (Kayarian et al. 2020). It was not chosen for this study as stimulation typically increases activity in the targeted frequency band and gamma-band enhancement has been associated with increased negative affect (Marshall et al. 2011). Speculatively, tDCS could therefore exacerbate nightmare symptoms raising ethical and safety concerns. Nonetheless it might be an option for future studies determining whether dream experiences change in healthy participants after stimulation to mitigate the previously mentioned ethical concerns.

Instead of beta activity, potentially reactivation-related effects unexpectedly emerged instead in delta, alpha, and spindle activity. The increase in delta and alpha activity when the reactivation odour was present may be linked to increased relaxation, in line with previous research (Beck et al. 2021; Bing-Canar et al. 2016). However, as there were no significant interactions between odour on and off periods and type of odour, these results need to be interpreted with caution regarding the causal relationship with the reactivation odour. The observed reduction in fast spindle count and density during reactivation nights was unexpected. First, it contrasts with previous TMR studies, which generally reported increased spindle activity following reactivation (Ngo et al. 2015). However, these studies reactivated semantic memories in healthy participants rather than a relaxation intervention in individuals with frequent nightmares. Evidence on spindle activity in nightmares remains scarce. Picard-Deland et al. (2018) reported alterations in slow spindle activity in individuals with frequent nightmares and related these to dream recall, fear during dreams, anxiety, depression, and emotional memory consolidation. In contrast, our findings were confined to fast spindles. One possible interpretation relates to sleep stability, as increased fast spindle activity has previously been associated with reduced subjective sleep depth and sleep quality (Stephan et al. 2021; Chen et al. 2023). Consistent with this, higher fast spindle count was associated with greater subjectively reported wake after sleep onset in the present study. Alternatively, changes in fast spindle activity have also been described in PTSD (Denis et al. 2021; Wang et al. 2020), where they have been interpreted as reflecting altered memory consolidation (van der Heijden et al. 2022). Whether similar mechanisms apply to nightmares remains unknown. Overall, the functional significance of the reduced fast spindle activity observed after reactivation remains unclear and warrants further mechanistic investigation, especially since the role of sleep spindles in sleep disorders at large is still unclear (Weiner and Dang-Vu 2016).

Despite the observed changes in sleep physiology, odour-cued reactivation did not improve nightmare symptoms. The only association with behavioural measures was a correlation between fast spindle count and subjective wake after sleep onset. Several methodological and conceptual explanations may account for the absence of clinical effects. From a methodological point of view, the reactivation dose may have been insufficient. Previous TMR research indicates that a single pairing of learning material and cue can be sufficient to induce effects at the behavioural level (e.g., Rasch et al. 2007). However, reactivating a complex intervention such as a relaxation exercise or psychotherapeutic treatment may require more extensive cueing. Consistent with this notion, 2 weeks of auditory reactivation enhanced the effects of imagery rehearsal therapy (Schwartz et al. 2022), whereas a single night of odour-cued reactivation following exposure therapy did not improve anxiety symptoms (Rihm et al. 2016). Interestingly, Rihm et al. (2016) likewise reported physiological changes following reactivation, although these differed from the present findings and were characterized by increased spindle activity. Together, these findings suggest that reactivation of complex interventions may require longer reactivation periods, with physiological effects potentially preceding measurable clinical benefits. From a more conceptual level, it is also possible that cortical hyperarousal is not a causal driver of nightmare symptoms but rather a consequence of them. In

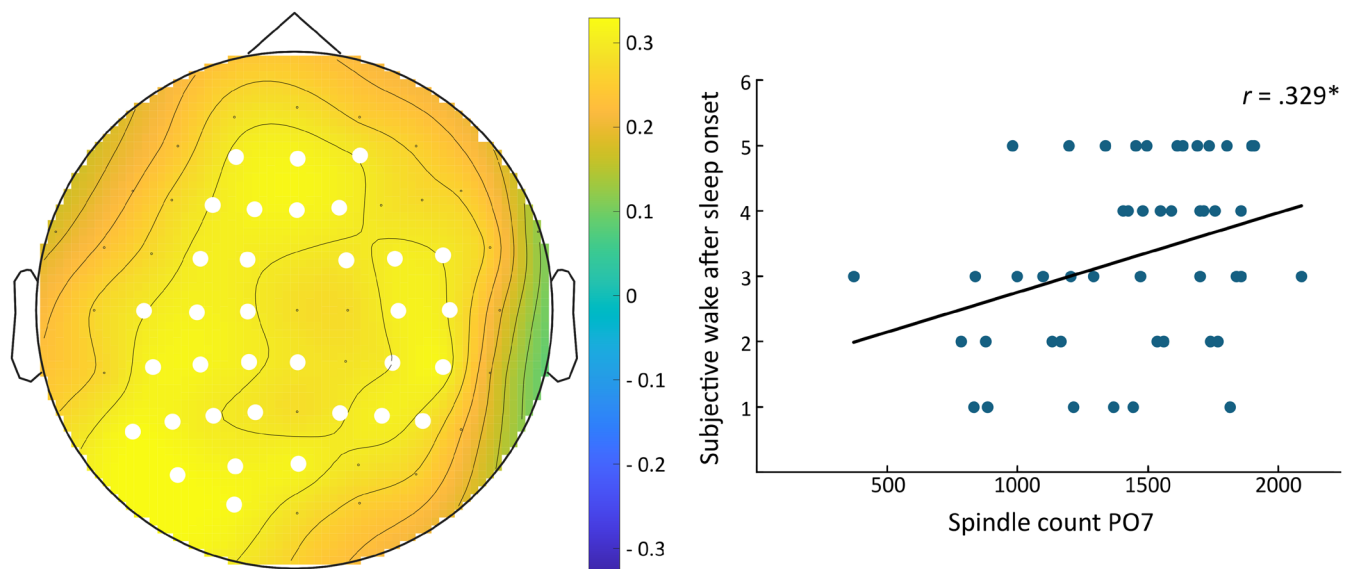


FIGURE 5 | Correlation between fast spindle count and subjective wake after sleep onset. Topographical map showing the correlation coefficients (r) between fast spindle count and subjectively rated wake after sleep onset. Significant electrode clusters identified by cluster-based permutation testing ($p < 0.05$) are outlined in white. The scatterplot (right) illustrates the correlation at the representative electrode PO7, showing that higher spindle counts were associated with greater subjectively reported wake after sleep onset.

this scenario successful treatment of nightmares reduces cortical hyperarousal, whereas reducing cortical hyperarousal alone is insufficient to alleviate nightmare symptoms. Our previous finding of reduced gamma activity following imagery rehearsal therapy (IRT) (Sayk et al. 2024) is consistent with this interpretation. Given that IRT is the current gold-standard treatment for nightmares (Morgenthaler et al. 2018) and has previously been shown to be amenable to TMR (Schwartz et al. 2022), future studies combining IRT with odour-cued reactivation while assessing cortical hyperarousal may help disentangle the relationship between physiological and clinical change.

5 | Limitations

Several methodological limitations should be considered when interpreting the present findings. First, participants were screened by telephone rather than assessed using structured diagnostic interviews according to DSM-5 or ICSD-3 criteria. Consequently, the sample may have included a heterogeneous mixture of individuals with idiopathic and trauma-related nightmares. As these subtypes may differ in their underlying neurobiology and responsiveness to treatment, this heterogeneity may have introduced additional variability, making intervention effects more difficult to detect. Second, a custom questionnaire to assess nightmare symptoms (Sayk et al. 2024) was used to increase comparability with our previous studies. However, the use of a non-validated measure may have reduced the sensitivity to detect changes in nightmare symptoms that established instruments, such as the Nightmare Distress Questionnaire (NDQ), might have captured. Third, repeated odour exposure during the week of home practice may have resulted in olfactory habituation, potentially weakening the odour-relaxation association and reducing the effectiveness of subsequent reactivation in the sleep laboratory. Fourth, olfactory perception has been shown to vary across the menstrual cycle (Pause et al. 1996) and sex

differences have been observed in olfactory TMR (Diekelmann et al. 2016). Because menstrual phase was neither assessed nor controlled for, hormonal influences may have introduced additional variability in our predominantly female sample. Future studies should therefore assess and, where possible, control for menstrual phase when investigating olfactory TMR. Finally, the present study was not preregistered. Although the primary hypotheses were specified in advance, preregistration would have further strengthened the transparency of the planned analyses. Future preregistered studies should include longer reactivation periods, alternative interventions, standardised diagnostic assessments, and larger samples that permit meaningful subgroup analyses to further clarify the causal role of cortical hyperarousal in nightmare aetiology.

6 | Conclusions

The reactivation of a deep-breathing relaxation exercise did not reduce cortical hyperarousal or nightmare symptoms in participants with frequent nightmares. Although exploratory analyses revealed changes in spindle activity and delta power, the functional significance of these findings remains unclear. Future studies should investigate whether alternative interventions or longer reactivation periods are better suited to experimentally modulate cortical hyperarousal during sleep.

Author Contributions

C. Sayk: conceptualization, investigation, methodology, writing – original draft, writing – review and editing, visualization, data curation, project administration. **A. Probst:** data curation, investigation. **K. Junghanns:** conceptualization, validation, resources. **J. Amores:** methodology, resources. **I. Wilhelm:** supervision, resources, funding acquisition, writing – review and editing, validation, conceptualization. **S. Eickemeier:** data curation, conceptualization, software. **H.-V. Ngo:**

software, formal analysis, resources, writing – review and editing. **M. Ehsanifard:** validation, conceptualization. **F. Lange:** data curation, investigation.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Figure S1:** Spectral power across the spectrum (1.25–45 Hz) in NREM and REM sleep. **Figure S2:** Slow Spindle Activity (9–12 Hz) between reactivation and control nights. **Figure S3:** Delta activity in NREM sleep during odour cueing with the relaxation-associated and control odours. **Figure S4:** Alpha activity in REM sleep during odour cueing with the relaxation-associated and control odours.