1. Summary and overview

The primary aim of this grant was to conduct psychophysiological research on lucid dreaming and sensory connection during REM sleep, which can be used to optimize techniques to successfully induce lucid dreams. Methods for reliably inducing lucid dreams could help open the door to this unique state for exploration by humanity. Such a technique is widely desired but has remained elusive. In the current research, we systematically tested stimuli and physiological variables to move scientific research forward toward this goal. In Section 1, we report research results in which we systematically tested whether stimulus salience would increase incorporation rates as well as evaluated physiological conditions conducive to sensory incorporation during REM sleep. In Section 2, we report research results in which we studied the ongoing physiological conditions during REM sleep that are most conducive to becoming lucid during an ongoing dream. These two complimentary lines of research have both resulted in intriguing findings which help build the scientific knowledge necessary to realize the goal of experimentally inducing the lucid dream state.

Section 1

The first part of this project had two main aims. Our first aim was to investigate an approach to optimize sensory cues for lucid dream induction based on increasing the salience of sensory cues. Our second aim was to investigate potential differences in EEG and autonomic physiology when external sensory cues are incorporated into ongoing dreams and lead to lucidity.

Methods

Procedures. Nineteen healthy volunteers between the ages of 18 and 40 were recruited from the Madison, WI community for the experiment (7 female, mean age=23, range=19-27). All participants had normal or corrected-to-normal vision, normal hearing and no history of neurological or psychiatric disease. Participants were recorded in the sleep lab on two separate visits. Participants arrived at the lab at 7am. Setup for the recordings lasted approximately 1 hour. Participants were then allowed to sleep for a "morning nap" between 8am and 11:30am. Participants were provided with an overview of the study procedures and were instructed on how to set their prospective memory to recognize the visual cues while dreaming according to standard procedures (e.g., Baird et al., 2019).

20 channel electroencephalography (EEG) together with sleep polysomnography (PSG) including electromyography (EMG), electooculagraphy (EOG) and heart rate, was continuously recorded. Sleep stage was continuously monitored and scored online according to standard AASM sleep scoring criteria. For every REM cycle during the recording, participants received visual stimulation after reaching stable REM sleep for at least 3 minutes. Either 2 Hz or 4 Hz visual stimulation was delivered through a custom designed sleep mask in a counterbalanced order. Each stimulation sequence consisted of 3 sets of stimuli with a jittered inter-stimulus interval. Visual stimuli were delivered simultaneously through 3 LEDs over each eye in an enclosed sleep mask and were delivered in red color. 2 Hz stimulation consisted of 125 ms ON, 375 ms OFF and 4 Hz stimulation consisted of 125 ms ON and 125 ms OFF. Thus, the ON period (duration of light flash) for each visual stimulus was matched across conditions.

After conclusion of the stimulation sequence, participants were awoken via an auditory alarm. Participants then completed a questionnaire via intercom about their experience in which they gave a free report of their experience before awakening, whether they perceived the flashing lights in their dream, whether they became lucid, and the cause of their awakening (natural, alarm, stimulus-induced). If they reported that they perceived the flashing light in the dream, they reported additional details, including the number of flashes, the color the light appeared in the dream, and whether the light prompted them to check whether they were dreaming.

Signal processing and analysis. Data analysis was conducted with MATLAB using the EEGLAB toolbox and custom scripts. EEG data were bandpass filtered from 1 to 50 Hz using a two-way least-squares FIR filter. Data segments and channels containing artifactual activity were visually identified and removed. EEG data were denoised using the Extended Infomax ICA algorithm. ICA components with specific activity patterns and component maps characteristic of artifactual activity (ocular, cardiographic and myogenic) were visually identified and removed. Finally, bad channels were interpolated using spherical splines.

Data were segmented into 10 second (long) and 2 second (short) pre-stimulus epochs for further analysis. Spectral power density was computed using Welch's modified periodogram method in 2-s Hamming windows (50% overlap) to decompose EEG time series signals into frequency bands of interest. We analyzed average power spectral density (PSD) in the delta (1-4 Hz), theta (4-7 Hz), alpha (8-12 Hz) and beta (13-30 Hz) frequency bands. Statistical comparisons for sensor topographical analysis were conducted within-subjects and used two-sided paired *t*-tests between behavioral states. Group level analyses on average power values were performed separately for each frequency band. An ROI analysis was performed to examine differences in oscillatory power over the visual region (O1, O2, Oz).

Results

Participants reported an average dream recall between 2 and 4 dreams per week. In total, 14 out of 19 participants achieved stable REM sleep in the sleep lab. Visual stimuli were delivered during REM sleep to these 14 participants on 49 trials. Of these, 16 stimuli (32.6%) successfully incorporated into an ongoing REM sleep dream (see Table 1 for examples of sensory incorporations into ongoing dreams). 5 out of 21 stimuli (23.8%) were successful incorporations in the 2 Hz condition, and 11 out of 28 stimuli (39.2%) were successful incorporations in the 4 Hz condition. While the 4 Hz condition provided numerically higher incorporation rates, the difference between conditions was not statistically significant (b=0.14, p=0.24, CI=[-0.13, 0.42]). Participants woke up from the visual stimulation on 11 out of 46 trials (23.9%). Stimulus-induced awakenings did not differ between conditions (b=0.02, p=0.86, CI=[-0.17, 0.21]). No participants in either condition reported achieving lucidity as a result of the incorporated flashing lights.

Table 1. Examples of light cue incorporation during REM sleep dreaming

"I was looking at my cell phone in the dream and the phone kept flashing"
"I was playing basketball in a gym and the lights were flashing on and off"
"I was watching TV and strobes of light kept interrupting. I felt that the neighbors were responsible for the flashing and this was annoying"
"A friend and I were on a city bus talking about something. We had a polaroid camera, and it wouldn't stop flashing. The lights were so bright!"
"I was playing video games that were produced by a friend. There were Greek letters you had to tap on the keyboard for the corresponding letter. I failed. When I failed there were blinking lights."

We next compared autonomic physiology and ongoing neural oscillatory activity in the period preceding successful cue incorporation vs. no incorporation. There was no difference in heart rate (BPM) for incorporation (M=63.31, SD=15.51) compared to no incorporation (M=68.28, SD=9.71) [b=1.14, p=0.46, CI=[-2.54, 4.84]] (Figure 1a). There was also no difference in REM density (REMd) between successful incorporation (M=6.38, SD=3.32) compared to no incorporation (M=6.61, SD=4.45) [b= -0.23, p=0.85, CI=[-2.92, 2.51]] (Figure 1b). We next examined differences in EEG oscillatory power over the whole scalp and over a visual ROI. Pre-stimulus alpha power (8-12 Hz) was significantly reduced at occipital electrodes (O1,O2,Oz) for incorporated stimuli compared to unincorporated stimuli (t(7)=-2.73, p=0.03) [Figure 1c, left]. Similarly, prestimulus beta power (13-30 Hz) was significantly reduced at occipital sites for incorporated compared to unincorporated stimuli (t(7)=-2.67, p=0.03) [Figure 1c, right]. There were no other differences in EEG oscillatory power at other electrode sites.

Figure 1.

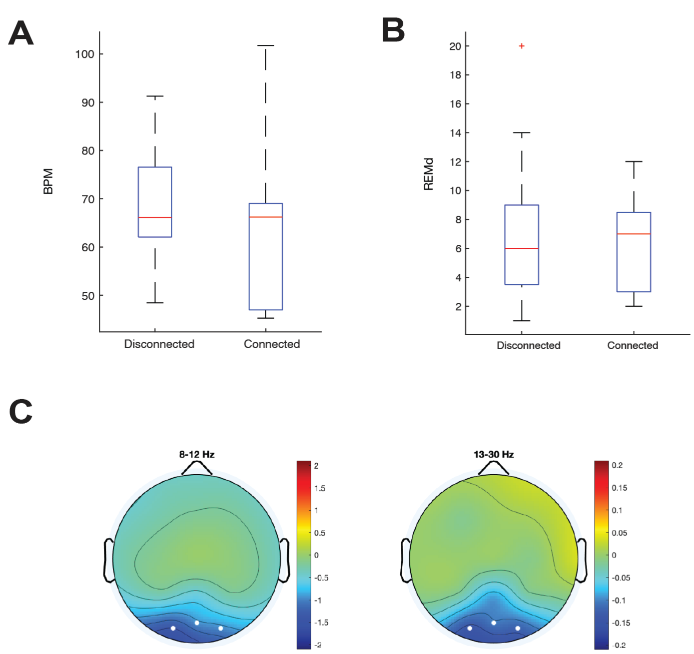

Fig 1. No significant difference in pre-stimulus heart rate (1a) or REM density (1b) were observed between successful and unsuccessful cue incorporation. Pre-stimulus oscillatory power between 8 and 30 Hz was significantly reduced in occipital regions prior to successful cue incorporation.

Discussion/future directions

The main finding to emerge from this study was that ongoing neural oscillations in visual regions in the alpha and low beta range (8-30 Hz) inhibit sensory stimuli from being consciously perceived (incorporated into an ongoing dream) during REM sleep. We believe this finding is of scientific importance for multiple topic areas and applications, including the neural mechanisms of sensory disconnection and consciousness. This result connects to a large and emerging literature of an inverse relationship between pre-stimulus activity in the 8-30 Hz range and conscious awareness of stimuli during wakefulness in paradigms such as masking and near-threshold stimulus presentation. To the best of our knowledge, the current results are the first to show a relationship between pre-stimulus power in this frequency range and (dis)connection during the REM sleep state, which, in contrast with wakefulness, is a state where a subject becomes completely disconnected from the external environment.

Unfortunately, however, in the current study these successful sensory incorporations did not result in participants achieving lucidity during dreaming even though they were able to report after awakening that they perceived the light stimulus in the dream (Table 1) as well as the fact that they knew that their task was to look for this light stimulus. Apparently, participants were able to perceive the stimulus in their dream when physiological conditions were favorable, but they lacked sufficient cognitive resources to understand its meaning as the cue/reminder that they were currently dreaming. As a next step, we therefore turned our attention to studying in greater detail the physiological conditions in REM sleep optimal for lucid dreaming itself to occur. Our motivation and reasoning is that if optimal moments for cue incorporation can in the future be paired with optimal moments for lucidity, dreamers may be best placed to be able to not only perceive the external cue but also recognize its meaning and make use of it to realize they are currently dreaming.

Section 2

In this study, our goal was to analyze the ongoing physiology and EEG characteristics associated with lucid dreams in REM sleep. As noted, our results reported above suggest that optimal lucidity induction requires more than successful cue incorporation and pre-sleep intention-setting. Our previous analysis reported above, as well as analysis of related studies, lead us to believe that future approaches to sensory induction will likely be best optimized by targeting stimulus delivery during periods of REM sleep that are most conducive to lucidity. Thus, understanding the physiological conditions in which lucid dreams are most likely to occur may be crucial for optimizing induction of lucid dreams. We therefore next analyzed signal-verified lucid dreams in REM sleep to better understand the physiological conditions most conducive to lucidity.

Methods

Participants. 14 recordings from six participants (4 males, 2 females; range 1-6) were included in the analysis. All recordings contained artifact-free EEG data with clearly discernible voluntary eye movement signals marking lucidity onset. Additional inclusion criteria included the following: a sufficient period of artifact-free EEG (> 10 seconds) following the LR2c signal in which the participant was not completing a task (see Data segmentation below), a sufficient period of baseline REM sleep (>= 30 seconds) at least 30 seconds prior to and proximal to the onset of the lucid dream eye movement signal. All participants had excellent dream recall (typically recalling more than one dream per night) and were able to have lucid dreams in the laboratory with a success rate greater than approximately 50%.

Procedure. Participants were recorded for multiple nights in the sleep laboratory. Recording on multiple nights allowed participants to become familiar with the sleep laboratory setting, procedures for recording signal-verified lucid dreams, as well as provided more opportunities to record lucid dreams. On each night, EEG (28 channels), sub-mental EMG, and vertical and horizontal EOG were continuously recorded. Participants signaled when they realized they were dreaming by means of left-right-left-right-center saccadic eye movements (referred to as “LR2c”), in which, while in REM sleep and embodied in a dream, they rapidly shifted their gaze according to the following instructions: “look at your left ear, then your right ear, then your left ear, then your right ear, and back to center. Make the signal without turning your head and make the full eye signal as one rapid continuous movement without pausing.” All signals were validated by two expert judges (SLB and BB). Upon awakening, participants were interviewed by the experimenter as to whether they had a lucid dream. For dreams in which participants reported that they were lucid, they filled out a dream report form in which they described the narrative of the dream, including the sequence of events and any thoughts, feelings or sensations that they experienced. Following standard procedures, lucid dreams were validated through the convergence between phenomenological reports of lucidity and objective eye movement signals in the EOG with concurrent EEG/polysomnography evidence of REM sleep.

Data processing. Electroencephalogram (EEG) and simultaneous polysomnography recordings were made with a Neuroscan 32 channel SynAMP system with a DC amplifier and sampled at 250-1000 Hz. Differential electro-oculogram (EOG) was recorded from the left vs. right outer canthi (horizontal EOG) and above vs. below the left eye (vertical EOG), as well as submental electromyogram (EMG). Submental EMG was scored visually in 30s epochs. Any epochs containing tonic EMG activation of twice the individual level of atonia for >50% of the epoch was marked as a loss of atonia. In addition to other PSG channels, h-reflex was also recorded from one participant. Sleep staging was performed offline using standard criteria of the American Academy of Sleep Medicine (AASM). Offline EEG data analysis was conducted with MATLAB using the EEGLAB toolbox and custom scripts. EEG data were bandpass filtered from 1 to 55 Hz using a two-way least-squares FIR filter. Bad channels were visually identified and interpolated using spherical splines. Data were denoised using the Extended Infomax ICA algorithm. Only ICA components with specific activity patterns and component maps characteristic of artifactual activity were removed.

Results

PSG REM sleep features analysis. All LR2c eye movement signals and lucid data segments occurred during unambiguous REM sleep according to AASM scoring criteria.[20] There was no difference in occipital alpha between lucid and baseline REM sleep [$\beta = -0.08$, $P = 0.63$, 95% CI (-0.45, 0.29)]. No epochs of lucid REM sleep showed tonic EMG activation. There was no difference in h-reflex amplitude between lucid (M=48.92, SD=51.88) and baseline REM sleep (M=45.11, SD=45.27) [$P = 0.63$, Monte Carlo permutation test].

Eye movements. Confirming previous findings, EM density was higher in lucid REM sleep (median: 71.17/min) compared to baseline REM sleep (median: 37.8/min) [$\beta = 0.85$, $P = 0.002$, 95% confidence interval (CI) (0.38, 1.41)] (Fig. 2). The 30 s interval preceding the LR2c eye movement signal also had significantly elevated EM density compared to the REM baseline period ($P < 0.001$). There was no difference in the density of leftward and rightward EMs in either lucid ($\beta = -0.09$, $P = 0.56$) or baseline ($\beta = 0.07$, $P = 0.76$) REM sleep. Correspondingly, the EM interval was also lower in lucid REM sleep (median: 0.72 s) compared to baseline REM sleep (median: 1.29 s) [$\beta = -0.55$, $P = 0.006$, 95% CI (-0.92, -0.18)]. EM amplitude was also higher in lucid REM sleep (median: 15.07 deg) compared to baseline REM sleep (median: 11.65 deg) [$\beta = 0.73$, $P = 0.03$, 95% CI (0.13, 1.45)]. No significant differences were observed in either average EM velocity (lucid REM median: 162.32 deg/s; baseline REM median: 154.52 deg/s) [$\beta = 0.17$, $P = 0.57$, 95% CI (-0.45, 0.91)] or peak EM velocity (lucid REM median: 301.49 deg/s; baseline REM median: 278.37 deg/s) [$\beta = 0.14$, $P = 0.65$, 95% CI (-0.53, 0.94)].

Figure 2.1

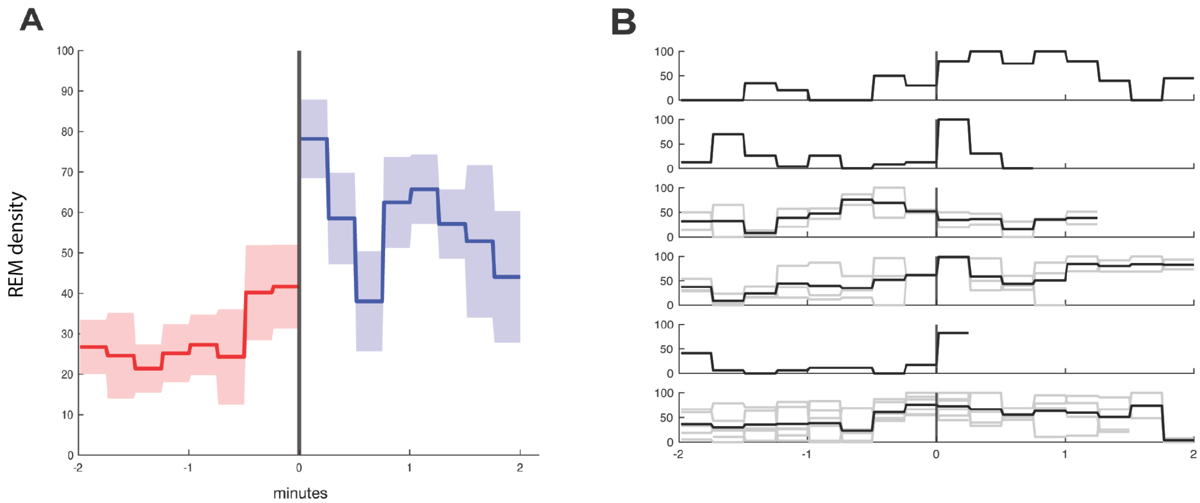

Fig. 2.1. (a) REM density from 2 minutes before the lucid dream eye movement signal (vertical black line) to 2 minutes afterwards. Each bar shows the mean (averaged within and then across subjects) for a 15 second time interval. The blue line indicates the lucid REM segment while the red line indicates the time before the lucidity eye movement signal. Error bars show the standard error of the mean. **(b)** REM density plots for individual participants. The solid black line shows the mean for that participant while gray lines show individual recordings.

EEG spectral power. We conducted an exploratory analysis of differences in spectral power in standard frequency bands across all scalp regions. Significant decreases in both delta (2-4 Hz) and beta (12.5-35 Hz) frequency bands were observed in lucid contrasted with baseline REM sleep ($P < 0.05$, SnPM cluster corrected). Reductions in both frequency bands occurred globally over widespread regions of the scalp (Fig. 5a). No other significant differences in spectral power were observed between conditions. In order to test whether these results were due to the amount of phasic and tonic REM sleep in the baseline period, we conducted several follow-up analyses. First, we compared spectral power in lucid REM sleep to a baseline REM sleep segment that matched the number of tonic and phasic segments with permutation-based resampling of segments from the baseline period. Compared to this matched baseline, a significant reduction in delta band power was observed in lucid REM sleep ($P < 0.05$, SnPM cluster corrected), while a numerical but non-significant decrease was observed in beta band after correcting for multiple comparisons. Next, we computed the distribution of spectral power in both delta and beta bands in a 4 second sliding window to visualize the distribution of spectral power for both baseline and lucid REM sleep. The results revealed overlapping distributions for both frequency bands, with a modest reduction in the number of segments for larger values and a modest increase in the number of segments for lower values.

EEG coherence. Long-range coherence was significantly reduced in the delta frequency band during lucid contrasted with baseline REM sleep [$\beta = -0.61$, $P = 0.02$, 95% CI (-1.12, -0.02)] (Fig. 5b). No significant differences in long-range coherence were observed for theta [$\beta = -0.37$, $P = 0.24$, 95% CI (-0.98, 0.34)], alpha [$\beta = 0.01$, $P = 0.98$, 95% CI (-0.55, 0.65)], beta [$\beta = 0.09$, $P = 0.74$, 95% CI (-0.49, 0.76)], or gamma [$\beta = 0.19$, $P = 0.47$, 95% CI (-0.34, 0.81)] frequency ranges (Fig. 5b). Scalp topographies depicting all channel pairs with statistically significant ($P < 0.01$, uncorrected) differences in coherence are shown in the right column of Fig. 5a.

Signal complexity. We additionally conducted an exploratory analysis of differences in signal complexity and entropy between lucid and baseline REM sleep. Higher Lempel-Ziv complexity (LZC) was observed in 13/14 recordings for threshold 1 (binomial test; $P = 0.002$) and 12/14 recordings for threshold 2 (binomial test; $P = 0.013$). However, in a LMM analysis, LZC was only significantly higher at threshold 2 (mean + 1.3 SD) [$\beta = 0.78$, $P = 0.002$, 95% CI (0.35, 1.28)], but was not significant at threshold 1 (mean + 1 SD) [β = 0.44, P = 0.23, 95% CI (-0.21, 1.20)] (Fig. 5c, left). Permutation entropy was also significantly higher in lucid contrasted with baseline REM sleep [β = 0.40, P = 0.02, 95% CI (0.08, 0.78),] (Fig. 5c, right). Increased PE was observed 12/14 recordings (binomial test; P = 0.013).

Figure 2.2

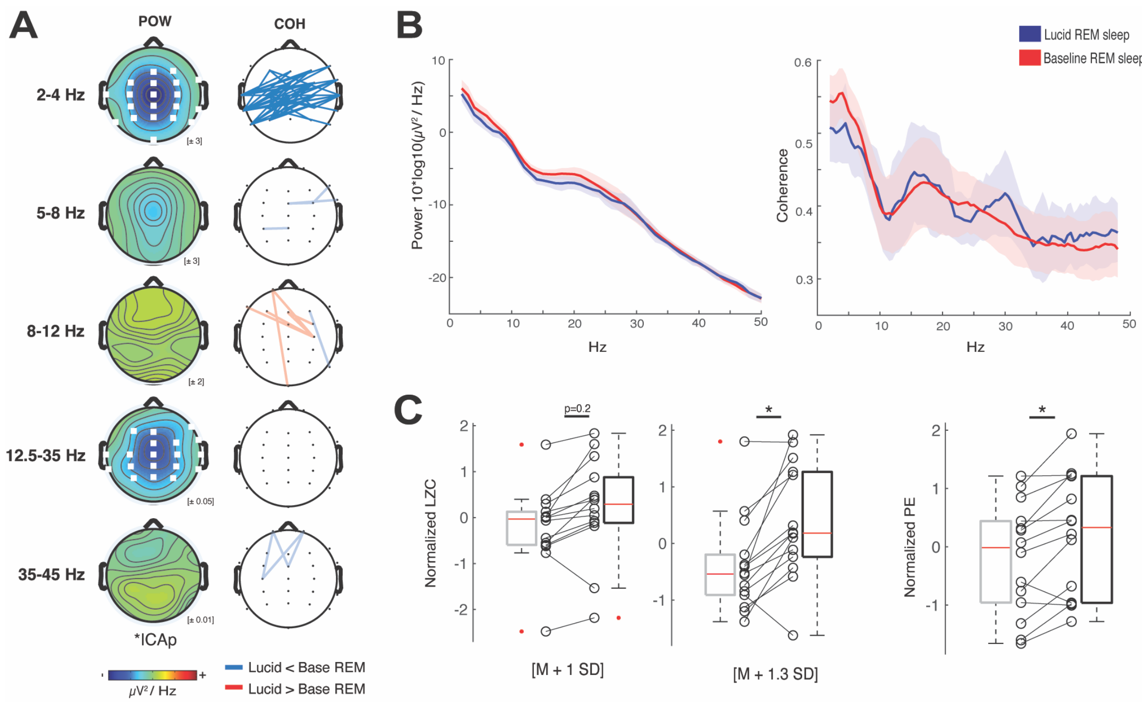

Fig. 2.2. Topography of EEG power in lucid REM sleep contrasted with baseline REM sleep for all frequency bands. White dots indicate electrode clusters with significantly decreased power, SnPM cluster corrected. **(a, right column)** Scalp plot of all electrode pairs showing increased (red lines) or reduced (blue lines) coherence. Solid lines show significant coherence clusters after corrections for multiple comparisons with spatial pairwise clustering (p < 0.05) and semi-transparent lines show all significant coherence pairs uncorrected for multiple comparisons (*p* < 0.01, uncorrected). **(b)** Group average long-range coherence values for all electrode pairs > 12 cm shown across all frequencies for lucid (blue) and baseline (red) REM sleep. Error bars show 95% confidence intervals. **(c)** Left panel: Boxplots showing normalized Lempel-Ziv complexity (LZC) for lucid (black box) and baseline (gray box) REM sleep across two thresholds. Right panel: Boxplots showing Permutation Entropy (PE) z-scores for lucid (black box) and baseline (gray box) REM sleep. The bottoms and tops of the boxes show the 25th and 75th percentiles (the lower and upper quartiles), respectively; the inner band shows the median; and the whiskers show the upper and lower quartiles ± 1.5 × the interquartile range (IQR). Circles and lines show individual recordings. Asterisks indicate significant (*p* < 0.05) differences between conditions.

Discussion

Our findings converge with previous research in finding that lucid dreaming is associated with increased physiological activation during REM sleep, including elevated eye movement density. Furthermore, our analysis revealed that lucid dreams are associated with multiple markers of cortical activation, including decreased low frequency spectral power and coherence and increased signal complexity. Together these data show that lucid dreams are associated with higher-than-average levels of cortical and subcortical activation during REM sleep. Importantly, these elevations fall within the normal variance of fluctuations in activation during baseline (non-lucid) REM sleep, while all physiological markers of REM sleep (e.g., REMs, submental atonia, suppression of occipital alpha, h-reflex suppression) are preserved.

The finding that lucid REM sleep is associated with a reduction of low frequency power and coherence compared to the entire REM sleep period replicates a previous study of lucid dreaming in individuals with narcolepsy (Dodet et al., 2014). Decreased oscillatory activity in this range during lucid REM sleep is consistent with the observation that lucid dreams tend to occur during periods of increased cortical activation, as delta power is inversely related to sustained activation of cortical neurons.

We also found that two separate measures of signal complexity—Lempel-Ziv Complexity (LZC) and Permutation Entropy (PE)—were elevated in lucid compared to the entire baseline REM sleep period. This finding is complementary to the reduction in low frequency activity and overall increase in cortical activation. Specifically, as the cortex becomes more activated, more neurons become desynchronized and as a result the complexity or entropy tends to increase.

Overall summary and future directions

Altogether, this grant allowed for us to conduct several important studies that we believe are foundational for developing a method for reliably inducing lucid dreams. First, we discovered that pre-stimulus oscillatory power in the 8-30 Hz range in visual scalp regions is reliably associated with whether an external visual stimulus will be incorporated into an ongoing dream during REM sleep. While in the current studies such successful visual cue incorporation unfortunately was not effective at inducing lucid dreams for novice participants, this finding is nonetheless important for understanding the optimal physiological conditions to deliver such cues.

Our results also suggest that lucid dreams are most likely to occur during periods of heightened cortical activation during REM sleep, including increased eye movement density, reduced slow frequency activity in the EEG, and increased signal complexity. We think that, when combined with our earlier results, these findings point to a highly promising approach for sensory induction of lucid dreams. Specifically, the findings point to a new synergistic approach to target external visual cues during REM sleep in which, 1) the overall physiological criteria and heightened cortical arousal associated with lucid dreaming is observed, and simultaneously, 2) reduced alpha/low beta oscillations are observed in visual brain areas. Our research suggests that the confluence of these two physiological states may be the most optimal moments for an external sensory cue to be both successfully incorporated into the dream and also for the participant to be able to achieve lucidity on the basis of the cue.

Developing a successful method of inducing lucid dreams necessitates solving both the problem of sensory incorporation of an external stimulus as well as the problem of the participant being able to successfully recognize the meaning of the cue to prompt lucidity. In our view, combining our findings for successful cue incorporation together with our findings regarding the physiological conditions associated with lucid dreaming is an exciting next step for this research area and holds much promise for developing a reliable means of lucid dream induction.

Overall, we think that the above studies have helped greatly advance this area of research and will be central to future research in this field. For a variety of reasons, we continue to hold that external stimulation is the most promising approach to making lucid dreaming widely accessible to humanity. Continued development and refinement of sleep technology that can deliver external sensory cues with optimized physiological targeting will be the next key step toward realizing this goal.

We are happy to report that this grant has resulted in several major publications, listed above, with additional manuscripts in preparation. We take this opportunity to send our sincere thanks to the BIAL Foundation for supporting our research.