Fear in dreams and in wakefulness: evidence for day/night affective homeostasis.

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SUMMARY

Despite partial experimental evidence, it is a long-standing and common belief that dreams serve an emotion regulation function. Revisiting this idea, recent neuroscientific theories have proposed that emotions experienced in dreams contribute to the resolution of emotional distress (e.g. fear extinction) and preparation for future waking affective reactions. Here we addressed one emerging common prediction from these theoretical accounts, namely that experiencing fear in dreams is associated with more adapted responses to threatening signals during wakefulness. Using a stepwise approach across two studies, we first identified brain regions activated when experiencing fear in dreams and then showed that frightening dreams modulated the response of these same regions to threatening stimuli during wakefulness. As predicted, more fear in dreams was associated with reduced activation of those fear-responsive regions during wakefulness. Specifically, in Study 1, we performed serial awakenings in 18 participants recorded throughout the night with high-density EEG and asked them whether they experienced any fear in their dreams. Insula activity increased (i.e. decreased delta and increased gamma power) for dreams containing fear. In Study 2, we tested 89 participants and found that those with higher incidence of fear in their dreams showed reduced fMRI response to fear-eliciting stimuli in the insula, amygdala and midcingulate cortex, while awake. Consistent with better emotion regulation processes, the same participants also displayed increased medial prefrontal cortex activity. These findings support that emotions in dreams and wakefulness engage similar neural substrates, and substantiate a link between emotional processes occurring during sleep and emotional brain functions during wakefulness.

Keywords: dreaming, wakefulness, sleep, emotion regulation, fear, EEG, fMRI, amygdala, insula

INTRODUCTION

Converging evidence from human and animal research suggests functional links between sleep and emotional processes [1-4]. Chronic sleep disruption can lead to increased aggressiveness [5] and negative mood states [6], while affective disorders such as depression and PTSD are frequently associated with sleep abnormalities (e.g., insomnia and nightmares). Experimental evidence indicates that acute sleep deprivation impairs the prefrontal control over limbic regions during wakefulness, hence exacerbating emotional responses to negative stimuli [7]. Neuroimaging and intracranial data further established that, during human sleep, emotional limbic networks are activated (e.g., [8-12]). Together these findings indicate that sleep physiology may offer a permissive condition for affective information to be reprocessed and reorganized. Yet, it remains unsettled whether such emotion regulation processes also happen at the subjective, experiential level during sleep, and may be expressed in dreams. Several influential theoretical models formalized this idea. For example, the Threat Simulation Theory postulated that dreaming may fulfill a neurobiological function by allowing an offline simulation of threatening events and rehearsal of threat-avoidance skills, through the activation of a fear-related amygdalocortical network [13, 14]. Such mechanism would promote adapted behavioral responses in real life situations [15]. Other models suggested that dreaming would facilitate the resolution of current emotional conflict [16, 17], the reduction of next-day negative mood [18] and extinction learning [19]. Although these two main theories differ, because one focuses on the resolution of current emotional distress (e.g. fear extinction[19]) and the other on the optimization of waking affective reactions [2, 13], both converge to suggest that experiencing fear in dreams leads to more adapted responses to threatening signals during wakefulness. The proposed mechanism is that memories from a person's affective history are replayed in the virtual and safe environment of the dream so that they can be reorganized [2, 19]. From a neuroscience perspective, one key premise of these theoretical models is that experiencing emotions in dreams implicates the same brain circuits as in wakefulness [20, 21]. Yet, to date only indirect evidence comes from an anatomical study showing that decreased microstructural integrity of the left amygdala was associated with reduced emotional intensity (irrespective of emotional valence) in dreams [22].

Like during wakefulness, people experience a large variety of emotions in their dreams, with REM dreaming being usually more emotionally-loaded than NREM dreams [23, 24]. While some studies found a relative predominance of negative emotions, such as fear and anxiety, in dreams [25, 26], others reported a balance of positive and negative emotions [27], or found that joy and emotions related to approach behaviors may prevail [28, 29]. When performing a lexicostatistical analysis of large datasets of dream reports, a clear dissociation between dreams containing basic, mostly fear-related, emotions and those with other more social emotions (e.g. embarrassment, excitement, frustration) was found, highlighting distinct affective modes operating during dreaming, with fear in dreams representing a prevalent and biologically-

relevant emotional category [13, 30]. Thus, if fear-containing dreams serve an emotion regulation function, as hypothesized by the theoretical models, the stronger the recruitment of fear-responsive brain regions (e.g. amygdala, cingulate cortex, insula) during dreaming, the weaker the reaction of these same regions to actual fear-eliciting stimuli during wakefulness should be. This compensatory or homeostatic mechanism may also be accompanied by an enhanced recruitment of emotion regulation brain regions (such as the medial prefrontal cortex, mPFC, which is implicated in fear extinction) during wakefulness.

Here, we collected dream reports and functional brain measures using high-density EEG (hdEEG) and functional MRI (fMRI) across two studies to address the following questions: (i) do emotions in dreams (here fear-related emotions) engage the same neural circuits as during wakefulness, and (ii) is there a link between emotions experienced in dreams and brain responses to emotional stimuli during wakefulness. By addressing these fundamental and complementary topics, we aim at clarifying the grounding conditions for the study of dreaming as pertaining to day/night affective homeostasis.

RESULTS

Study 1: Identifying the neural correlates of fear in dreams

In Study 1, we performed serial awakenings in 18 participants recorded throughout the night with hdEEG (256 channels) and identified brain regions activated prior to awakenings from a dream containing fear (vs. without fear; Figure 1). We only analyzed the EEG data from those participants who reported at least one dream containing fear and one without fear, within the same recording night and for a given sleep stage (N2 or REM). We conducted spectral analyses for the 20-seconds EEG epochs preceding each awakening and compared epochs associated with the presence of fear in dreams with those without fear. We found significant modulations in the delta (1-4 Hz) and gamma (25-50Hz) ranges, which we interpreted in terms of underlying local neuronal population activity as follows. Increased (respectively decreased) low frequency power in the delta range (<4Hz) corresponds to neuronal inhibition (respectively activation) [22, 31, 32], while increased high EEG frequencies in the gamma range reflect increases in neuronal firing [33, 34] and positively correlate with local BOLD fluctuations [35].

For NREM, we obtained a total of 79 awakenings in N2 sleep from 12 participants (average per night 6.58 ± 2.13 [mean ± SD, range]). Of these awakenings, 57 yielded reports of dream experience (from which 18 without recall of any content), while 22 yielded no report. Fear was present in 14 reports (average per subject 1.16 ± 0.37 [mean ± SD, range]) and absent in 25 reports (average per subject 2.08 ± 1.03 [mean ± SD, range]). N2 reports with presence of fear (compared to without fear) were associated with decreased delta power (1-4Hz) in the right insula, and increased gamma power (25-50Hz) in the bilateral insular cortex (Fig. 1B). No significant changes were found for other frequency bands.

For REM sleep, we obtained a total of 32 awakenings from 8 participants (average per night 4.00 ± 0.86 [mean ± SD, range]). Of these awakenings, 28 yielded reports of dream experience (from which 1 without recall of any content), while 4 yielded no report. Fear was present in 12 reports (average per subject 1.50 ± 0.50 [mean ± SD, range]) and absent in 15 reports (average per subject 1.87 ± 1.05 [mean ± SD, range]). REM reports with presence of fear compared to those without fear were associated with decreased delta power (1-4Hz) in the bilateral insula and midcingulate cortex, thus partly replicating the results from N2 (Fig. 1B). No significant changes were found for other frequency bands. Although EEG source reconstruction should be considered with caution, the present data also suggest that experiencing fear in NREM and REM dreams could involve different portions of the insula, with slightly more anterior dorsal insula activity during REM (Figure 1B). Together these results demonstrate that the occurrence of frightening dreams coincided with increased activation of insula cortex during both NREM and REM sleep, and of the midcingulate cortex during REM sleep.

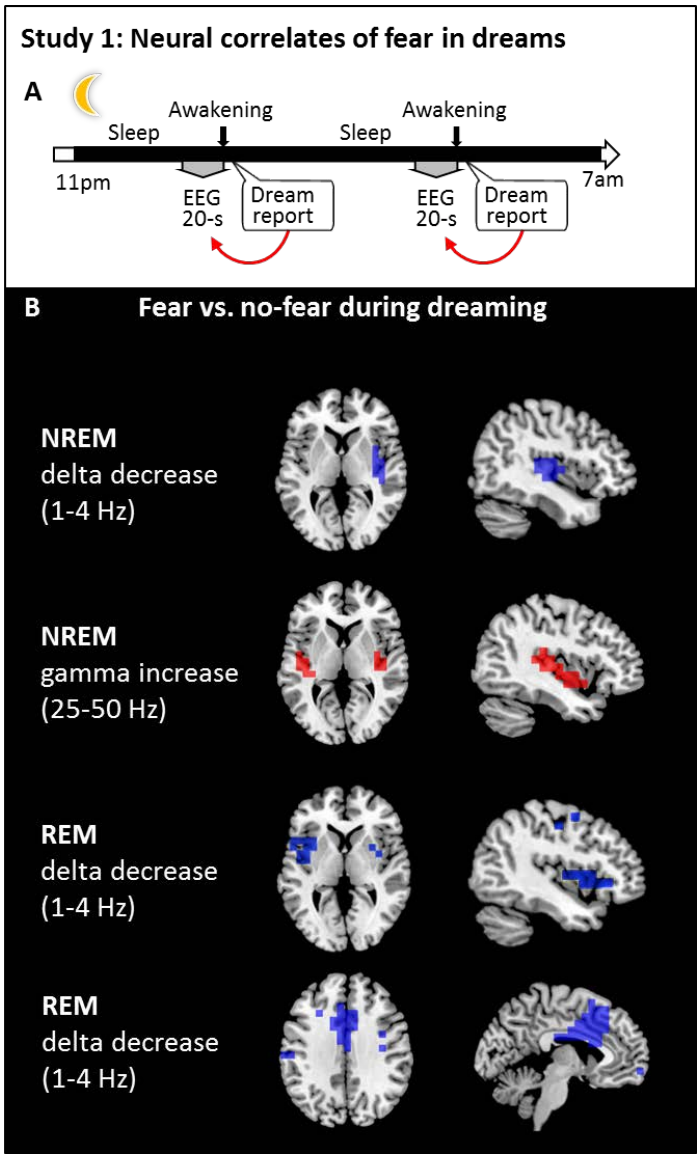

Figure 1. A. *Participants under hdEEG were awakened several times while sleeping in the sleep laboratory and were asked to report "the last thing going through their [your] mind prior to the alarm sound" and then underwent a structured interview via intercom. Participants were also asked whether they felt fear. Twenty-second epochs of EEG recording prior to each awakening were then sorted as a function of the presence or absence of fear in the dream.* ***B.*** *Brain maps showing modulations of delta and gamma power during N2 sleep and delta power during REM sleep when comparing trials with fear to those without fear. Only significant differences at the p<.05 level, obtained after correction for multiple comparisons are shown at the source level (two-tailed, paired t-tests, TFCE corrected).*

Study 2: Linking awake brain responses to aversive stimuli and fear in dreams

After we established that fear in dreams implicated some brain regions known to contribute to the processing of aversive stimuli during wakefulness (Study 1), we asked whether frequently experiencing fear in dreams may relate to individual neural sensitivity to fear during wakefulness. To this end, we collected a large amount of dream reports from 127 participants and correlated individual scores on a fear component (how frequently one individual may experience fear in dreams) with fMRI responses to aversive stimuli during wakefulness recorded in the same participants (Figure 2). All participants filled the same sleep and dream diary over 7.38 (± 4.90; mean ± SD) nights with the same instructions before an MRI session (with a minimum of 3 nights; Figure 2A). From the sleep diary, participants reported a mean sleep duration of 7.93 (± 0.76 SD) hours per night and a sleep quality of 7.02 (± 0.97 SD; on a 10-point scale from 0 - very bad sleep to 10 - very good sleep quality). From the dream diary, an average of 5.47 nights (± 3.63 SD) were associated with the feeling of having dreamt, and participants answered specific questions related to the content of their dreams after 3.75 (± 2.66 SD) nights. In particular, they were asked to indicate whether an emotion was present/absent for 8 distinct emotions (see Table S1). We performed a principal component analysis (PCA) on these data and found that the variance in emotion ratings was best explained by two main components. The first component (20.39% of the variance) distinguished between negative and positive (here "joy") emotions, thus representing emotional valence. The second component or "fear" component (16.40% of the variance) contrasted emotions related to basic negative emotions (fear, anger, sadness, disgust), with fear having the strongest contribution, and those related to non-basic, social negative emotions (i.e., embarrassment, confusion, frustration; see [30] for a similar finding). For clarity, the component reflecting basic negative emotion is described as "fear component" in the rest of the manuscript. We did not use the first component in our fMRI analysis primarily because it does not directly address our main hypothesis about fear, and because of the imbalance between the number of negative and positive emotions participants had to choose from (7 vs. 1; Table S1), as also captured by the PCA.

Using the second PCA component, we asked whether the frequency of basic negative emotions in dreams correlated with other individual characteristics. We observed no significant correlation with sleep quality (PSQI, R^2 <0.001, P=0.51), sleep duration (sleep diary, R^2=0.005, P=0.44), sleepiness (ESS, R^2=0.02, P=0.12), depression (BDI, R^2=0.008, P=0.34), anxiety (STAI-T, R^2=0.001, P=0.77), or frequency of dreaming (percentage of nights with dreams, R^2=0.003, p=0.54). This pattern of results supports that basic (mostly fear) negative emotions in dreams might represent an individual affective measure that is largely independent of sleep variables and waking affective dimensions.

At the brain level, we first looked at the main effect of emotion during wakefulness (comparing aversive vs. neutral stimuli) using fMRI data collected on 89 participants (from 3

experiments, see Methods for more details), and found a set of expected brain regions including the amygdala, the insula, and occipital regions (Table S2, Figure S1). Then, to test the hypothesis of a link between fear in dreams and brain responses to aversive stimuli during wakefulness, we used the individual scores on the second PCA component as a regressor in a whole-brain regression analysis (for the contrast aversive vs. neutral stimuli, see Methods). This analysis revealed that the medial prefrontal cortex and retrosplenial cortex (Figure 2B, Table 1) were more activated when those participants experiencing more frequent fear-eliciting dreams faced aversive stimuli while awake. Conversely, and as predicted by theoretical models (see Introduction), the same analysis yielded negative correlations with activity in the right amygdala, right insula, and midcingulate cortex (Figure 2B).

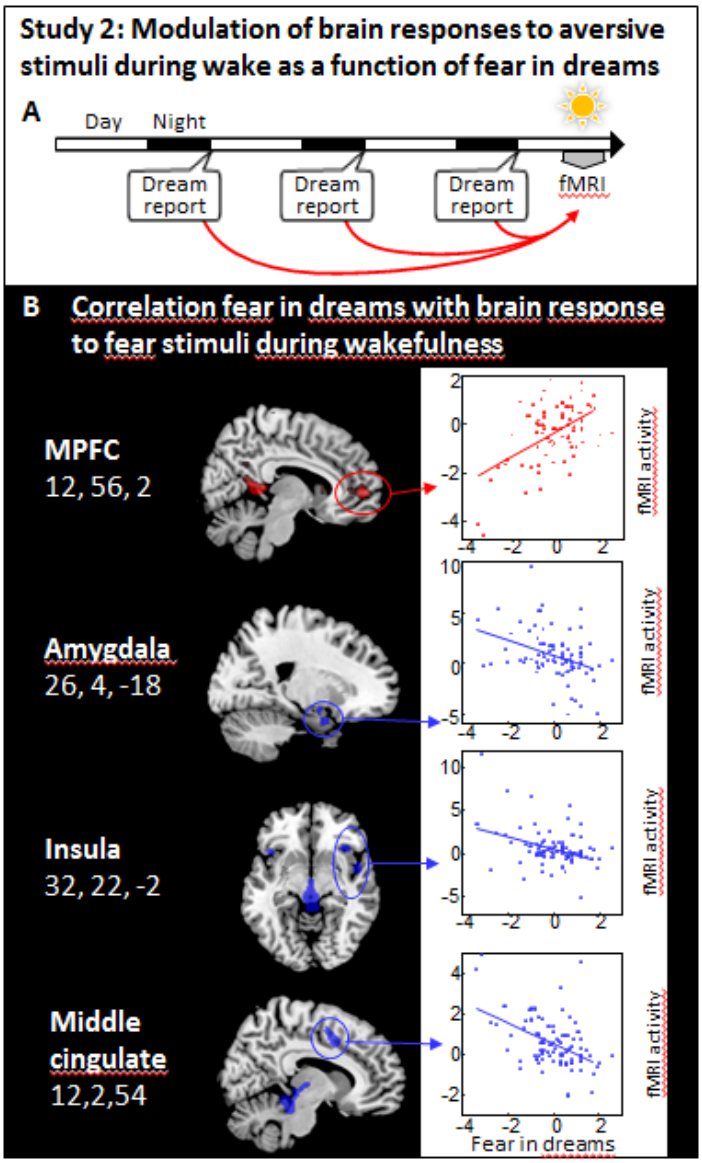

Figure 2. A. *During one week, participants filled a dream diary at home, and their responses to aversive stimuli were assessed using fMRI. To test for a link between fear in dreams and brain responses to fear stimuli during wakefulness, the individual propensity to experience fear in dreams (second PCA component, Table S1) was used as a regressor in a whole-brain analysis.* ***B.*** *Responses of the medial prefrontal cortex and retrosplenial cortex to aversive stimuli were greater in those individuals who frequently experienced fear-eliciting dreams (top panel), while activation of the amygdala, insula, and midcingulate cortex decreased in the same individuals (middle and bottom panels). Scatterplots (on the right side) show the individual data for the fMRI parameters estimates (negative vs. neutral items) and scores on the second (fear-related) PCA component. Whole-brain regression results are displayed on the mean structural image at p=0.001 uncorrected. Statistics were corrected for Family Wise Error at p<0.05 on the entire brain volume or using anatomical masks of regions of interest.*

Name	**MNI coordinates**	**# voxels**	**Z-score**	**$P_{FWE\text{-}corr}$**
Positive correlations				
Medial PFC	12,56,2	5.14	5.59	0.004 †*
Retrosplenial cortex	-12,-50,14	17	3.69	0.045*
Negative correlations				
Amygdala	26, 4, -18	35	3.48	0.008*
Amygdala/striatum	22,0,-12	35	3.48	0.006*
Anterior insula	-30, 26, 4 32, 22, -2	16 73	3.33 3.65	0.058* 0.019*
Midcingulate cortex	12, 2, 54	284	4.68	0.034 †*

Table 1: *Whole-brain correlation between fMRI activity elicited by negative (vs. neutral) stimuli during wakefulness and basic (fear) emotions in dreams (as captured by the score on the second PCA component). Results are corrected for multiple comparisons using family-wise error correction at p<0.05 (FWE-corr) i) on the entire volume (marked as †*) or ii) on small volumes defined by anatomical masks (marked as *).*

DISCUSSION

Here we investigated the neural correlates of fear in dreams and their relation with brain responses to threatening stimuli during wakefulness. In Study 1, we found that experiencing fear (vs. no fear) in dreams was associated with the activation of the insula and midcingulate cortex (the latter during REM dreams; Figure 1B), which were both also activated when experiencing fear during wakefulness (Study 2; Table S2), as also classically reported in previous research [36-38]. We recently reported that specific dream contents—such as faces, places, movement, speech, and thoughts— engage similar cortical networks as during wakefulness [39, 40]. Here we show, for the first time to our knowledge, that a specific emotional state, fear, activated the insula and midcingulate during both dreaming and awake consciousness. The consistency of the present results across brain states and their correspondence with classical work on brain structures involved in fear are encouraging. Note however that hdEEG has limited accuracy in source localization, especially for the detection of signal originating from deep cerebral structures. Thus, combined EEG/fMRI studies are needed to further elucidate the exact contribution of subcortical structures, such as the amygdala, especially during REM sleep [9, 12].

The insula, especially its anterior part, may contribute to social–emotional experience and associated visceral states, possibly giving rise to conscious feelings [41, 42], and participates in the emotional response to distressing cognitive or interoceptive signals [43]. Insula activation during dreaming could thus reflect the integration of internally-generated sensory, affective and bodily information culminating in a subjective feeling of danger, as we further discuss below. During REM sleep, we also found an activation of the midcingulate cortex, a region known to critically contribute to behavioral/motor responses to dangers [38]. Because REM sleep is characterized by activation across sensory and motor cortices [44], while muscle atonia prevents the overt expression of motor behaviors, this sleep stage could provide a well-suited physiological condition for the (re)activation of threatening situations with associated emotional and motor reactions.

Based on the results from Study 1, which suggested an anatomo-functional correspondence between fear in dreams and wakefulness, we then asked whether frequently experiencing fear in dreams may relate to the individual neural sensitivity to fear during wakefulness. In Study 2, we analyzed awake fMRI response to aversive stimuli as a function of whether participants reported a high incidence of fear in their dreams. Here we thus considered fear in dreams as an individual trait, which we determined based on the analysis of a large dataset of dreams. Interestingly, this measure of fear in dreams did not correlate with depression, anxiety, sleep quality, and the frequency of dream recall (all $p>0.05$). This observation indicates that the observed results are specific to dreaming, and not to other classical dimensions of mood or sleep/dreams. We found decreased activity in the insula, amygdala and midcingulate cortex, while activity of the mPFC and retrosplenial cortex was increased. Along with the insula, both

the amygdala and the cingulate cortex have been associated with fear and the perception of negative emotions during wakefulness [45], as we found in Study 2 (Table S2). On the other hand, the mPFC is believed to regulate the response to threatening stimuli by modulating the activity of the amygdala [46, 47]. Specifically, the mPFC exerts an inhibitory control on fear expression by decreasing amygdala output and has been associated with extinction learning (i.e., when a neutral conditioned stimulus that previously predicted an aversive unconditioned stimulus no longer does so, and conditioned response subsequently decreases) [48, 49]. Consistent with the proposal that dreaming may serve an emotion regulation function [16, 50-52], participants who frequently (but not excessively, see below) experienced frightening dreams, showed a stronger inhibition of the amygdala by the mPFC.

In the domain of sleep research (irrespective of dreaming), REM sleep was found to play a role in emotional memory consolidation [53-55], especially fear memory consolidation [56] and successful fear/safety recall [57], while both NREM [58, 59] and REM sleep stages [56, 57] have been found to promote the retention and generalization of extinction learning. In addition, it was proposed that specifically REM sleep contributes to the attenuation of the emotional tone of waking-life memories [1, 60]. Importantly, total sleep deprivation may cause a reduction of mPFC control over the limbic system, resulting in an accentuation of emotional responses to negative stimuli [7] and an impairment of extinction recall [61]. We have previously suggested that these findings from sleep studies may putatively extend to or even depend on concomitant dreaming [2, 52]. In particular, the exposure to feared stimuli (objects, situations, thoughts, memories, and physical sensations) in a totally safe context during dreaming would thus resemble desensitization therapy [62]. Besides, several studies have demonstrated that dreaming of negative waking-life experiences (e.g. divorce) contributes in a resolution of previous emotional conflicts and a reduction of next-day negative mood [16, 63]. While this was not the objective of the present work, we would like to emphasize that the present data do not allow us to make any inference about a possible immediate influence of one occurrence of one specific emotion in a dream on emotional waking experience on the following day.

Contrasting with this beneficial role of negative but benign dreams, recurrent nightmares, such as those observed in PTSD patients, might represent a failure of the fear extinction function of dreaming [19, 64]. Thus, nightmare patients may be more prone to emotional dysregulation, as suggested by one recent study reporting decreased mPFC activity during the viewing of negative pictures in these patients [65]. Furthermore, exerting ineffective emotional regulation strategies (e.g. fear suppression) and elevated anxiety during wakefulness may lead to increased excitability of fear-related memories at sleep-onset or even during sleep [66-68], namely in conditions where monitoring from the prefrontal cortex is reduced [8, 9]. Such disruption in the regulation of emotions during wakefulness and sleep has been proposed as a major contributing factor to insomnia [69].

Here, we experimentally show that, beyond sleeping, experiencing negative emotions *specifically* during dreaming promotes adapted emotional responses during waking life. One limitation of these results is that Study 2 combines data from 3 different tasks testing for brain responses to aversive stimuli (vs. neutral). This allowed us to include a very large set of participants, which is needed to exploit interindividual differences, as we do here. In all studies dream reports were collected using the same instructions and the same questionnaires. Moreover, we demonstrate highly consistent results across the 3 studies for the group effects of negative vs. neutral emotions, despite the diversity of stimuli used. As a consequence, Study 2 yielded significant results in brain regions, for which we had strong a priori hypotheses. This pattern of results is consistent with the Threat Simulation Theory [13], which claims that dreaming might serve to simulate responses to threatening events in a totally secure environment and by doing so, it would help the individual to respond in an adapted and efficient way to dangerous real-life events. The opposing neural effects of fear in dreams and during wakefulness thus support recent theoretical claims that dreaming (beyond sleep) benefits emotion regulation processes, by achieving a form of overnight affective recalibration (e.g. through extinction learning and generalization), which fosters adapted emotional responses during wakefulness [2, 19]. Studying the role of positive social interactions and emotions in dreams (especially in NREM dreams, [70]) and their link with brain responses to emotional stimuli during wakefulness may be needed to further corroborate or refine existing models. Finally, based on our results, we would like to suggest that future studies should address the potential therapeutic role of sleep and dreaming in exposure and extinction-based therapies for anxiety disorders.

ACKNOWLEDGMENTS

This study was supported by the Swiss National Science Foundation Grants 155120 (to L.P.) and 320030-159862 (to S.S.), NIH/NCCAM P01AT004952 (to G.T.), NIH/NIMH 5P20MH077967 (to G.T.), Tiny Blue Dot Inc. grant MSN196438/AAC1335 (to G.T.).

AUTHOR CONTRIBUTIONS

VS, LP, GT, and SS designed the experiments, VS and LP conducted the experiments, VS, LP, and SS analyzed the data, VS, LP, GT and SS wrote the paper.

DECLARATION OF INTERESTS

The authors declare no competing interests.

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METHODS

Study 1: Neural correlates of fear in dreams

Participants

Eighteen healthy participants were included (4 males, age 39.77 ± 13.12 years, 25-63 [mean ± SD, range]). From these 18 participants, twelve (N=12) were used for the analysis of fear vs. no fear conditions in NREM sleep (N2 stage), while eight (N=8) were used for the analysis of fear vs. no fear conditions in REM sleep. All participants had no history of neurological or psychiatric disorder. Signed informed consent was obtained from all participants before the experiment, and ethical approval for the study was obtained from the University of Wisconsin-Madison Institutional Review Board.

Procedure

Serial awakenings during sleep

Dream sampling during sleep was accomplished using the 'serial awakening' method, as described in detail elsewhere [39] (Figure 1A). In brief, participants were awakened several times during the night while sleeping and were asked to describe *'the last thing going through your mind prior to the alarm sound'*, and then underwent a structured interview via intercom. Among other questions, they were asked to name any specific emotion that they experienced, and to report the presence/absence of fear or anxiety. Awakenings were performed at intervals of at least 20 minutes in N2 sleep or REM sleep using an alarm sound. Participants must have been asleep for a minimum of 10 minutes and must have been in a stable sleep stage for a minimum of 5 minutes before any experimental awakening was triggered.

EEG recordings

Recordings were made at the University of Wisconsin (Wisconsin Institute for Sleep and Consciousness) using a 256-channel high-density EEG (hdEEG) system (Electrical Geodesics, Inc., Eugene, Ore.) combined with Alice Sleepware (Philips Respironics, Murrysville, PA). Additional polysomnography channels were used to record and monitor eye movements and submental electromyography during sleep. Sleep scoring was performed over 30s epochs according to standard criteria [71].

EEG Preprocessing

The EEG signal was sampled at 500 Hz and band-pass filtered offline between 1 and 50 Hz. The EEG data were high-pass filtered at 1Hz instead of lower frequencies as there were sweating artifacts in some of the participants which caused intermittent high-amplitude (>300uV) slow frequency oscillatory activity around 0.3 Hz. Noisy channels and epochs containing artifactual

activity were visually identified and removed. To remove ocular, muscular, and electrocardiograph artifacts, we performed Independent Component Analysis (ICA) using EEGLAB routines [72, 73]. The previously removed noisy channels were interpolated using spherical splines (EEGLAB). Finally, EEG data was referenced to the average of all electrodes.

EEG Signal analysis

Source localization

The cleaned, filtered and average-referenced EEG signal corresponding to the 20s before the alarm sound was extracted and analysed at the source level. Source modelling was performed using the GeoSource software (Electrical Geodesics, Inc., Eugene, Ore.). A 4-shell head model based on the Montreal Neurological Institute (MNI) atlas and a standard coregistered set of electrode positions were used to construct the forward model. The source space was restricted to 2447 dipoles in 3-dimensions that were distributed over 7x7x7 mm cortical voxels. The inverse matrix was computed using the standardized low-resolution brain electromagnetic tomography (sLORETA) constraint. A Tikhonov regularization procedure ($\lambda=10^{-1}$) was applied to account for the variability in the signal-to-noise ratio [74]. We computed spectral power density using the Welch's modified periodogram method (implemented with the *pwelch* function in MATLAB (The Math Works Inc, Natick, MA) in 2s Hamming windows (8 segments, 50% overlap) to decompose the source signals into frequency bands of interest before taking the norm across dimension to produce a single power value for each dipole.

Statistical Analysis

Statistical analysis was carried out in MATLAB. To compare brain activity between trials with fear and those without, source-space power was averaged within standard frequency bands (Delta: 1-4Hz, Theta: 5-8Hz, Alpha: 8.5-12Hz, Sigma: 12.5-17Hz, Beta: 17.5-24Hz, Gamma: 25-50Hz). We then averaged the power values within trials with fear and those trials without fear for each participant and for each frequency band separately. Group level analyses used paired two-sample t-tests (two-tailed) between the fear and no fear conditions, performed separately for each frequency band, and thresholded at corrected $p< 0.05$ using non-parametric threshold-free cluster enhancement (TFCE) (weighing parameters E= 0.5 and H=2) [75].

Study 2: Modulation of brain responses to aversive stimuli during wake as a function of fear in dreams

Participants

A total of 127 healthy individuals (45 males, age 22.00 ± 3.15 years, 18-37 [mean ± SD, range]) participated in the second study and filled in the sleep and dream diary. All participants had no history of neurological or psychiatric disorder. Signed informed consent was obtained from all participants before the experiment, and ethical approval for the study was obtained from the University of Geneva (Switzerland) and University of Liège (Belgium). Among these 127 participants, 13 participants did not report any dream in their dream diary before the experimental session. We also did not include 25 participants who were presented with emotional (and neutral) words, unlike all other participants who saw emotional pictures. All these subjects were removed from further fMRI analysis. The final group of 89 participants included 58 females, 21.5 ± 2.4 (mean ± SD) year-old. Participants came for 3 different experiments (Exp. 1: N=28, age 21.36 ± 2.70 years, 16 men, University of Geneva; Exp. 2: N=19, age 22.16 ± 2.48 years, 19 men, University of Geneva, Exp. 3: N=42, age 21.31 ± 2.08, 23 men, University of Liège). Forty-seven (47) subjects came from Geneva and 42 participants came from Liège. All participants filled several questionnaires on sleep quality (PSQI)[76], daytime sleepiness (Epworth sleepiness scale, ESS)[74], depression (Beck Depression Scale, BDI)[75], anxiety (State-Trait Anxiety Inventory Trait, STAI-T)[77]. These results correspond to a secondary analysis because part of these data was already presented elsewhere [78, 79]. Note that the sleep and dream diaries were designed specifically for this analysis and the stimuli of the 3 different studies correspond to similar visual items (emotional and neutral pictures, see below), activating the same brain regions.

Collection and analysis of dream data

The week before the fMRI session, participants were asked to fill out a sleep and dream diary at home (Figure 2A). Every morning, they responded to a mini-questionnaire about the content of the dreams they may have had during the preceding night, and had the possibility to then write down their dreams in more details. Among other questions in the mini-questionnaire, participants were asked to report the presence/absence of specific emotions in their dreams (anger, disgust, confusion, embarrassment, fear, sadness, joy, frustration). Note that joyful dreams may be slightly over-represented because joy was the only positive emotion among the emotions assessed. We computed the percentage of nights with dreams containing specific emotions (for example, 3 nights with dreams containing fear over a total of 5 nights with dreams leads to a percentage of fear in dreams of 60%). We entered the percentage for each emotion from each participants (8 values by participant) into a Principal Component Analysis

(PCA) to i) identify main affective components (or dimensions) according to variance explained and ii) characterize each participant by component (or factor) scores, which we can then use as regressors in an fMRI design matrix. Specifically, here we used the individual scores on the second component that contrasted basic negative emotions (fear, anger, sadness, disgust) to non-basic social negative emotions (embarrassment, frustration, confusion) (see [30] for a similar data structure; Table S1). The first component organized the data according to emotional valence, that we did not exploit here .due to the imbalance between negative (n=7) vs. positive (n=1) emotions in the original data.

Functional MRI session

Emotional tasks

Data from 3 different fMRI experiments were included in the analysis. Two of these 3 sets of data have already been reported elsewhere, but none of these former publications concerned emotions in dreams [78, 79]. Common to these 3 experiments was that participants were exposed to aversive and neutral images, and that dream data were collected. Below, we briefly describe the task used in each experiment, focusing only on those aspects that are relevant to the purpose of the present work.

In Experiment 1, participants were presented with conditioned and unconditioned faces. Stimuli were presented in an intermixed, random order, one at a time (2.5 s each) followed by a varying interval (ISI: 4–5.5 s, mean = 4.75). Participants watched the faces, and we then compared the brain responses to the conditioned (aversive) face and to the non-conditioned (emotionally neutral) faces (see [79] for more details). In Experiment 2, 60 negative, 60 funny and 60 neutral pictures were presented to participants. Each picture was displayed for 3s, preceded by a fixation cross of 1 s. Participants had a maximum of 2s to judge the valence of each stimuli on a 7 point scale (from -3: very negative, 0: neutral, +3: very funny). Here, we compared activity elicited by the presentation of the negative vs. the neutral pictures. In Experiment 3, participants saw 90 negative and 90 neutral faces (see [64] for more details). Each face was displayed for 3 s, and then participants judged it for emotional valence and arousal. Each trial started with a fixation cross for 1.5-s duration and ended as soon as participants responded, resulting in a jitter between trials (range 5.03-12.1 s). Here, we focused on the differential activation measured during the presentation of the negative vs. the neutral faces.

All visual stimuli were presented on a back projection screen inside the scanner bore using an LCD projector, which the participant could comfortably see through a mirror mounted on the head coil. Responses were recorded via an MRI-compatible response button box (HH-1 × 4-CR, Current Designs Inc., USA).

MRI acquisition

For Experiments 1 and 2, MRI data were acquired on a 3 T whole body MR scanner (Tim Trio, Siemens, Erlangen, Germany) using a 12-channel head coil. For Experiment 3, data were acquired on a 3T head-only magnetic resonance scanner (Allegra, Siemens, Erlangen, Germany). Functional images were acquired with a gradient-echo EPI sequence with the following parameters. For Experiment 1: repetition time (TR): 2200ms, echo time (TE): 30ms, flip angle (FA): 85°, field of view (FOV): 235 mm, 36 transverse slices, voxel size: 1.8 × 1.8 x 3.4 mm. For For Experiment 2: TR: 2100 ms, TE: 30 ms, FA: 80°, FOV: 205 mm, 36 transverse slices, voxel size: 3.2 x 3.2 x 3.8 mm. For Experiment 3: TR: 2460 ms, TE: 40 ms, FA: 90°, FOV: 220 mm, 32 transverse slices, voxel size: 3.4 x 3.2 x 3.4 mm. T1-weighted structural images were also acquired in each participant.

MRI analysis

Functional volumes were analyzed by using Statistical Parametric Mapping 8 (SPM8; www.fil.ion.ucl.ac.uk/spm/software/spm8) implemented in Matlab (The MathWorks Inc, Natick, Massachusetts, USA). Functional MRI data were corrected for head motion, slice timing and were spatially normalized to an echo planar imaging template conforming to the Montreal Neurological Institute (MNI) template (voxel size, 3 × 3 × 3 mm). The data were then spatially smoothed with a Gaussian kernel of 8 mm full width at half maximum (FWHM). For each participant, we used a General Linear Model (GLM) approach to estimate brain responses at every voxel, and computed the main contrast of interest (negative vs. neutral). For each experiment, the other conditions (e.g. positive items) were modeled as variable of no interest. Movement parameters estimated during realignment were also added as regressors of no interest. The resulting individual maps of t-statistics (the contrast images for each individual) were then used in second-level random-effects analyses. We used one-sample t-tests for testing common effects of negative vs. neutral stimuli. For the regression analysis, individual scores on the second component of the PCA (higher for more fearful basic negative emotion in dreams; see Procedure and analysis of dream diaries) were used as a regressor at the group level for the contrast negative vs. neutral stimuli. Brain maps of the main effects were thresholded at $p<0.05$ FWE corrected and brain maps of the correlations analysis were thresholded at $p < 0.001$ (uncorrected). Statistical inferences were corrected for multiple comparisons according to the Gaussian random field theory at $p<0.05$ Family wise error (FWE) corrected i) on the entire volume or ii) using correction on a small volumes created from anatomical regions of the AAL atlas (SVC for Small Volume Correction). The anatomical masks were created for the retrosplenial cortex, the amygdala, the insula and the mid cingulate cortex using the toolbox Anatomy [76].

SUPPLEMENTAL INFORMATION

Table S1: PCA analysis

Type of emotion	Percentage of emotion in dreams	PCA component 1 (correlation coefficient)	PCA component 2 (correlation coefficient)
Fear	19.42 ± 27.38%	-0.40	0.36
Anger	15.63 ± 23.11%	-0.65	0.16
Sadness	9.65 ± 20.10%	-0.41	0.07
Disgust	5.15 ± 13.82%	-0.62	0.04
Frustration	13.21 ± 21.16%	-0.62	-0.33
Confusion	18.05 ± 25.44%	-0.35	-0.53
Embarrassment	8.31 ± 18.59%	-0.08	-0.59
Joy	34.84 ± 32.41%	0.22	-0.64

Table S2: Functional MRI responses to aversive (vs. neutral) stimuli across the 3 fMRI experiments (see Methods). Results are corrected for multiple comparison using family-wise error correction at p<0.05 (FWE-corr) on the entire volume or at p<0.001 after small volume correction [+]

Name	MNI coordinates	# voxels	Z-score	$P_{FWE\text{-}corr}$
Negative > Neutral				
Amygdala	20, -4, -16	246	4.51	<0.001[+]
Anterior insula	-36, 26, -6	560	6.24	<0.001
	32, 16, -18	43	5.20	0.003
Inferior frontal gyrus	-32, 14, -26	115	5.87	<0.001
	44,32,-4	123	5.64	<0.001
Motor cortex	-42, 0, 46	22	4.95	0.010
Anterior middle temporal gyrus	50, -2, -24	383	6.74	<0.001
Posterior middle temporal gyrus	-54, -58, 10	617	6.16	<0.001
	52, -56, 14	875	6.06	<0.001

Thalamus	0,-16,-4	878	6.45	<0.001
Superior temporal sulcus	50, -18, 10	181	5.75	<0.001
Calcarine/lingual gyrus	-10, -78, 8	85	4.87	0.015
	20, -60, 4	92	5.19	0.003
Cuneus	14, -100, 6	27	4.98	0.009
	-28, -86, -6	45	4.90	0.013
Midcingulate cortex	10, 18, 42	254	4.43	0.002[+]
Superior frontal sulcus	42, 12, 24	35	5.04	0.007
Neu > Neg				
Medial PFC	10, 44, -8	5	4.77	0.009
Motor cortex	-52, -16, 50	27	4.91	0.013
Angular gyrus	50, -62, 46	43	4.99	0.009
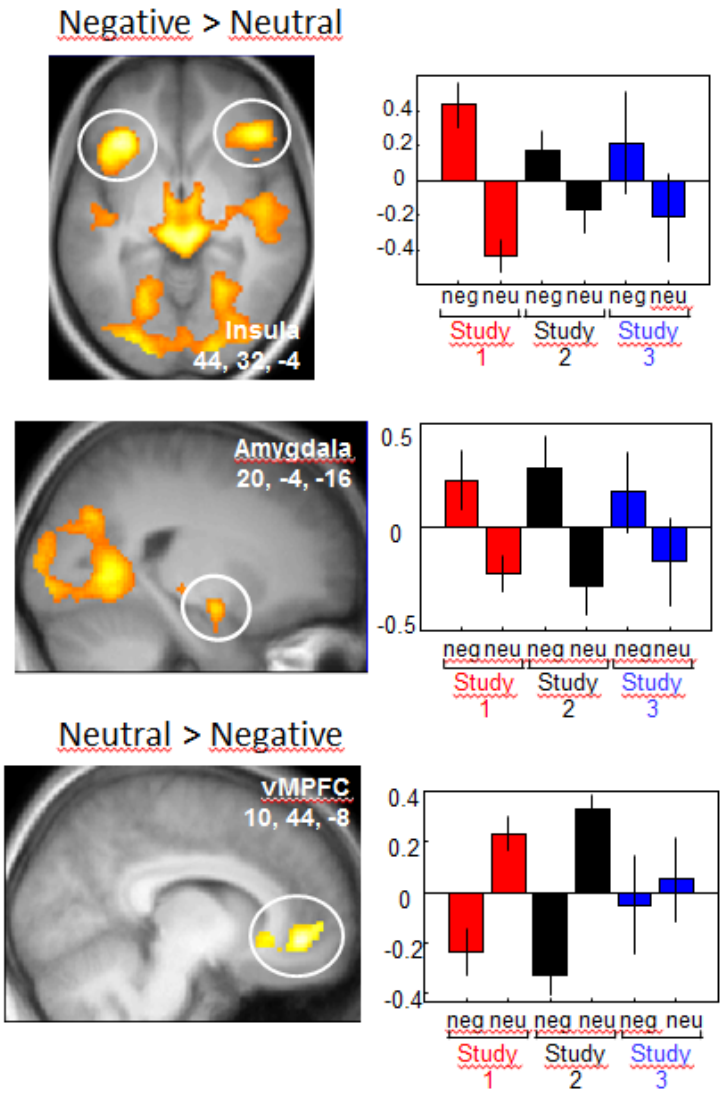
Figure S1: Functional MRI responses to aversive (vs. neutral) stimuli across the 3 fMRI experiments. Right panels show the parameters estimates for each experiment separately for negative and neutral items. For display purposes, whole-brain results are displayed on the mean structural image at p=0.001 uncorrected