September 15, 2020

Light, Sound, and Lucidity Signals

A non-replication of inducing morning nap lucid dreams with targeted meta-cognitive reactivation and EEG case study on the neural correlates of lucid dreaming

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Abstract

Lucid dreaming is a rare phenomena in which the dreamer is aware that they are dreaming while remaining asleep. Current focus in the field studying lucid dreams (LDs) largely revolves around reliably inducing the state in participants. Recently, a study reported a 50% success rate in inducing LDs with a protocol that combines several induction methods with REM sleep sensory cueing. In the present study, we aimed to replicate these results and additionally obtain neural correlates of lucid dreaming with high-density electroencephalography (EEG) measurements. We were unable to replicate the previous results. On the contrary, we were did not observe any verified LD (N=29). Additionally, our analysis was unable to establish an effect of the induction procedure on subjective lucidity measures. This raises questions as to what underlies these differences in results, which we attempt to elucidate.

In additional experimental sessions, one participant was able to become lucid during multiple REM periods. Thus, in a single-case study, EEG data from three verified LDs were contrasted to nonlucid REM periods using non-parametric comparisons in the spatio-spectral domain. We find that LDs are associated with increased frontocentral alpha and an increased slope of 1/f power, both of which indicate a heightened state of arousal during LDs. However, the experimental circumstances warrant caution against drawing strong conclusions about these data.

1 Introduction

1.1 Background

A lucid dream (LD) is a dream in which the dreamer is aware that they are dreaming while remaining asleep (Baird, Mota-Rolim & Dresler, 2019). Often, in a LD one can influence dream content intentionally (LaBerge & Ornstein, 1985). The occurrence of LDs have been reported in various cultures for centuries, most notably in the Tibetan Buddhist tradition where gaining awareness of dreaming while remaining in the dream state has long been part of spiritual practices (Gillespie, 1988; Wallace & Hodel, 2012).

The scientific study of lucid dreaming began in the late 1970s. Preliminary results then suggested that LDs often occur (when they do) during rapid eye movement (REM) sleep (Ogilvie, Hunt, Sawicki, & McGowan, 1978). Shortly thereafter, two groups of researchers (Hearne, 1978; LaBerge, Nagel, Dement & Zarcone 1981) were able to objectively verify the occurrence of LDs during polysomnographicly determined REM sleep by having participants perform specific pre-agreed sequences of eye movements in their dream during lucid episodes. This works because the sequence of eye movements that the dreamer executes are reproduced by their physical body (thus being measurable) due to the muscles innervating the eye being exempt from the muscular atonia that accompanies REM sleep (Aserinsky & Kleitman, 1953). Since then, asking participants to perform pre-agreed eye-signals during lucidity has become the gold-standard for identifying LDs in the field (Mota-Rolim, 2020). A LD that has been verified by polysomnographic measurements and eye-signals is commonly known as a signal verified lucid dream (SVLD).

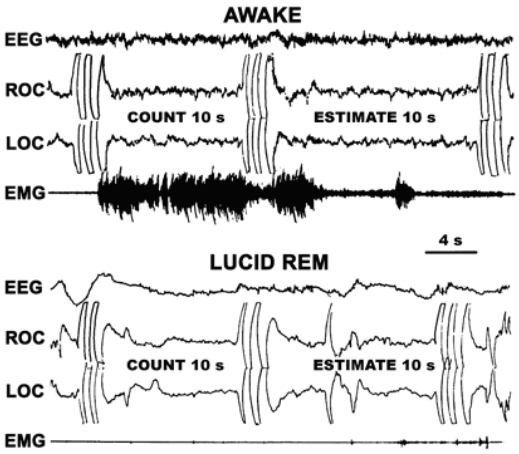

Fig. 1.1. Polysomnographic traces of resting wake participant performing left-right left-right eye movments (top) and the same eye movements during lucid REM sleep (bottom). EEG = Electroencephalogram. ROC = Right outer canthus. LOC = left outer canthus. EMG = electromyogram. Image reproduced from LaBerge & Kahan, 1994.

Demographic studies of LDs suggest they are relatively uncommon. In one study of German adults, 51% reported having had at least one LD in their life (Schredl & Erlacher, 2011). In other studies numbers have ranged from 26% (Stepansky et al., 1998) to 77% (Mota-Rolim et al., 2013). A meta-analysis of 34 studies found 55% of people

have experienced LDs at least once. Comparatively, its rare to experience LDs frequently (1 a month) which appears to only occur in about 20-23% (Schredl & Erlacher, 2011; Saunders, Roe, Smith & Clegg, 2016). Moreover, even among highly experienced lucid dreamers, most people are unable to enter a LD on demand. Because of this, it has proven difficult to study LDs in laboratory settings, causing most studies to suffer from limited sample sizes even when participants were selected specifically for their high rates of LD (Appel, Pipa & Dresler, 2017). The rarity of LDs and the difficulty of reliably obtaining data on them in controlled settings is not only a problem for more basic research, but also hinders investigations into clinical applications of lucid dreaming for treating pathological recurrent nightmares featured in PTSD and nightmare disorders (Gieselmann et al., 2019; Spoormaker and van den Bout, 2006; Holzinger et al., 2015; Gavie & Revonsuo, 2010) and for motor rehabilitation (Mota-Rolim & Araujo, 2013). Therefore, the bulk of current research focuses largely on inventing and optimizing reliable induction methods for lucid dreaming.

1.2 Methods of Lucid Dream Induction

Typically, LD induction methods involve either cognitive strategies, behavioral practices, sleep modifications, the use of external stimulation, or oneirogenic drugs. The most effective cognitive technique is considered to be mnemonic induced lucid dreaming (MILD; LaBerge, Philips & Levitan, 1994), which encompasses setting a clear intention upon going to bed to remember and recognize that one is dreaming and to vividly imagine becoming lucid during dreaming repeatedly until falling asleep. Using MILD, participants average LD frequency increased from 3.7% to around 13%. Among sleep modifying techniques, a successful method has been the Wake-Back-To-Bed (WBTB) approach, where one intentionally wakes up (usually after 4-6 hours of sleep) for 10-60 minutes before returning to sleep. This was found to increase the rate of LDs in the sample from 10% (in the first part of the night) to 40 % (LaBerge, Philips & Levitan, 1994). Cholinergic drugs have also been tested as LD inducers, primarily in combination with other strategies. A recent study (LaBerge, LaMarca & Baird, 2018) found that the administration of 4 and 8 mg of galantamine – an acetylcholinesterase inhibitor – during the wake period of a WBTB sequence led to 27% and 42% percent, respectively, of participants to become lucid in the following sleep period compared to 14 % in the placebo group. While these results are very promising, the efficacy of galantamine remains to be tested in a laboratory setting with signal-verification. Furthermore, galantamine may cause mild side effects (nausea, vomiting, gastrointestinal discomfort; Budson & Solomon, 2011) and the physiology of galantamine-induced LD might differ in important regards from LDs induced by non-pharmacological means (Blatt & Riedel, 2011) leading researchers interested in the neural correlates of lucid dreaming to pursue other methods of induction.

Another route of LD induction that has been explored is through sensory stimulation. In an exploratory study, Paul, Schdlich, and Erlacher (2014) attempted LD induction using sensory stimulation during REM sleep periods. This resulted in 5.6 % verified LD from visual stimulation and 7.4 % from tactile stimulation to the wrist or ankle, suggesting that sensory stimulation alone might not be sufficient for inducing LDs reliably. Instead, it appears that combining external stimulation with other LD techniques is more effective. In an early series of studies on the matter (LaBerge, 1988; LaBerge & Levitan, 1995), a face mask delivering light stimuli in the form of flashing red lights during REM sleep

was tested with the expectation that the light flashes would be noticed by the wearer during a dream and cue them to become lucid. The mask-delivered lights combined with the MILD technique led to 20% of dreams reported over an 8-week period being lucid (LaBerge et al., 1988). In the latter study (LaBerge et al., 1995), it was found that 88% of participants (who were frequent LDers) had at least once in a 28-day span combining the mask and MILD. Furthermore, using acoustic stimulation Kueny (1985) found 31 % of participants had a LD over 4 nights of stimulation. It should be noted that these results are limited in their interpretability due to difficulties in establishing (induction) success rates when LD frequency is measured over larger timespans and an unknown number of attempts. The lack of physiological verification of reported LDs is another limitation. Nevertheless, these results suggest that sensory stimulation combined with additional techniques could effectively induce LDs.

Indeed, several recent studies have made it clear that although no one technique is a very effective inducer of LDs alone, a combination of techniques is more likely to produce positive results (Stumbrys & Erlacher, 2014; Stumbrys, Erlacher, Schädlich & Schredl, 2012). For instance, combining WBTB and MILD with reality checks (questioning and testing whether one is presently dreaming or awake) during one week of training was associated with 53% of participants reporting a LD (Aspy et al., 2017). More recently, Appel et al. (2020) successfully induced SVLDs in 40% of participants within two nights using a combined induction technique (Stumbrys & Erlacher, 2014), while Schdlich, Erlacher, and Schredl (2017) induced LDs in nine of 15 (60% success rate) participants in one laboratory overnight. One study combined REM sleep auditory stimulation and reality testing during early-morning sleep with a 14.3 % success rate (Schmid & Erlacher, 2020). Similarly, olfactory stimulation during REM sleep combined with MILD and WBTB techniques resulted in 12.5% (N=16) in a single night sleep study (Erlacher, Schmid, Schuler & Rasch, 2020). Though these results are not particularly impressive, in a recent pilot study auditory stimulation resulted in LDs from 5 of 6 participants after 3 months of preparatory cognitive training (Kumar, Sasidharan, Nair & Kutty, 2018).

In conclusion, recent studies suggest that several methods can be combined effectively increase the incidence of LDs in the laboratory. However, studies with larger sample sizes and signal-verification are required to better assess the effectiveness of induction protocols. Furthermore, while attempts of using sensory stimulation to induce LD had relatively poor success rates, there are some indications that sensory stimulation might be effective when integrated in more extensive induction procedures.

1.3 Neurocognitive Mechanisms of Lucid Dreaming

Per definition, a lucid dream (LD) requires the dreamer to gain meta-cognitive insight into their current mental state. Therefore, it has been hypothesized that LDs occur when certain meta-cognitive processes are triggered during dreaming (Kahan & LaBerge 1994; Kahan, 2001; Hobson and Voss, 2011). In line with this hypothesis, several neuroimaging studies have associated lucid dreaming with brain structures implicated in meta-cognitive processes.

Research on meta-cognition has identified the anterior or rostrolateral prefrontal cortex (aPFC/rlPFC) as a key region involved in tasks (Baird, Smallwood, Gorgolewski & Margulies, 2013; Fleming, Huijgen & Dolan, 2012; McCraig, Dixon, Keramatian, Liu & Christoff, 2010). A recent meta-analysis of neuroimaging studies also implicated parietal

regions in meta-cognitive judgements (Vaccaro & Fleming, 2018). However, it's important to note that meta-cognition appears to be not one, but rather a category of context-dependent cognitive functions that may depend on different neural substrates (Norman et al., 2019).

The link between meta-cognitive task performance and LD was directly investigated in a recent neuroimaging study (Filevich, Dresler, Brick & Khn, 2015). While the researchers were not able to establish a direct link between LDs and meta-cognitive ability it was found that lucidity scores were associated with increased grey matter volume in frontopolar BA9/10. Additionally, increases in BOLD activity was localized to these regions during a thought-monitoring task. In another study, electrical stimulation was delivered over participant's frontal brain areas during REM sleep (Voss et al. 2014) and was associated with an effect on subjective measures of dream lucidity, indicating that the activation of prefrontal areas might facilitate LDs (see also Stumbrys, Erlacher & Schredl, 2013).

Indeed, the same research group previously published results indicating that there is an increase in 40 Hz oscillatory power in frontal and frontolateral regions during REM LDs (Voss et al., 2009). Similar results have reportedly been observed in an unpublished study (see Baird, Mota-Rolim, Dresler, 2019). Neuroimaging studies have further suggested that dream lucidity might be associated with activity in prefrontal areas. In a fMRI case study with two observations of REM SVLDs, Dresler et al. (2012) found increased BOLD activity in the aPFC during lucid trials. Furthermore, significant activations were also found in the parietal cortex and temporal gyri. Interestingly, LD frequency was associated with increased resting-state functional connectivity between the aPFC and several subregions in parietal and temporal cortices.

Taken together, these studies provide evidence that lucid dreaming may depend on neural activity in frontopolar brain regions engaged in meta-cognitive processing. Moreover, parietal and temporal lobe regions may additionally be involved. It should be noted, however, that this view has not been directly supported by results from the few EEG studies conducted on the topic in the 80's and 90's (see Baird, Mota-Rolim, & Dresler, 2019), nor by a more recent study of lucid dreaming in narcolepsy (Dodet, Chavez, Leu-Semenescu, Golmard & Anrulf, 2015). Instead, Dodet et al. (2014) found that LDs were associated with lower spectral power in the delta frequency range as well as lower coherence between delta, theta and beta-band activity in frontal and central electrodes.

Most published studies on the neural mechanisms of lucid dreaming have been severely underpowered, primarily due to the difficulty of obtaining neural measures of ongoing verified LDs. Despite this limitation and, in general, widely disagreeing results (see Baird, Mota-Rolim & Dresler, 2019), multiple experiments have yielded converging evidence that well complement the conceptual-level link between metacognition and lucid dreaming. While further investigations are needed before any confident claims can be made about the neurocognitive basis of lucid dreaming, this suggests a path for future studies to explore.

1.4 Study Aim & Hypotheses

In Part I (Testing Targeted Lucidity Reactivation) of this study, we assess the effectiveness of a presleep cognitive training combined with visual and auditory stimulation for LD induction in morning naps. To this end, we apply and replicate an induction method

and protocol developed by Carr et al. (2020) that resulted in a 50% success rate (N=41) of signal-verified lucid dreams (SVLDs) induction in the laboratory. The induction method, which Carr et al. (2020) refer to as Targeted Lucidity Reactivation (TLR) combines elements of WBTB and MILD with external stimulation and is inspired by a methodology in sleep and memory research known as Targeted Memory Reactivation (TMR) where associations between cues and memories are formed during wakefulness in order to later replay the cues during sleep and reactivate the associated memories (Oudiette & Paller, 2013). Hence, the TLR method aims to form associations between sensory stimuli and a lucid mind state during wakefulness and subsequently replay these stimuli during REM sleep to trigger dream lucidity.

We expected to replicate the primary results reported by Carr et al. (2020); finding a significant positive effect of the TLR procedure on SVLD induction with a comparable odds ratio (2.45). While replicating high success rates in LD induction was one aim of our study, a primary motivation for adopting this induction procedure was to extend previous results by obtaining neural correlates of lucid dreaming from a larger sample of healthy volunteers. We, therefore recorded brain activity using high-density EEG during experimental naps, as opposed to the minimal PSG setup used by Carr and colleagues. Furthermore, in order to increase the number of SVLDs and non-LDs in our sample, several subjects were tested on repeated occasions. To accurately model EEG source activity, 3D head modeling, and anatomical magnetic resonance imaging (MRI) data were also collected.

In order to address additional research questions that will not be further discussed here, an alternative TLR protocol inspired by body scan meditation techniques was also employed in our study. Moreover, we collected sleep and dream journals from participants for 7 days leading up to experimental morning naps.

Our study was unsuccessful in replicating Carr et al.'s (2020) results; we did not observe any SVLDs during the first nap in our sample. However, one participant had three SVLDs in two subsequent experiments. In Part II (EEG case study) of this study, EEG data from these SVLDs were contrasted against four nonLDs recorded from the same individual to find neural correlates of lucid dreaming through exploratory analysis. On the basis of previous investigations and theoretical considerations, we expected to find higher spectral power in the gamma frequency range during LDs localized to anterior-frontal electrodes. Additionally, it was expected that SVLDs would be associated with increased EEG FC between the gamma wave activity at these sites with parietal, and temporal channels.

To further explore spatio-spectral differences between lucid and nonlucid dreaming, we also employ a recently developed method to decompose spectral power into its oscillatory and fractal (1/f) components (Wen & Liu, 2016), which are thought to have distinct mechanisms and functional properties (Buzsáki, Anastassiou, & Koch, 2012; He et al., 2014). Importantly, neuronal oscillations have attracted research interest for a long time and have linked oscillatory activity to specific underlying sources and mechanisms (Mayer, Schwiedrzik, Wibral, Singer, & Melloni, 2016; Mehta, 2015). In contrast, the functional role of 1/f activity – which accounts for a majority of signal power – is poorly understood, but being investigated more and more (Muthukumaraswamy & Liley, 2018). Interestingly, 1/f-related activity has been observed to be modulated during altered states of consciousness induced by psychedelics (Atasoy et al., 2017; Timmerman et al., 2019),

propofol as well as by vigiliance states (Lendner et al., 2020). Therefore, understanding how oscillatory and fractal power components fluctuate between lucid and nonlucid REM sleep could provide valuable additional insight.

2 Methods

2.1 Data Acquisition Part I: Testing Targeted Lucidity Reactivation

Participants & General Procedure. 29 healthy participants between ages 18 and 40 (8 male, 21 female, mean$_{AGE}$=23.28 SD=4.28) were recruited via flyers and the Radboud Research Participation System at Radboud University. Participants were invited for a first meeting to inform them about the setup of the study, to get Informed Consent (ethics approved by the Research Ethics Committee of the Faculty of Science of Radboud University), and to fill in several questionnaires for screening and evaluation of their eligibility to participate. Inclusion criteria were to have had at least 1 LD in their life and recall on average 3 dreams per week and to be fluent in either Dutch or German. Participants diagnosed with psychiatric or neuropsychological disorders, that suffered from sleep-related disturbances or consumed psychotropic medications were excluded from the study. Priority was given to participants with frequent LDs, consistent sleep schedule in the month before the assessment, low self-reported sleep latency, and good sleep quality. After screening, participants were scheduled for an experimental session at a later date and were blindly assigned to. During 7 days leading up to this experimental session, participants were asked to record an oral dream journal upon awakening each morning and fill in a questionnaire that included items related to both sleep and dream experiences.

Experimental sessions began at 07:00. Upon arrival, participants were fitted with an electroencephalography (EEG) cap and external electrodes for polysomnography (PSG) measurements. Attaching the electrodes and ensuring an adequate signal quality typically took 2 to 2.5 hours. During this participants were orally informed about the definition of a lucid dream, how lucid dreams can be objectively verified and details of the experimental procedure. Thereafter, PSG-fitted participants had the 3D position of electrodes on their head recorded using the Structure Sensor by Occipital (https://structure.io) before being led to a Faraday-shielded bedroom. From this room, participants were monitored using a thermographic camera, and two-way communication was facilitated via an intercom system. While lying in bed, participants went through one of two possible (randomly assigned) lucid dreaming training protocols for 20 minutes.

During the training, audio and visual cues were played to the participant alternatingly with 1-minute intervals simultaneously as the participant was given oral instructions to associate these stimuli with being in a state of critical self-awareness. After approximately 5 minutes of guided training, participants were asked to continue with the training independently for 10 more minutes while the cues continued to be played as before. After this period, if participants had not yet fallen asleep they were told that they can now try to sleep. Following training, participants were given a 2 to 2.5-hour opportunity to sleep while their PSG was continuously monitored. During this period, whenever a participant entered REM sleep the cues were again played (cued condition). After a maximum of 10 administered cues or 3 minutes of cues, participants were awakened for dream reports collected via a semi-structured interview. In the non-cued condition, cues were not played and the participant was awakened for dream reports after 3 minutes. At the end of the experiment, participants filled in the same questionnaire as in their sleep diary. From start to end the experiment including the PSG setup took 5 hours maximum to complete.

Participants who reported lucidity or had high ratings of subjective lucidity during the experiment were invited for two further repetitions of the same procedure or a variation thereof attempting to establish two-way communication during sleep (see Konkoly et al., in preparation). T1-weighted MRI scans were acquired for EEG source localization.

Questionnaires. During the screening, participants completed a general demographic questionnaire, a dream frequency, and attitudes questionnaire, the Mannheim Dream Questionnaire (MADRE; Schredl, 2014), the Munich Chronotype Questionnaire (MCTQ; Roenneberg, Wirz-Justice & Merrow, 2003), the Munich Parasomnia Screening (MUPS; Fulda et al., 2008), the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) and the Plymouth Sensory Imagery Questionnaire (PSI-Q; Andrade, May, Deeprose, Baugh, & Ganis, 2014).

Sleep & Dream Journals. In the 7-days leading up to the experimental nap, participants were instructed to record a dream journal as soon as they woke up. They were instructed to in as much detail as they can recall, retell their experiences of the previous night's sleep and provide information about any dream scenarios, sensory experiences, animate objects, emotional valence, conversational content, and thoughts that they may have experienced. It was also stressed that it is normal that one does not always recall any dream at all, quickly forget specific details and that they need not feel compelled to remember anything. They were furthermore instructed to proceed to fill in the sleep questionnaire, which contained several items about bed and rise time, nocturnal awakenings, self-ratings of various aspects of sleep quality as well as questions about dream experiences (see De Gennaro et al., 2003 for details) in addition to the Dream Lucidity Questionnaire (DLQ; Stumbrys, Erlacher & Schredl, 2013).

Electroencephalography & Polysomnography. During the experimental nap, polysomnographic data were acquired from participants using custom-made 128-channel EEG caps (BrainVision BrainCap) with electrode placement according to the standard 10-5 system (Oostenveld & Praamstra, 2001). In addition, electrodes were placed 1,5 cm away from the outer canthus of both eyes for horizontal electrooculography (EOG) and above and below the sclera of the right eye for vertical EOG. Electromyographic activity (EMG) was recorded submentally with electrodes attached to the chin area. Heart rate was measured with one electrocardiography (ECG) electrode placed below the collarbone on the left side. The signal was recorded using BrainVision Recorder at 500 Hz. A subset of electrode channels (see AASM, 2017) was displayed for online sleep stage detection.

Offline, PSG recordings were sleep scored according to standard methods (AASM, 2015) using SpiSOP, RRID:SCR_015673 (see www.spisop.org) by three researchers, of which one researcher was blind to the administered experimental conditions. Scorings from each researcher were later compared for congruency. Where disagreements appeared the researchers attempted to reach a consensus where the blinded scorers' assessment was weighted heavier.

Audio & Visual Cues. The audio stimulus was a three-tone sequence of 500-700-900 Hz delivered with a 200 ms inter-tone interval. The sounds were delivered using two symmetrically positioned speakers on either side of the participants bed ends. The visual cue was a red light, flashed three times at a rate of 500 ms. This light was delivered via a

LED strip attached to a plastic pipe that arched across the bed above the participants' head such that the light was visible in 180 degrees around the bed.

The intensity of both stimuli was calibrated while the participant was lying in bed with lights off before presleep training. The participant was instructed to indicate a level of the signals such that they were noticeable but would not arouse them when played. In addition to the cues, a white noise sound generated using Audacity (https://www.audacityteam.org/) was played in the background continuously throughout the experiment to cancel environmental noise. The level of the white noise was adjusted to the individual participant's preference such that it was deemed whisper-quiet and at a comfortable level.

Lucidity Eye Signal. During the PSG setup, participants were explained that LDs can be signal verified during REM sleep if they can perform a specific eye signal when becoming lucid in a dream. They were told that the eye signal is given by looking as far to the left as possible, as far to the right as possible in quick succession twice (LRLR signal). Moreover, they were requested to continue giving the eye signal with 10 to 15-second intervals if they become lucid during dreaming, to the extent that they remember and can. After this instruction, they were asked to provide the eye signal with open eyes while a researcher checked to make sure they performed it correctly. Later, as the participant was lying in bed, they were again asked to perform the signal to ensure that it is performed properly, clearly visible on the EOG trace, and is recorded during wakefulness. During the lucidity training participants were instructed not to overtly perform the eye signal, but that they should only do so if they believe that they are having a or notice the cues while asleep.

Presleep Cognitive Training. During the pre-sleep cognitive training (TLR training) was given in either Dutch or German according to methods described in Carr et al. (2020) — which will be refered to here as the standard training (S) — or as a variant of the method which incorporated a body scan meditation (BS). Both training conditions began with the following instructions:

> Now we are going to practice becoming lucid. We want to train your mind to recognize the flashing lights and beeping sounds as lucidity cues so that you can have a LD. While you rest here, we are going to play the cues at approximately 1-min intervals. Whenever you hear or see one of the cues, you should remain in the same position with your eyes closed, but you will become lucid by attending to where your mind has been, attending to your body, and attending to your surroundings. Focus on how aspects of your experience might be in any way different from your normal waking experience. You do not need to do eye signals while awake, only do the signals when you become lucid in a dream.

For further details about the S procedure, please see Carr et al (2020). In the BS condition, the above instructions were followed by:

> Now I will teach you a method to help you become aware of yourself. This training is divided into three parts. First, I ask you to become aware of yourself and your thoughts. Then, I will ask you to focus your attention on your body and to go through it with your attention. Lastly, focus on your thoughts and where your mind is wandering. Try to imagine everything as vividly as possible. These are the instructions for the lucid dream task and lucidity signal: Become aware of the lucidity. Perform the LRLR eye movement to signal that you are lucid. Focus on your body

> and investigate sensations in different body parts as you will be instructed in the following body scan technique. Give the lucidity signal again every 10–15 seconds. Focus on the dream scene and try to interact with your environment and characters within it. Give the lucidity signal again. Try to repeat these steps until you wake up.

After these instructions had been given, the audio and visual cues were played alternatingly at intervals around 1 minute (adjusted to be timed to simultaneous oral instructions) for 5 to 6 minutes while the following instructions were read:

> Gently close your eyes. As you notice the cue, you become lucid. Observe your breathing... [pause]... remain critically aware, lucid, and notice how aspects of this experience are in any way different from your normal waking experience. Take a moment to breathe gently as we get into deepening your practice [pause] Make use of your breath as an anchor to just ground yourself in this moment. [pause] And so now try to vividly imagine yourself from the outside and become critically aware of your body. Bring your awareness and imagination to your feet and notice the sensations in the soles of the feet the toes the top of the feet and up to the ankle joints [pause] bring up your sense of curiosity to this practice as if you never noticed ths sensations before and shifting your awareness up from the feet and ankles and the legs [pause] and shifting up from there into the hips[pause] and shift the attention up from there now to the torso[pause] being aware of the back region[pause] the chest the abdomen[pause] and being aware now of the arms and the hands [pause]and now bring the attention from the torso to the shoulders[pause] and just being aware of up from there now to the neck[pause] and from the neck to the face, noticing sensations into entirely the face [pause]and breathing in breathing out and releasing any awareness on the head and the face and the torso and arms[pause] and the hips and the legs and the feet and [pause] And again observe yourself from the outside, and vividly imagine your body and now just coming back to the breath move your attention from your body to your thoughts. Try to focus on the last important dream you can remember thinking about the important events of the dream, try to live again the entire scenemove your attention to the surroundings, the scenes, the landscapes. Try remembering the characters of your dream the way they moved the sound of their voices the conversations you had Now again, try to focus on how that experience was in any way different from your everyday life. Now focus on what could make you understand that was a dream try to think of yourself as lucid and capable of changing the dream to something you would enjoy something that could make you feel good, or better, in that situation. Now imagine yourself as lucid focusing on the surroundings, the characters, your action, their voices try to change the aspect of the place youve been the scenes the words you said and heard the action you madeNow again bring your attention to your thoughts, notice where your mind has wandered[pause]Now observe your body, sensations, and feelings.

The participant was then told that the cues would continue to be played in intervals for the next 10 minutes without guidance and that each time they observe a cue they should observe their thoughts, body, and emotions while continuing to practice the body scan they just learned, attempting to perceive how aspects of their experience are different from normal waking life. Finally, for the last minutes of the training, unless the participant had already fallen asleep, they were instructed that they can go to sleep but that they should continue to practice becoming lucid each time they notice a cue, which will be delivered at longer and longer intervals. A reminder was given that the signals might be played again when they sleep and that they should remember to give the eye signal if they become lucid in a dream or noticed a cue.

REM Sleep Cueing. Cued or non-cued were balanced to obtain an even distribution of each condition between and within participants.

Cued Conditions. When the experimenter detected 30 s of uninterrupted phasic REM sleep. They began playing the audio and visual cues alternating at 15 to 30 s intervals. If an arousal was observed during this period, the experimenter waited for the arousal indication (e.g. elated muscle tone) to dissipate before the next cue was played. Participants were instructed to give the LRLR lucidity signal when becoming lucid at 10–15 second intervals or every time they noticed a cue. If the signal was given by the participant, cues were not played any further until the participant woke up or did not give the lucidity signal for more than 30s, in which case they were woken up for a dream report. If the participants did not perform any LRLR signals after 10 cues, they were also woken up.

Non-cued Conditions. Upon detection of phasic REM sleep, the experimenter did not play any cues and waited 3 minutes before awakening the participant. If the participant displayed the LRLR signal the experimenter let the participant continue to sleep until 30 s passed before a previous LRLR signal. Once participants were awake, dream reports were collected.

Dream Reports. Immediately upon awakening, participants' were interviewed via intercom. They had previously been given instructions that we may wake them up during the experiment to ask questions about what they were experiencing, at which point they should remain still and recollect their experience. The experimenter interviewed according to the following outline:

> [Call Name]—Can you describe anything that was going through your mind just before I called you? Is there anything else you can remember, any thoughts or feelings, sensations, or sensory experiences? Were you aware that you were dreaming? Did you see or hear any of the cues, the flashing lights or beeping sounds? (If yes, how many visual, how many audios; what did the cues look/sound like in the dream). Did you do any eye signals? (If yes, how many, what was it like, what were you thinking as you did them).

When necessary, clarifying open-ended questions were asked following these standard questions. Thereafter, the participant was asked to rate on a scale of 1–9 the level of control/awareness/lucidity/perception of visual cues/perception of audio cues that they experienced during their dream. Finally, the participant was asked to respond to the DLQ. Audio recordings were made of the dream interview and scale ratings of later processing.

Finally, the participant was either instructed to attempt to sleep again or told that the experimenter will help them come out bed, depending on how much time remained. After getting out of bed, the participant was sat in front of a computer to fill in some the same questionnaires provided in the home sleep and dream journals.

2.2 Analysis Part I: Testing Targeted Lucidity Reactivation

Statistical analyses of behavioral data and sleep parameters was conducted with JASP (version 0.13.1; JASP Team, 2020). Extraction of sleep parameters from PSG recordings sleep scored in SpiSOP was done using SleepTrip (a branch of FieldTrip with added functionality for sleep analyses; Weber, 2020). Analysis of experimental data were performed only on the first morning nap of participants.

Questionnaire Analysis. To calculate overall lucidity scores, DLQ total scores from each REM condition were calculated by summing ratings (0 to 4) across all items, excluding item number 7 and 12 (Stumbrys, Erlacher & Schredl, 2013). To calculate the vividness of mental imagery in participants, ratings (1 to 10) on all items of the PSI-Q were averaged to calculate a global score (Andrade et al., 2014). Sleep quality was evaluated by deriving component scores, on the basis of which global scores (ranging 0 to 21) according to Buysse et al. (1989). Chronotype and absolute social jet lag for each participant was computed based on MCTQ scores and instructions provided by Roenneberg et al. (2015).

Lucid Dream Induction Rates. A dream was classified as a SVLD if the participant performed the LRLR signal during REM sleep and subsequently reported being lucid and having performed the eye signal during dreaming prior to awakening. All other dreams were classified as non-SVLDs. No statistical testing were performed on SVLD induction rates since no SVLDs were observed in the first nap.

Effects of Cueing and Training Type on Lucidity. To assess the effect of cueing and training type on lucidity, we compare DLQ scores between We compared DLQ scores between experimental conditions using two-way analysis of variance (ANOVA).

Sleep Measures & Group Sleep Comparison. Self-reported bed and rise times the night preceding the experiment were used to calculate sleep duration and morning wake intervals. Nap sleep architecture was compared between experimental groups for minutes in stage N1, N2, N3, and REM sleep using multivariate ANOVA.

2.3 Data Acquisition Part II: EEG Case Study

Participant & Procedure. Following an almost identical procedure and protocol as described in Sec. 2.1 one participant (female, age: 26) came to the lab for a total of 3 experimental morning naps. During the EEG setup of the 2nd and 3rd experimental sessions, it was explained to the participant that if they become lucid and provided the lucidity eye signal in a dream we would attempt to give them math problems via the intercom system every 10–15 seconds and that they should attempt to solve them with eye movements, where the total number of (left/right) saccades should equal the desired response (see Konkoly et al., in preparation for more details). For example, if the participant is provided the math problem $1 + 2$, the correct response 3 is provided by moving their eyes either L-R-L or R-L-R. A more precise saccade training followed, where the participant was provided a few sample math problems during wakefulness to respond to via LRLR-eye movements to control that they understand the instructions. Only math problems where a correct response is an integer between 1 and 6 were administered. The participant further instructed to signal single numbers if they could only partially understand a math problem.

The procedure and timing of cue administration was identical to the pure cueing condition (see Sec. 2.1). Moreover, when a LRLR eye signal could be recognized online by the experiment, math problems we administered with a 10–15 s interval. The experimenter kept administering math problems if no clear eye-signal responses were detected on the EOG traces. The math problems were randomly generated and administered in a random order.

2.4 Analysis Part II

EEG Analysis & Statistics. EEG data from a single subject with 3 episodes of REM-SVLDs and 4 episodes of non-lucid REM sleep were preprocessed and analyzed using FieldTrip toolbox (Oostenveld, Fries, Maris & Schoffelen, 2011).

During preprocessing, EEG data was mastoid referenced and bandpass filtered between 0.3-35 Hz and bandstop filtered between 48-52 Hz and 98-102 Hz for channel inspection. The time course was visually inspected for quality assessment, electrodes with excessive noise, or poor signal quality marked for later spline interpolated. The raw (non-filtered) data were then preprocessed for independent component analysis (ICA) and artifact rejection by applying bandpass filter between 1-45 Hz. The data were visually inspected in 5 s windows and segments either containing artifacts or arousals were discarded. For SVLDs, all segments prior to the first LRLR-signal as well as any time points exceeding 15 seconds from a previous lucidity signal were considered as non-lucid and rejected. ICA was performed and components related to cardiac activity and (rapid) eye movements were removed from the data. A comparable amount of components across nonLDs (M=8.5 SD=1.29) and SVLDs (mean=7 SD=1) were removed from raw data. Alternate ICA cleaning was performed with less components removed, SVLDs (mean=5.33 SD=0.5), nonLDs (mean=6.25 SD=0.5), before applying channel interpolation, bandpass filtering between 0.3-45 Hz and re-referencing to the average of all electrodes.

Total spectral density was estimated between 1–30 Hz using Hanning tapers with a threepoint moving average frequency smoothing. For frequencies 30–45 Hz Slepian multitapers (Thomson, 1982) were used with smoothing of ±4 Hz. For all frequencies, spectral power was estimated from 5 second segments with 50% overlap (Welch's method). Power estimates for canonical frequency bands (delta:1-4 Hz, theta: 4-8 Hz, alpha: 8–12 Hz, beta: 13-30 Hz, (low) gamma: 30–45 Hz) were created by averaging the power across constituent frequencies into their respective bands.

To decompose the power spectra into its oscillatory and fractal (1/f) components, an implementation of the irregular-resampling auto-spectral analysis (IRASA; Wen & Liu, 2016) method was applied in FieldTrip. IRASA was performed on Hanning tapered data between 1–45 Hz with a threepoint moving average spectral smoothing to obtain an estimate of the 1/f contributions to total signal power, which was then subtracted from total power to isolate the oscillatory components. In IRASA analysis, Slepian tapers could not be applied to higher frequencies due to limitations of the analysis method. However, total spectral power and it's topographic distribution was highly similar for Hanning and Slepian tapered data in the 30–45 Hz range.

Statistical analyses using non-parametric permutations of dependent samples T-tests with cluster-based correction for multiple comparisons were performed to test if SVLDs and nonLDs differ in total spectral power, oscillatory and fractal power for each frequency band and to locate the spectral and topographic attributes of the largest difference(s) in power between SVLDs and nonLDs. In statistical analyses, the overlapping 5 second trials from both conditions were considered as individual trials matched between conditions (N=108).

To investigate EEG FC differences between SVLDs and nonLDs for the gamma frequency band, coherence (Shaw, 1981) was computed for all frontal and anterior electrodes extracted from a cluster of electrodes found to be associated with significant difference

in gamma-band activity between conditions. The coherence between this subselection of electrodes and all other channel pair combinations was computed using permutation based statistical testing of independent Z-scores according to methods outlined by Maris, Schoffelen, and Fries (2007) as implemented in FieldTrip. However, due to computational limitations, instead of using cluster-based thresholding for multiple comparison correction, the false discovery rate (FDR) was used.

3 Results

3.1 Part I: Testing Targeted Lucidity Reactivation

From 29 participants, 12 underwent TLR with BS presleep training (7 female, 5 male, mean_{AGE}=22.08, SD_{AGE}=2.75) and 17 S training (14 female, 3 male, mean_{AGE}=24.12, SD_{AGE}=5). There was no significant difference in age between groups, t(27)=1.276, p=.213. On average, participants slept for 5 hours and 50 minutes in the night preceding the experiment and were subsequently awake for a 3 hour and 51 minute interval before lights off in the laboratory (missing data N=12).

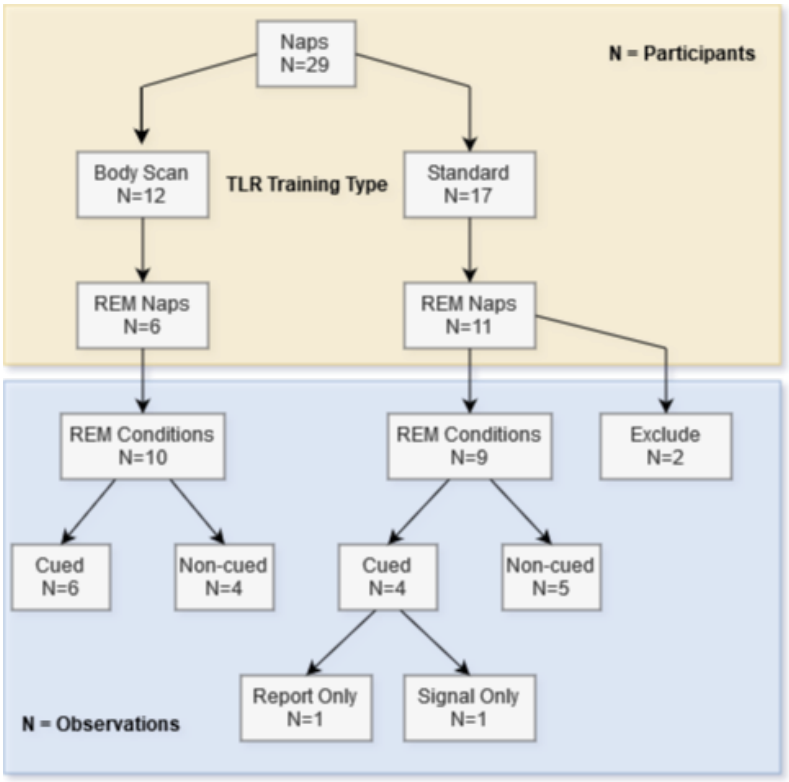

Fig. 3.1. Counts of participants, conditions and outcomes. In the yellow shaded area, numbers in each box represent participants. In the blue shaded area, numbers represent observations.

In total, 17 (6 BS, 11 S) participants obtained REM sleep during their naps. In two cases, no dream report or condition was obtained from the participants REM sleep. The remaining 15 participants contributed a total of 19 REM conditions (10 cued, 9 non-cued); all 19 conditions were non-SVLDs and 0 SVLDs were observed. In the cued conditions, 5 (50%) participants reported perceiving external cues during sleep (incorporation), two (20%) of which reported the external cues being contextualized in an ongoing dream. One participant had a self-reported LD and in 1 participant repeated LRLR signals were observed during REM sleep without a subjectively reported LD. In the non-cued

conditions, no participant reported cue incorporation, contextualization or having had a LD.

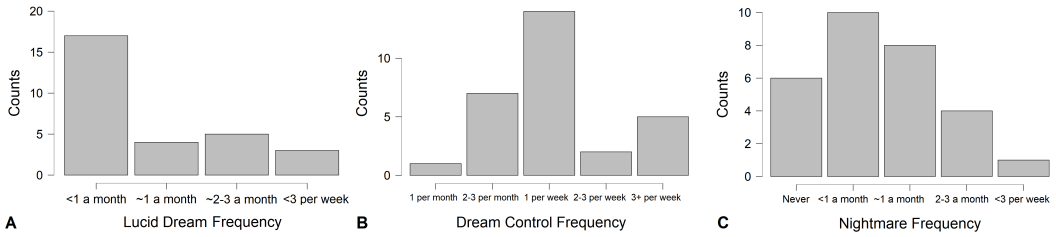

Fig. 3.2. Bar plots display counts of responses on questionnaire measures of LD frequency (How often do you have a lucid dream?), dream control frequency (How often do you take control of your dreams?) and nightmare frequency (How often do you have nightmares?). Only options with only or more responses are displayed.

Group Sleep Comparison. We compared nap sleep architecture between experimental groups using Pillais trace and found a significant effect of TLR training on sleep architecture, V=0.342, F(1, 27)=3.119, p=.034, though a Shapiro-Wilk test suggests the data does not satisfy multivariate normality, W=0.902, p=.011.

Separate univariate ANOVAs revealed that there was no effect of TLR training on minutes in N1 — F(1,27)=0.857, p=.363 — N3 — F(1,27)=0.795, p=.380 — or REM sleep — F(1,27)=0.046, p=.832. However, there was a significant effect of training type on N2 sleep, F(1,27)=5.729, p=.024. Specifically, the BS group had more minutes of N2 stage sleep and than the S group.

Effects of Cueing and Training Type on Lucidity. Cueing did not successfully induce any SVLD, although 1 LD was reported after awakening from cueing. Also during REM sleep cueing, one participant repeated LRLR signals without reporting LD after awakening. Neither SVLD nor report-only LDs were observed in non-cued conditions. Independent two-way ANOVA showed no significant effect of cueing on DLQ scores, F(1,13)= 3.509, p=.084, η^2=.185, 95% CI=[-14.48 1.03] (missing data N=2). No significant effect of training type was found F(1,13)=1.226, p=.288 on DLQ scores, η^2=.065, 95% CI=[-11.73 3.78] (missing data N=2). Nor was there a significant interaction effect between cue condition and training type on DLQ scores found, F(1,13)= 1.226, p=.228, η^2=.065 (missing data N=2). Levenes test indicated equal variances, F(3,13)=0.991, p=.427.

Exploratory Analyses

Effects of Cueing on REM Sleep. To explore if cueing had an effect on REM sleep architecture, we tested if number of REM sleep arousals were greater in cued compared to non-cued conditions and whether cueing had an effect on REM sleep duration. A Shapiro-Wilk test showed the number of arousals were not normally distributed, W_{CUED}=0.581, p<.001, $W_{NON-CUED}$=0.637, p<.001. Therefore, a one-tailed Mann-Whitney U test was performed and showed that cueing did not significantly increase the number of REM

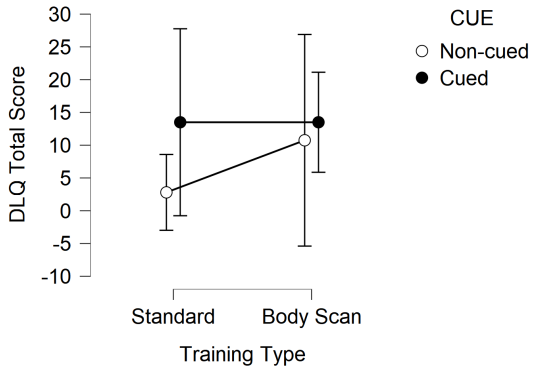

Fig. 3.3. Average total DLQ scores for cued (black circles) and non-cued (white circles) depending on training type condition. Error bars indicate 95% confidence intervals

sleep arousals, U=53, p=.800, 95% CI=[-0.34 0.61]. Similarly, a two-tailed independent samples T-test revealed there was no significant effect of cueing on minutes in REM sleep, t(17)=0.113, p=.911, 95% CI =[-1.76 1.97].

Factors Predicting Lucidity Ratings. To explore factors predicting lucidity ratings, multiple linear regression was used to model DLQ scores with minutes in REM sleep, REM onset latency, number of REM arousals, number of cues administered, sleep onset latency, age of first LD, PSI-Q global score as predictors.

Table 1. Model summary for prediction of lucidity ratings.

Model	R	R^2	Adjusted R^2	RMSE
1	0.749	0.560	-0.209	9.755
2	0.741	0.549	0.008	8.836
3	0.719	0.517	0.115	8.349
4	0.706	0.498	0.211	7.880
5	0.695	0.483	0.290	7.478
6	0.611	0.373	0.234	7.766
7	0.505	0.255	0.181	8.031

Factors Predicting REM Sleep. To explore factors predicting the occurrence of REM sleep in participants during the experimental nap, a multiple logistic regression was calculated with PSQI scores, chronotype (missing data N=9), absolute social jet lag (missing data N=9), lights off (LO) time, sleep onset (SO) latency, age, gender and training type modeled as predictors.

3.2 Part II: EEG Case Study

The participant had three SVLDs and four nonLDs in the sleep laboratory. After rejecting artifacts and arousals from the data, SVLDs had an average length of 3 minutes (SD: 2.88 minutes) while nonLDs had an average length of 2.5 minutes (SD: 0.44 minutes). All SVLDs and two nonLDs occurred during REM sleep cueing. Cues were incorporated

Table 2. Model summary for prediction or REM Sleep.

Model	Deviance	AIC	BIC	df	X^2	p	McFadden R^2	Nagelkerke R^2	Tjur R^2	Cox & Snell R^2
1	12.190	32.190	42.148	10			0.000	0.000	0.596	0.000
2	12.205	30.205	39.167	11	0.015	0.902	-0.001	-0.002	0.595	-7.519e-4
3	12.282	28.282	36.248	12	0.077	0.782	-0.007	-0.010	0.597	-0.005
4	12.609	26.609	33.579	13	0.327	0.568	-0.033	-0.044	0.585	-0.021
5	14.540	26.540	32.515	14	1.931	0.165	-0.162	-0.215	0.546	-0.111
6	16.122	26.122	31.101	15	1.582	0.209	-0.244	-0.322	0.491	-0.178
7	17.233	25.233	29.216	16	1.111	0.292	-0.293	-0.386	0.440	-0.223

and contextualized in the ongoing dream in one nonLD. During two of the recorded SVLDs, the participant was engaged in two-way communication with the experimenters (see Konkoly et al., in preparation for details). In all 3 SVLDs either cues or the experimenters voice was incorporated and contextualized. Example dream reports for SVLDs and nonLDs are provided in Table 3. Epochs of wakefulness with LRLR signal, nonlucid REM and lucid REM with LRLR are shown in Fig. 3.4. Total spectral, fractal and oscillatory power plots for SVLDs and nonLDs aswell as and the difference in spectral power between conditions are shown in Fig. 3.5. The graphs illustrate that for total spectral power, SVLDs and nonLDs differ most strongly in the 8–12 Hz, where SVLDs have higher power. The strongest negative difference is centered around 15 Hz. For both total and fractal power, the negative slope of the power spectra appears to be greater in the lucid condition. Moreover, the power spectra for fractal and total power are highly similar and have a comparable range, suggesting that the contribution of 1/f-activity to the total power estimate is large. This is further corroborated by the fact that the oscillatory power spectra for lucid and nonlucid conditions almost completely overlap, as well as have scale one order of magnitude smaller than total and fractal power. Topographic distributions of power differences for each frequency band are shown in Fig. 3.6.

Permutation-based statistical testing was performed comparing average total, fractal and oscillatory power within alpha, beta, delta, theta and gamma frequency bands respectively between SVLDs and nonLDs with dependent samples T-tests. To threshold results additionally for multiple comparisons across frequency bands, Bonferroni correction was applied.

For total power, results reveal that there are a significant differences in spectral power between the conditions in all frequency bands. Moreover, these differences appear to be driven by differences in power localized to specific electrode clusters for each frequency band. Specifically, in the delta, alpha and, gamma range, two positive clusters were found; one in left frontocentral electrodes and one in the right temporal-occipital (all ps$<$ .01). As can be seen in Fig. 3.6, these positive clusters vary in size across the frequency range but are largely localized to the same electrodes. Two negative clusters were also found in delta, beta and theta bands covering a widespread area of right frontocentral and primarily left-sided occipitoparietal channels (all ps$<$.01).

Significant differences between conditions were also found in all frequency bands for fractal power (all ps$<$.01). Results are visualized in Fig. 3.7 In delta and theta bands, a negative cluster which included all electrodes was found. In beta and alpha bands, a negative cluster was also found in right frontocentral electrodes. In the alpha band, a

Table 3. Example dream reports of SVLD and nonLDs.

Dream Type	Dream Interview
SVLD	*Can you describe what was going through your mind just before I woke you up?* ...Well I had a good dream session holy moly ...I remember that in my dream I was thinking about that I had to remember things... Yes because I heard the sounds and... heard you talk while I was dreaming and at one moment I was in the car and then I noticed a part of the assignment the math sum and after that I had a bit less with the assignments but then I was in a sort of horror story house something where we wanted to escape from and yes after that some more dreams a lot of dreams. *Is there anything else you can remember, any thoughts or feelings, sensations or sensory experiences?* I have had a lot of emotions coming past at the end Ive been seriously scared in the horror but I was also really proud that the math sum worked out that I heard them and in the dream that I was aware that I was dreaming and yes so there are different em emotions came past. *Were you aware that you were dreaming?* Yes (laughs) *Did you hear the (math) assignments?* The equations - yes. *How many assignments do you think that you solved?* Four-five. *Have you solved the assignments with eye-movements?* Yes. *Could you give an example of one of the calculations you solved?* ....Yes just the standard two plus two and ... you had a deduction so I think five minus two. I had also once five plus two and I thought that was very weird I thought how should I seven that takes such a long time but well *Did you hear or see the signals? The flickering light or beeping sounds.* The beeping sounds I heard them. *What did they look or sound like in your dream?* Yes. I heard it. It was also a bit out of context, so I did have to try hard to keep on dreaming but there was by the way something with sitting in the car that... that and I did the equations but there was something else so maybe that I thought once again like hee I see light but I dont know there was something else with that car.
nonLD	*Can you describe what was going through your mind just before I woke you up?* ... I had the feeling that I ...Yes just a very free feeling *Is there anything else you can remember, any thoughts or feelings, sensations or sensory experiences?* ...No I was just seeing a lot. I was not walking or doing anything. I was just watching a lot and letting that come over me everything I was seeing. *Were you aware that you were dreaming?* No Did you hear or see the signals? The flickering light or beeping sounds? No *Did you perform any eye-signals?* At the moment I was dreaming? No.

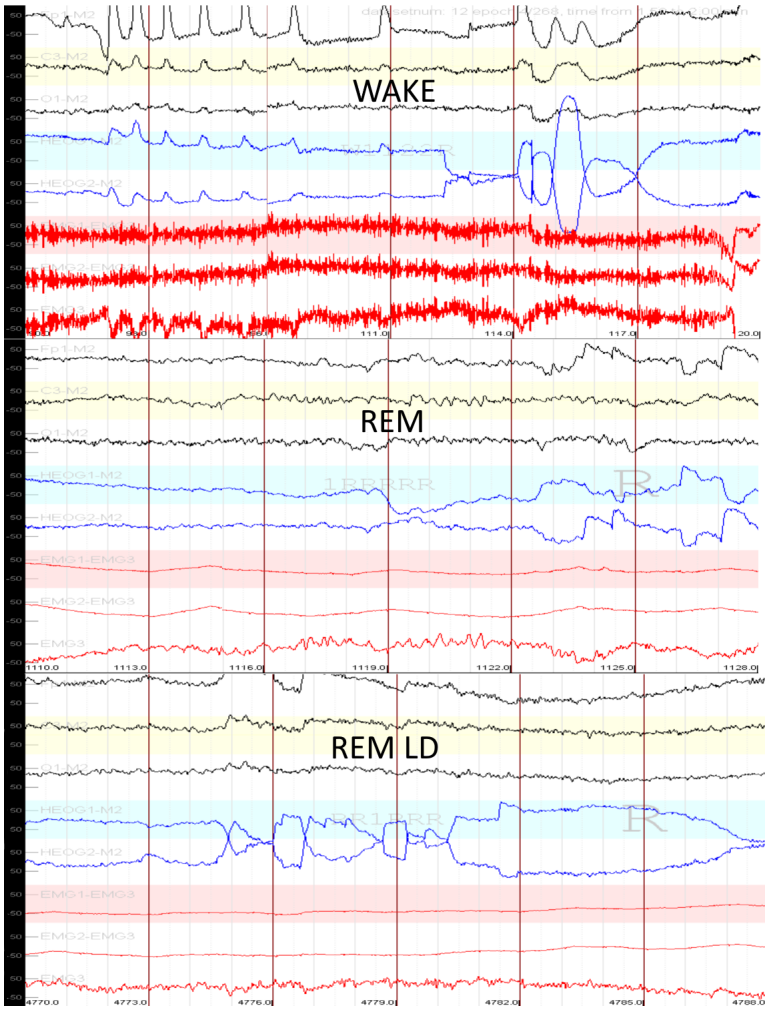

Fig. 3.4. Polysomnograms showing wake with LRLR eye movements (top), REM sleep (middle) and lucid REM sleep with LRLR eye movements (bottom). Black lines show EEG traces for Fp1, C3 and O1 respectively. Blue lines show horizontal EOG traces and red show EMG1, EMG2 and EMG3 respectively. For all traces range displayed is -50–50μV.

negative cluster was additionally found in occipial and parietal channels primarily on the left side. Furthermore, in positive clusters were found in the alpha, beta and gamma band in left frontocentral and right temporoparietal channels.

For oscillatory power, significant differences were only found in the theta, alpha and beta bands (all ps<.05) with positive clusters localized to central electrodes in alpha and beta band, as well as a negative beta band cluster in right central positions (see Fig. 3.8). However, these significant differences did not survive Bonferroni correction.

Coherence was calculated between anterior and frontal electrodes in the gamma-band cluster (AF3, AF7, AFF1h, AFF2h, AFF5h, AFp2, AFpz, F1, F3, F5, F7) and all other electrodes in pairs. Calculated coherences were stastically tested for the difference between conditions in the 30-45 Hz range using a non-parametric permutation test of independent samples Z-scores (two-tailed) thresholded for multiple comparisons with

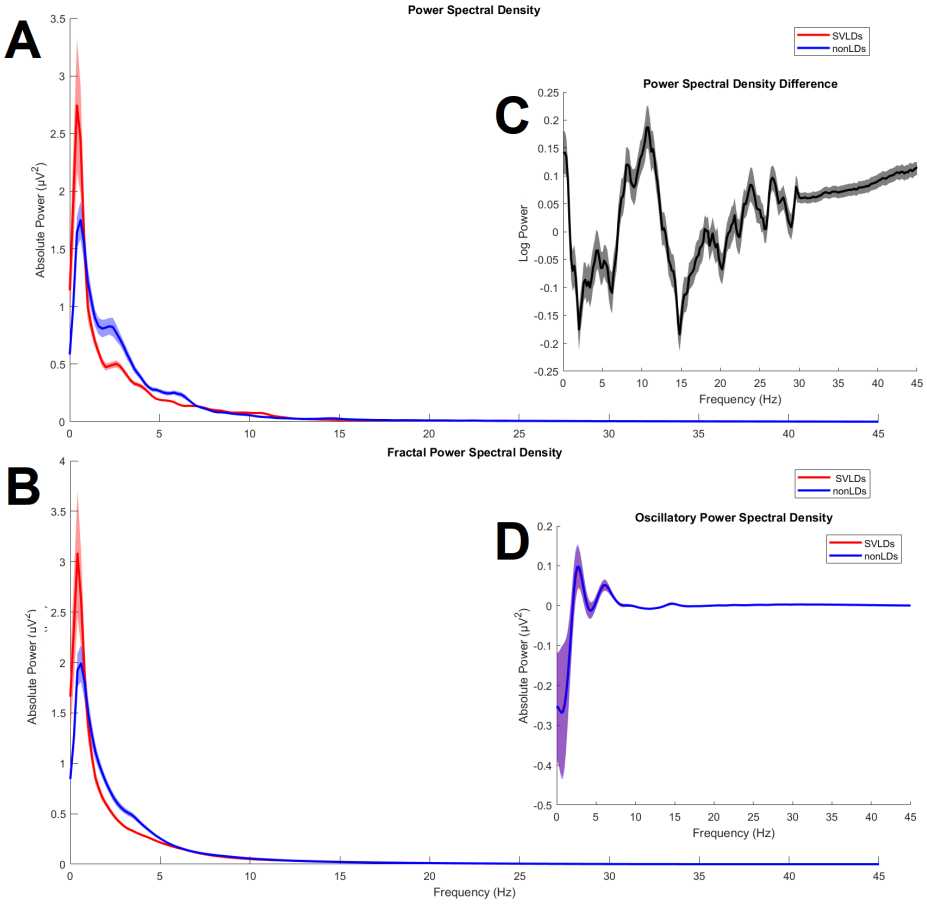

Fig. 3.5. A,B,D: Total, fractal and oscillatory power (y-axis) per frequency (x-axis) averaged across all electrodes and 5 second trials for SVLDs (red lines) and nonLDs (blue lines). Colored patches plot SEMs computed across trials for each condition. C: Black line traces the average difference in log10-transformed power (y-axis) between SVLD and nonLD trials (SVLDs−nonLDs) per frequency (x-axis). Black patches plot the SEM of the difference computed across trials.

FDR. Results revealed that there was no significant coherence between any channel pairs, suggesting that there are no changes in high frequency functional coupling associated with LDs.

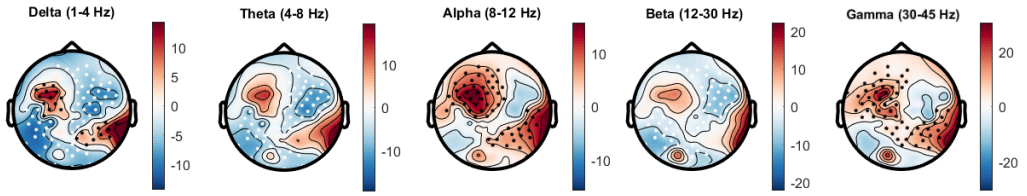

Fig. 3.6. Topographic plot of total power differences showing positive (black dots) and negative (white dots) electrode clusters at p<0.01. Color map ranges from negative (blue) to positive (red) T-values and are adjusted to the maximum absolute T-statistic for each frequency band.

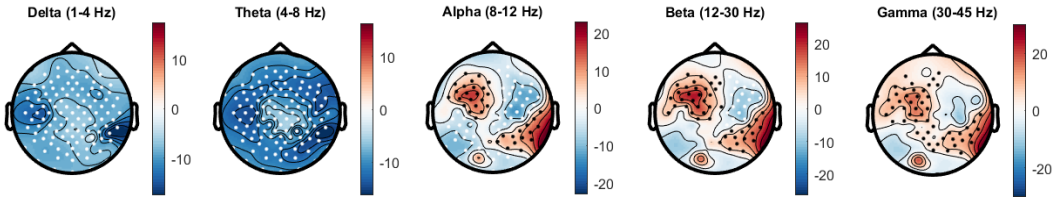

Fig. 3.7. Topographic plot of fractal power differences between conditions showing positive (black dots) and negative (white dots) electrode clusters at p<.01. Color map ranges from negative (blue) to positive (red) T-values and are adjusted to the maximum absolute T-statistic for each frequency band.

4 Discussion

4.1 Part I: Testing Targeted Lucidity Reactivation

We tested an experimental procedure for LD induction combining sensory cueing (visual and auditory) with presleep cognitive training in morning naps. Our results suggest this procedure is highly ineffective, with no SVLDs achieved in 29 participants, 19 REM sleep opportunities, and 10 induction attempts (cued conditions). This conclusion is further corroborated by the fact that we see no significant effect of cueing on subjective measures of dream lucidity, though we do observe a rather large effect size η^2=.185. We furthermore did not observe any significant difference in effect on dream lucidity between our two TLR training procedures, of which the BS condition was previously untested and unique to the present study. This suggests both training procedures are similarly ineffective. These findings were unexpected and stand in stark contrast to those made by Carr et al. (2020) with an almost identical procedure, who observed 16 SVLDs in 41 participants, 40 REM sleep opportunities, and 28 induction attempts. While we adopted the procedure developed by Carr and colleagues with slight modifications, there are a few salient differences between our methods that could potentially explain the disagreement in results.

Most notably, we used different devices for PSG and EEG measurement; Carr et al. (2020) conducted recordings with either an 8 electrode system (Traumschreiber; Appel, 2018) or 18 + 8 system (Trackit 18/8 system), whereas a PSG/EEG system with 120 EEG and 8 external electrodes was applied in the present study. Though using a high-density recording system enabled us to record potential neural signatures of LDs and other sleep-related phenomena with high fidelity and spatial specificity, a significant downside was the drawn-out EEG setup procedure. It took on average between 2 and 2.5 hours with two to three experimenters to apply the electrodes and ensure proper signal quality. We

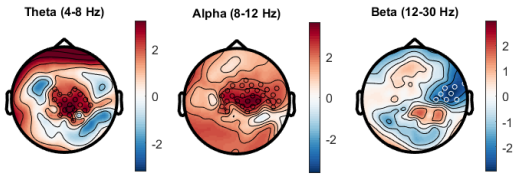

Fig. 3.8. Topographic plot of oscillatory power differences showing positive (black circles) and negative (white circles) electrode clusters at p<.05 (uncorrected). Color map ranges from negative (blue) to positive (red) T-values and are adjusted to the maximum absolute T-statistic for each frequency band.

believe that this process may have caused discomfort or unease to participants, rendering them unable to sleep well in the laboratory. Furthermore — while our participants' average sleep length (6 hours) in the night before the experiment was within the range of what has been reported to be effective in previous studies using the WBTB method (Erlacher & Stumbrys, 2020) — the average length of wake intervals for our participants (4 hours) was far greater than the 60-minute interval considered to be optimal for the method (LaBerge, Philips, & Levitan, 1994; Strumbrys & Erlacher, 2014), largely due to the long EEG application procedure. Likely, the suboptimal length of the wake interval contributed negatively to our results, however, it remains unclear to what extent given that we lack data on the wake interval for 12 out of 29 participants. Also, Carr et al. (2020) observed no difference in induction rates for participants tested at 07:30 and 11:00, despite significant group differences in previous night sleep and wake intervals.

Another important addition in our study design is that our participants were screened and conducted a week of sleep and dream journaling before the experimental session, rather than coming directly to the lab for the experimental session after being recruited (Carr et al., 2020). The intention behind this change was to make sure participants were suitable subjects and to collect data on potentially important controlling variables via questionnaires and journals. Furthermore, it has often suggested that dream journaling is an effective tool for increasing LD frequency due to the improvements in dream recall frequency (DRF) that it produces (Stumbrys & Erlacher, 2014; Schredl, 2002). Improving dream recall in our participants would presumably also make them better recollect dreams experienced during the experimental nap and provide more reliable dream reports. However, its possible that these additional steps counteracted our study aims and led participants to build expectations about the experimental nap and feeling pressured to LD, making them unlikely to do so.

Differences in the studied samples also exist between the two studies. We selected a sample of healthy participants with typical sleep, at least one previous experience of lucid dreaming, and a high DRF, due to previous observations that dream recall and LD frequency correlate strongly (Schredl & Erlacher, 2011). Carr and colleagues, on the other hand, report no strong restrictions on inclusion, but a preference for individuals with weekly bad dreams. In their exploratory analyses, Carr et al. (2020) furthermore find that participants' self-reported nightmare distress (non-significantly, but positively) increased the likelihood of having SVLD, having noticed cues, as well as subjective lucidity ratings. In our sample, only one participant reported a nightmare frequency higher than at least one per week.

Frequent nightmares have been associated with wake-like electrophysiological features of REM sleep (Simor, Horváth, Ujma, Gombos & Bódisz), suggesting that atypical increases in environmental vigilance related to those EEG features during REM sleep (Cantero, Atienza, & Salas, 2002) might facilitate successful SVLD induction via cueing. Given the limited statistical impact of nightmare distress Carr et al (2020) observe on their results, the contribution nightmare-related differences between our samples can only explain the different outcomes of our studies to some extent.

Nevertheless, it remains to be tested whether REM sleep sensory cues can be used to effectively induce lucidity. Our results suggest that cueing is not effective, but that it also does not disrupt REM sleep. Two other recent studies using REM sleep cueing with auditory (Schmid & Erlacher, 2020) and olfactory stimulation (Erlacher, Schmid, Schuler & Rasch, 2020) did not find positive effects on LD induction rates either, even when cues were trained to be associated with metacognitive processes, similar to the present study. Recent research on the mechanisms of sensory disconnection during REM sleep has also provided results suggesting informative sensory cues are selectively suppressed during phasic REM sleep (Koroma et al., 2020). Specifically, it was found that neural processing of (informative contrasted to pseudo-) speech was presented during phasic REM sleep. One proposed theory about the mechanisms underlying such effect suggests that during REM eye-movements, salient environmental stimuli may be selectively suppressed to prevent interference with internal activity. Whether this is the case or not in an open question, however, this line of research provides a possible explanation for why cueing was not effective in our study. Crucially, Koroma et al's (2020) study implies that presleep training with sensory cues, as in the TLR procedure, could have deleterious effects on the processing of the same stimuli presented during sleep.

Due to the absence of a statistically significant effect of cueing on either SVLDs or subjective ratings of dream lucidity, we explored if regression models based REM sleep, REM onset latency, number of REM arousals, number of cues administered, sleep onset latency, and PSI-Q global score could predict DLQ scores. We find that all our models performed poorly, suggesting that none of these variables are predictive of subjective dream lucidity. An accumulating body of research suggests that transitions from wakefulness and REM sleep, and their temporal proximity, are important factors influencing dream lucidity. For example, lucid dream frequency has been associated with self-reported nocturnal awakenings (Gott et al., 2020) and alarm clock snooze button use (Smith & Blagrove). Moreover, physiologically verified wake-REM transitions have been associated with factors of subjective lucidity (Gott et al., 2020). It has also been suggested on the basis of lucid dreaming research on narcoleptics, that quick transitions from wakefulness to REM sleep are predictive of lucidity. In line with such results, it would have been expected that REM onset latency and number of REM arousals have predictive power in our regression model.

Since a significant proportion of our sample (41%) did not enter REM sleep during the 2 to 2.5 hours of sleep opportunity during the experimental nap, we explored whether subjective sleep quality, chronotype, social jet lag, LO time, SO latency, age, gender, recurring nightmare percentages or training type could predict the occurrence of REM sleep using logistic multiple regression models. This analysis showed that these variables are not predictive of REM sleep, although the SO latency had the highest (though statistically insignificant, p=.072) predictive power. Its expected that the timing of SO is

predictive of REM sleep, since REM sleep typically occurs within 70 to 90 minutes after sleep onset, at least during nocturnal sleep (McCarley, 2007). Therefore, the quicker a participant fell asleep, the more likely they were to have sufficient time in sleep to enter REM sleep. Nevertheless, the overall result of the analysis suggests that these factors are not predictive of REM sleep. We speculate that the length of the wake interval, EEG setup as well as participants' own or perceptions about the researchers' expectations on them to sleep, could have impacted the occurrence of REM sleep.

Limitations & Future Directions This study suggests previous indications that the TLR-protocol developed by Carr et al. (2020) is highly effective for LD induction are overstated, since our results show a null-effect of the procedure on both LD incidence and dream lucidity, both with and without cue administration. However, the width of the 95% CIs of the mean differences between experimental groups with respect to lucidity scores (Sec. 3.1) suggests our study lacks sufficient power to confidently estimate the true effect of our conditions on DLQ scores (Colegrave & Ruxton, 2002).

Additionally, we were not able to include several self-reported measures of sleep and dreaming, such as DRF, nightmare frequency, and LD frequency, in our regression models due to incompatibility of ordinal predictors with classical linear and logistic regression models, unless specific preprocessing steps are applied (Helwig, 2017). Crucially, these exploratory analyses we are unable to establish which factors might have contributed to why our results are so drastically different from what we expected. Although we have made a few qualified guesses as to what underlies the contrasting results between this study and Carr et al. (2020), a more systematic comparison between our studies with regards to the samples and procedures would be able to better ascertain factors driving the different outcomes. In fact, given that finding a reliable LD induction method is an important step for progress in the field which many studies focus on with highly variable outcomes (Appel et al., 2020; Erlacher & Stumbrys, 2020; Erlacher, Schuler, & Rasch; Schmid & Erlacher, 2020) a meta-analysis of methodological and demographic differences between studies could provide necessary insights into factors mediating LD induction. Similarly, given that several studies have recently explored if sensory cues of various modalities can induce LDs, a meta-analysis or multicenter study could better elucidate the effects of REM sleep cueing on lucidity.

4.2 Part II: EEG case study

Previously EEG studies have produced largely disagreeing results as to the spatial and spectral characteristics of REM sleep lucid dreaming (Baird, Mota-Rolim, & Dresler, 2019). These experiments have been limited in statistical power primarily by small sample sizes, which is also a central limitation in the present study. On the other hand, this thesis is the first study to present EEG results obtained with a high spatial density of 112 electrodes, a number far greater than the highest number of electrodes (19; Voss et al., 2009) used in previous publications on lucid dreaming known to the authors.

Statistical analyses conducted using non-parametric permutation testing and spatial clustering algorithms suggest that lucid dreaming is associated with spectral changes in the five canonical frequency bands. In particular, we find that these significant differences have specific spatial attributes across the frequency range. In line with our apriori hypothesis, we find that there's a significant modulation in gamma activity during REM sleep

lucid dreaming that is large in frontal regions. However, these results are not particularly convincing given that the cluster observed in the gamma range has a large spatial extent, spanning from right temporal to left central and frontal electrodes with focal points that are centered posterior to frontal and anterior sites. What's more, a stronger modulation of activity in the same frontal electrodes that are implicated in the gamma cluster was found in the alpha band. Extraction of oscillatory power components and statistical testing of these furthermore do not indicate any increases in frontal gamma activity during SVLDs. In the context of this additional evidence, we, therefore, don't believe that our results provide sufficient grounds to conclude that lucid dreaming is associated with local increases in anterior-frontal gamma-band activity. On the basis of fMRI research (Baird et al., 2018; Dresler et al., 2012), we expected that EEG FC between anterior-frontal, parietal and temporal channels would be higher in LDs. Our analysis, which used coherence as a metric for functional coupling, did not reveal any differences in connectivity between lucid and nonlucid conditions. This result contrasts those of previous studies, where lucidity has been associated with decreased delta and gamma coherence (Dodet et al., 2015) and broadband increases in coherence (Voss et al., 2009).

Our results indicate that the largest difference in total spectral activity between lucid and nonlucid conditions occurs in the alpha frequency range (see figure 3.5) – with a peak at 10.6 Hz — and that this difference is statistically significant. Additionally, cluster-based correction suggests that lucidity-related alpha band increases occur in the right temporoparietal and occipital region, with a maximum t-statistic at TP8, and left frontal region, with a maximum t-statistic at FC3. Analysis of oscillatory power components also suggested that lucid dreaming sees an increase in alpha activity that is instead rather localized to a central-parietal area in the right hemisphere.

Alpha activity in posterior and central regions has been linked to lucid dreaming in a series of studies, though these results proved non-replicable by the same researchers later on (Ogilvie, Hunt, Sawiecki & McGowan, 1978; Ogilvie, Hunt, Tyson,Lucescu, & Jeakins, 1982; Tyson, Ogilvie & Hunt, 1984; Ogilvie, Vieira, & Small, but see Baird, Mota-Rolim, & Dresler for a review). Alpha wave oscillations are a fundamental electrophysiological characteristic of spontaneous human EEG that are well known to occur also during REM sleep (Cantero, Atienza, & Salas, 2002). Evidence suggests that REM sleep alpha has higher spectral power stemming from parietal associative areas and regions involved in auditory processing during tonic periods (Simor et al., 2013) and is associated with increased environmental vigilance (Simor, Wijk, Nobili, & Peigneux, 2020). Crucially, during phasic periods of REM sleep, a state that is strongly linked to dreaming, alpha activity appears to be suppressed over sensorimotor areas as well as occipital cortices (Cantero, Atienza, & Salas, 2000), which is suggested to index an disinhibition of activity in these regions that could be correlated with the onset of virtual sensory experience (Palva & Palva, 2007). To the background of such research, the results observed in this study are interesting in that they suggest that lucid dreaming is associated with an increase in environmental alertness.

Given the circumstances under which our sample of LDs was observed, this explanation appears plausible. In all 3 SVLDs, our participant is engaged in informational exchange with the external environment that is uncharacteristic of typical REM sleep, both via detection of sensory stimuli administered by the researchers and by giving a response through eye movements. In contrast, the external cues were only consciously

perceived in one nonLD. As such, it's likely that the observed difference in frontocentral alpha-band activity between our conditions reflects neurocognitive processing underlying a heightened state of vigilance and sensory processing during REM sleep. Though we cannot with confidence discern whether this is an effect of lucidity or confounding experimental circumstances.

Increased alpha power in right-lateralized parietal regions has previously been associated with increased top-down attentional mechanisms during wakefulness (Benedek, Schickel, Jauk, Fink, & Neubauer, 2014). If lateral parietal alpha plays a similar functional role in REM sleep cognition, the activity we observe in this area could reflect either increased attentional engagement with the internal sensory experience in lucid dreaming or also reflect top-down attentional recruitment related to eye-movement execution.

Using the IRASA method in spectral analysis (Wen & Liu, 2016), we were able to separately analyze fractal and oscillatory contributions to the total power spectrum. Visual comparison of three different power spectra resulting from this analysis (Fig. 3.5) shows a dominant contribution of the 1/f power components to the total power spectra, which is expected. Interestingly, however, the fractal component of power differs between the who conditions and most prominently so in sub-10 Hz frequencies, where nonLDs appear to have significantly stronger power across the entire scalp for delta and theta frequencies. In line with our interpretation of the increased alpha activity observed in the SVLD condition as reflecting a state of heightened vigilance, recent research has identified a greater slope of 1/f-power as a marker of arousal (Lendner et al., 2020). In fact, Lendner et al. (2020) propose that the steepness of the 1/f-slope can reliably delineate wakefulness from various stages of sleep. Note, however, that these results only link the 1/f-slope to arousal *between* states, since within-state differences in arousal are not addressed. Nevertheless, the authors (Lendner et al., 2020) hypothesize that 1/f dynamics may reflect cortical inhibition, where increased inhibition is related to a lower spectral slope and arousal. While we have not quantified the 1/f-slope directly in this study, our spectral plots indicate a higher slope for SVLDs, which are furthermore associated with attenuated fractal delta and theta activity. Given the circumstances under which these SVLDs were observed, it seems likely that these neural measures are linked to heightened arousal in our subject.

Overall, a comparison of spatio-spectral results for fractal and total power implies that the topographic characteristics of frequency band-specific spectral differences can be largely explained by fractal power contributions. However, since mechanisms and functional associations to differences in fractal power have not been well-researched, it is difficult to interpret these results.

Limitations & Future Directions. Due to the rarity of lucid dreaming, we were only able to obtain 3 REM sleep SVLDs from one subject in this experiment, causing us to only be able to report a single-case study. Apart from the limited sample size, some additional factors constrain our study. Our sample of LDs was collected concurrent with sensory cue administration, which given their subjectively experienced salience likely produced neural responses that contaminated our data. Given also that objective verification of lucidity required the participant to repeatedly signal lucidity, the neural processes underlying the generation of these movements are likely reflected in the EEG measurement. Additionally, during two ongoing REM SVLDs, the participant was able

to hear and respond to administered math problems, which as we have suggested, might have been reflected in neural activity atypical of REM sleep that we cannot separate from potential correlates of lucidity. In light of these limitations, no strong conclusions can be drawn from our study. Nevertheless, our results tentatively suggest that REM lucidity is associated with increased neural indices of environmental vigilance.

Given that previous results have indicated that REM SVLDs more closely resemble wakefulness (Voss et al., 2009), future studies should investigate the relationship between arousal and LDs more closely. Additionally, investigation of neural dynamics around the onset of LDs could provide important clues as to the difference between nonlucid and lucid dreaming. To aid inferences as to the biological mechanisms underlying lucidity-related neural measures, additional EEG studies should separate fractal and oscillatory power contributions as has been done in the present study.

However, given that LDs are so rarely observed in the laboratory, a crucial hurdle is to find a reliable induction method. While cognitive, behavioral (see Sec. 1.2), and brain stimulation (Blanchette-Carriére et al., 2020; Stumbrys, Erlacher, & Schredl, 2013; Voss et al., 2014) induction methods have been tried extensively without consistent success pharmacological induction using galantamine has been less explored despite promising early indications (LaBerge, LaMarca, & Baird, 2018).

5 Conclusion

Our findings suggest that metacognitive reactivation in REM sleep with sensory cues is not as effective for LD induction as previously suggested. Though sensory cueing might have an effect on lucidity, the present study was unable to detect an significant effect.

This study is also the first to report neural correlates of lucid dreaming obtained with high-density EEG, though from one participant only. Our EEG analysis, contrasting three SVLDs and four nonLDs, suggests that the neural correlates of lucidity overlap with indices of arousal, though these results are highly tentatively barring future replications. Larger sample sizes are needed for more reliable inferences about the neural mechanisms of lucid dreaming. However, given the apparent difficulties in observing LDs in the laboratory novel approaches might be necessary.

6 Acknowledgements

The work presented in this thesis is the product of contributions from a large number of people.

To begin with, I would like to thank my supervisor, Martin Dresler, for giving me the unique opportunity to study lucid dreaming and for the generous support and feedback throughout my thesis internship. Huge thanks also to my on-site supervisor, Anastasia Mangiaruga, who has been the lead figure in this research project and spent countless hours training me in how to make EEG measurement, sleep score, giving words of encouragement, and advice.

I'm sincerely grateful to Hong-Viet V. Ngo, not only for the many hours he has spent teaching and helping me with EEG analysis in FieldTrip, but also for being a great mentor and friend. Aside from Hongi, Frederik Weber has also given me a lot of important guidance in EEG analysis. Not only that, but as the developer behind SpiSOP and SleepTrip, access to Freddy's programming skills and EEG know-how have been invaluable. During my internship, Amber van der Stam has been my main accomplice. Without her help with running the study, sleep scoring, and translating dream reports, this thesis would not have been possible. Before us, Jarrod Gott, Famke Roest, and Bart Stuart also made significant contributions to the project. I want to thank Çağatay Demirel, Mahdad Jafarzadeh Esfahani, Pedro Reis Oliveira, Mariana de Carvalho Pereira, Emma Peters, and Linda Schlüter for sacrificing several hours of morning REM sleep to assist us with EEG recordings. I'd also like to thank Tim van Mourik for creating and sharing this Donders Institute-themed LaTeXstylesheet, without which my thesis would've been far less appealing to the eye.

Finally, I want to thank my girlfriend and partner, Madelene Holm, for keeping me sane throughout this pandemic, all the loving support, and putting a smile on my face in challenging times.

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Appendix

Appendix

Table 4. Coefficient table for linear regression models of DLQ scores

Model		Unstandardized	Standard Error	Standardized	t	p	95% CI Lower	95% CI Upper	Collinearity Statistics Tolerance	Collinearity Statistics VIF
1	(Intercept)	-61.740	44.893		-1.375	0.241	-186.383	62.903		
	Sleep Onset Latency	0.333	0.512	0.378	0.650	0.551	-1.089	1.755	0.326	3.072
	REM Onset Latency	0.044	0.137	0.147	0.319	0.765	-0.338	0.425	0.518	1.929
	# REM Arousals	5.329	4.511	0.580	1.182	0.303	-7.194	17.853	0.456	2.191
	PSIQ Global Score	6.355	3.865	0.815	1.644	0.175	-4.376	17.085	0.447	2.238
	REM Sleep Minutes	0.907	1.836	0.204	0.494	0.647	-4.191	6.005	0.645	1.549
	Age of first LD	0.604	0.916	0.276	0.659	0.546	-1.940	3.148	0.626	1.596
	Recurring Nightmare %	-0.091	0.100	-0.353	-0.915	0.412	-0.367	0.185	0.737	1.357
2	(Intercept)	-53.061	32.366		-1.639	0.162	-136.260	30.139		
	Sleep Onset Latency	0.325	0.463	0.369	0.702	0.514	-0.866	1.517	0.326	3.066
	# REM Arousals	5.013	3.986	0.545	1.258	0.264	-5.233	15.259	0.479	2.086
	PSIQ Global Score	5.587	2.741	0.717	2.038	0.097	-1.459	12.633	0.729	1.372
	REM Sleep Minutes	0.985	1.649	0.221	0.597	0.576	-3.253	5.223	0.657	1.522
	Age of first LD	0.533	0.805	0.244	0.662	0.537	-1.536	2.603	0.666	1.502
	Recurring Nightmare %	-0.080	0.084	-0.309	-0.946	0.387	-0.296	0.137	0.847	1.181
3	(Intercept)	-43.969	26.988		-1.629	0.154	-110.007	22.069		
	Sleep Onset Latency	0.181	0.374	0.206	0.485	0.645	-0.733	1.096	0.448	2.234
	# REM Arousals	4.645	3.721	0.505	1.248	0.258	-4.460	13.749	0.491	2.036
	PSIQ Global Score	5.471	2.584	0.702	2.118	0.079	-0.850	11.793	0.732	1.366
	Age of first LD	0.448	0.749	0.205	0.598	0.571	-1.384	2.280	0.687	1.455
	Recurring Nightmare %	-0.090	0.078	-0.348	-1.150	0.294	-0.280	0.101	0.882	1.134
4	(Intercept)	-34.880	18.325		-1.903	0.099	-78.211	8.452		
	# REM Arousals	3.404	2.550	0.370	1.335	0.224	-2.626	9.435	0.931	1.074
	PSIQ Global Score	5.150	2.357	0.661	2.185	0.065	-0.423	10.723	0.784	1.276
	Age of first LD	0.287	0.633	0.131	0.453	0.664	-1.210	1.784	0.856	1.168
	Recurring Nightmare %	-0.094	0.073	-0.366	-1.292	0.237	-0.267	0.078	0.895	1.117
5	(Intercept)	-33.182	17.023		-1.949	0.087	-72.437	6.074		
	# REM Arousals	3.360	2.418	0.365	1.389	0.202	-2.217	8.936	0.933	1.072
	PSIQ Global Score	5.459	2.141	0.701	2.550	0.034	0.522	10.396	0.856	1.169
	Recurring Nightmare %	-0.089	0.068	-0.347	-1.307	0.228	-0.247	0.068	0.914	1.094
6	(Intercept)	-29.515	17.438		-1.693	0.125	-68.962	9.932		
	# REM Arousals	3.267	2.511	0.355	1.301	0.226	-2.413	8.946	0.934	1.071
	PSIQ Global Score	4.650	2.128	0.597	2.185	0.057	-0.164	9.465	0.934	1.071
7	(Intercept)	-19.997	16.368		-1.222	0.250	-56.468	16.475		
	PSIQ Global Score	3.937	2.127	0.505	1.851	0.094	-0.802	8.676	1.000	1.000

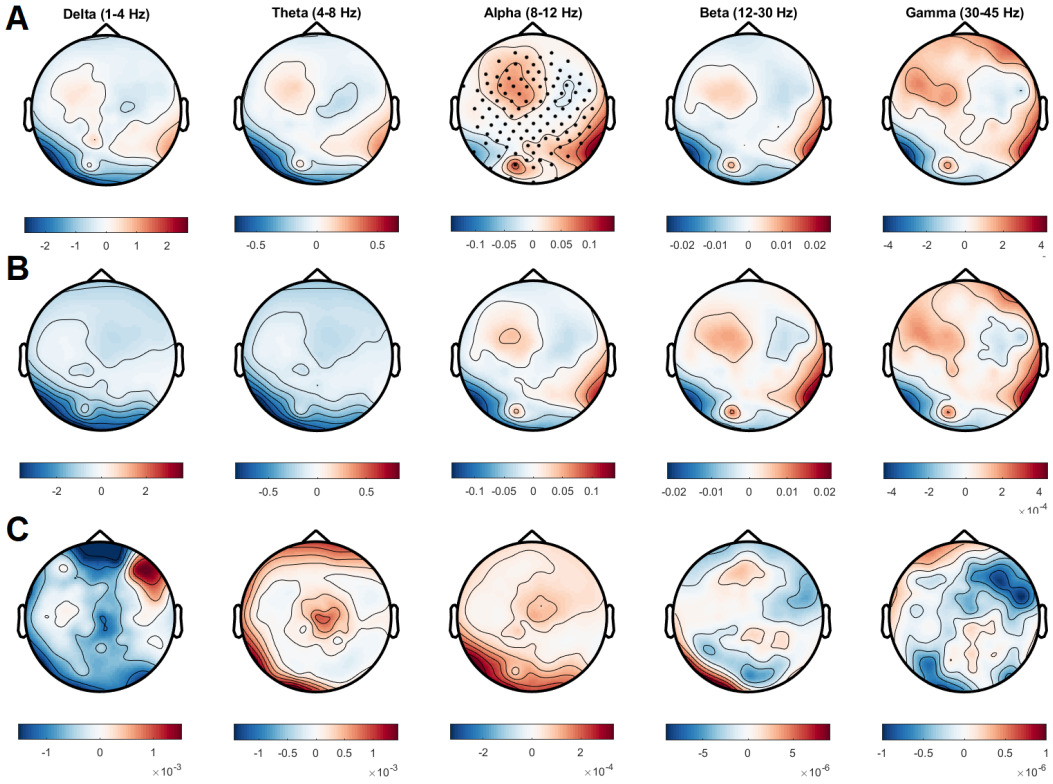

Fig. 6.1. Topographic plots showing the difference in total spectral (A), fractal (B) and, oscillatory (C) power (μV^2) between SVLDs and nonLDs (SVLDs−nonLDs) for each frequency band. Black dots in A indicate electrode positions. Color maps ranges from negative (blue) to (red) and are adjusted for each plot.

Table 5. Coefficients table for logistic regression of REM sleep

Model	Parameter	Estimate	Standard Error	z	Wald Test			95% Confidence interval	
					Wald Statistic	df	p	Lower bound	Upper bound
1	(Intercept)	6.419	16.325	0.393	0.155	1	0.694	-25.578	38.416
	Age	-0.829	0.799	-1.037	1.076	1	0.300	-2.395	0.737
	Training Type	-3.778	3.102	-1.218	1.483	1	0.223	-9.859	2.302
	Abs. SJL	0.323	0.340	0.950	0.903	1	0.342	-0.343	0.989
	Gender	-0.393	3.147	-0.125	0.016	1	0.901	-6.562	5.775
	Chronotype	0.585	2.180	0.268	0.072	1	0.789	-3.688	4.857
	PSQI Global	-1.177	1.321	-0.891	0.794	1	0.373	-3.765	1.411
	PSIQ Global Average	3.204	2.190	1.463	2.140	1	0.144	-1.089	7.496
	Recurring Nightmare %	0.033	0.053	0.615	0.378	1	0.538	-0.071	0.137
	SO Latency	-0.377	0.295	-1.276	1.628	1	0.202	-0.956	0.202
2	(Intercept)	6.118	16.212	0.377	0.142	1	0.706	-25.657	37.893
	Age	-0.841	0.841	-1.000	1.000	1	0.317	-2.490	0.808
	Training Type	-3.887	3.157	-1.231	1.515	1	0.218	-10.075	2.302
	Abs. SJL	0.346	0.320	1.078	1.163	1	0.281	-0.283	0.974
	Chornotype	0.610	2.258	0.270	0.073	1	0.787	-3.814	5.035
	PSQI Global	-1.267	1.235	-1.026	1.053	1	0.305	-3.687	1.153
	PSIQ Global Average	3.255	2.222	1.465	2.147	1	0.143	-1.099	7.609
	Recurring Nightmare %	0.031	0.052	0.599	0.359	1	0.549	-0.071	0.133
	SO Latency	-0.385	0.303	-1.268	1.608	1	0.205	-0.979	0.210
3	(Intercept)	4.731	15.182	0.312	0.097	1	0.755	-25.025	34.486
	Age	-0.678	0.519	-1.306	1.707	1	0.191	-1.696	0.339
	Training Type	-3.335	2.175	-1.533	2.350	1	0.125	-7.598	0.929
	Abs. SJL	0.285	0.198	1.439	2.070	1	0.150	-0.103	0.674
	PSQI Global	-1.049	0.839	-1.249	1.560	1	0.212	-2.694	0.597
	PSIQ Global Average	3.003	1.935	1.552	2.409	1	0.121	-0.789	6.795
	Recurring Nightmare %	0.026	0.046	0.563	0.317	1	0.573	-0.064	0.115
	SO Latency	-0.335	0.216	-1.548	2.398	1	0.122	-0.759	0.089
4	(Intercept)	-2.454	9.494	-0.258	0.067	1	0.796	-21.061	16.153
	Age	-0.472	0.302	-1.561	2.436	1	0.119	-1.064	0.121
	Training Type	-3.468	2.300	-1.508	2.274	1	0.132	-7.977	1.040
	Abs. SJL	0.256	0.173	1.481	2.193	1	0.139	-0.083	0.596
	PSQI Global	-0.899	0.726	-1.237	1.531	1	0.216	-2.322	0.525
	PSIQ Global Average	3.386	2.060	1.644	2.703	1	0.100	-0.651	7.423
	SO Latency	-0.350	0.219	-1.596	2.546	1	0.111	-0.779	0.080
5	(Intercept)	-1.229	8.279	-0.148	0.022	1	0.882	-17.455	14.998
	Age	-0.303	0.222	-1.366	1.865	1	0.172	-0.739	0.132
	Training Type	-1.863	1.567	-1.189	1.413	1	0.235	-4.935	1.209
	Abs. SJL	0.102	0.087	1.176	1.382	1	0.240	-0.068	0.272
	PSIQ Global Average	1.757	1.045	1.682	2.829	1	0.093	-0.290	3.805
	SO Latency	-0.178	0.120	-1.480	2.191	1	0.139	-0.413	0.058
6	(Intercept)	2.185	7.165	0.305	0.093	1	0.760	-11.858	16.228
	Age	-0.360	0.228	-1.580	2.495	1	0.114	-0.807	0.087
	Training Type	-1.324	1.302	-1.017	1.035	1	0.309	-3.876	1.227
	PSIQ Global Average	1.358	0.838	1.620	2.624	1	0.105	-0.285	3.001
	SO Latency	-0.120	0.072	-1.674	2.803	1	0.094	-0.260	0.020
7	(Intercept)	-0.934	6.836	-0.137	0.019	1	0.891	-14.332	12.465
	Age	-0.333	0.225	-1.481	2.193	1	0.139	-0.775	0.108
	PSIQ Global Average	1.416	0.806	1.756	3.084	1	0.079	-0.164	2.995
	SO Latency	-0.111	0.057	-1.941	3.768	1	0.052	-0.224	0.001