The cognitive neuroscience of lucid dreaming

Benjamin Baird[a*]

Sergio A. Mota-Rolim[b]

Martin Dresler[c]

[a] *Wisconsin Institute for Sleep and Consciousness, University of Wisconsin–Madison, Madison, Wisconsin, USA*

[b] *Brain Institute, Physiology Department and Onofre Lopes University Hospital - Federal University of Rio Grande do Norte, Natal, Brazil*

[c] *Donders Institute for Brain, Cognition and Behaviour, Radboud University Medical Centre, Nijmegen, The Netherlands*

* Corresponding author

*Address for correspondence:

Dr. Benjamin Baird

Department of Psychiatry, School of Medicine and Public Health

University of Wisconsin – Madison

6001 Research Park Blvd, Madison, WI, 53719

Email: bbaird@wisc.edu

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Abstract

Lucid dreaming refers to the phenomenon of becoming aware of the fact that one is dreaming during ongoing sleep. Despite having been physiologically validated for decades, the neurobiology of lucid dreaming is still incompletely characterized. Here we review the neuroscientific literature on lucid dreaming, including electroencephalographic, neuroimaging, brain lesion, pharmacological and brain stimulation studies. Electroencephalographic studies of lucid dreaming are mostly underpowered and show mixed results. Neuroimaging data is scant but preliminary results suggest that prefrontal and parietal regions are involved in lucid dreaming. A focus of research is also to develop methods to induce lucid dreams. Combining training in mental set with cholinergic stimulation has shown promising results, while it remains unclear whether electrical brain stimulation could be used to induce lucid dreams. Finally, we discuss strategies to measure lucid dreaming, including best-practice procedures for the sleep laboratory. Lucid dreaming has clinical and scientific applications, and shows emerging potential as a methodology in the cognitive neuroscience of consciousness. Further research with larger sample sizes and refined methodology is needed.

Keywords: lucid dreaming; consciousness; meta-awareness; dreaming; REM sleep

1. Introduction

Becoming aware that one is dreaming while dreaming, what is today referred to as *lucid dreaming*, has been known about since antiquity. In Western literature, it may have first been mentioned by Aristotle in the fourth century BCE in the treatise *On dreams* of his *Parva Naturali*, in which he states: "often when one is asleep, there is something in consciousness which declares that what then presents itself is but a dream" (Aristotle, 1941, p. 624). Likewise, in Eastern cultures, particularly of the south Asian subcontinent, reports of individuals engaging in practices to cultivate awareness of dream and sleep states date back millennia (LaBerge, 2003; Norbu and Katz, 1992; Wallace and Hodel, 2012). These include meditative practices specifically designed to "apprehend the dream state" (Padmasambhava, 1998, p. 156).

Although numerous references to lucid dreaming can be found throughout world literature (see LaBerge, 1988a for an overview), the modern nomenclature of *lucid dream* was not introduced until 1913 by the Dutch psychiatrist Frederik Van Eeden. In a detailed and engaging account of his personal experiences with dreams, Van Eeden (1913) referred to lucid dreams as dreams in which "...the reintegration of the psychic functions is so complete that the sleeper remembers day-life and his own condition, reaches a state of perfect awareness, and is able to direct his attention, and to attempt different acts of free volition" (pp. 149-150). Research over the last four decades has largely confirmed Van Eeden's accounts: as we review below, evidence suggests that during lucid dreams individuals can be physiologically asleep while at the same time aware that they are dreaming, able to intentionally perform diverse actions, and in some cases remember their waking life (Dresler et al., 2011; LaBerge, 1985, 1990; LaBerge, 2015; LaBerge, Nagel, Dement and Zarcone, 1981c; Windt, 2015).

Despite the fact that such personal accounts of lucid dreams have been described for centuries, the topic faced skepticism from some scientists and philosophers (e.g., Malcolm, 1959), in part due to the lack of objective evidence for the phenomenon. This began to change in the late 1970s and early 1980s, however, with the first validation of lucid dreaming as an objectively verifiable phenomenon occurring during rapid eye movement (REM) sleep. Building on prior research that showed that shifts in the direction of gaze within a dream can be accompanied by corresponding movements of the sleeper's eyes (Dement and Wolpert, 1958), lucid dreamers were asked to move

their eyes in a distinct pre-agreed upon sequence (full-scale up-down or left-right movements) as soon as they became lucid (Hearne, 1978; LaBerge et al., 1981c).

Through this technique, which has since become the gold standard, reports of lucid dreams could be objectively verified by the presence of distinct volitional eye movement patterns as recorded in the electrooculogram (EOG) during polysomnography-verified sleep (Figure 1). The most common version of the eye signaling technique asks participants to signal when they realize they are dreaming by rapidly looking all the way to the left then all the way to the right two times consecutively then back to center in the dream without pausing (referred to as left-right-left-right eye signals, abbreviated as LRLR). As can be seen in Figure 1, the LRLR signal is readily discernable in the horizontal EOG, which exhibits a distinctive shape containing four consecutive full-scale eye movements that have larger amplitude compared to typical REMs. As we describe in detail below, lucid dreams can be validated by this method through the convergence between reports obtained after awakening of becoming lucid and making the eye movement signals during the dream, accompanied by the objective eye movement signals recorded in the EOG with concurrent polysomonographic evidence of REM sleep.

The eye signaling technique also offers a way of objectively contrasting lucid REM sleep to baseline non-lucid REM sleep, providing a method to investigate the changes in brain activity associated with lucid dreaming. Furthermore, lucid dreamers can not only signal to indicate that they are aware that they are dreaming, but they can also make the eye movement signals to time-stamp the start and end of experimental tasks performed during lucid dreams (LaBerge, 1990). By providing objective temporal markers, this technique has opened up a new method for studying the psychophysiology of REM sleep, allowing, for example, investigations into the neural correlates of dreamed behaviors (e.g., Dresler et al., 2011; Erlacher, Schredl and LaBerge, 2003; LaBerge, 1990; Oudiette et al., 2018). Lucid dreaming thus provides a way to establish precise psychophysiological correlations between the contents of consciousness during sleep and physiological measures, as well as enables experimental control over the content of dreams, and therefore provides a potentially highly useful experimental methodology.

While neuroscientific studies on lucid dreaming have been performed since the late 1970s, the topic has received increasing attention in recent years due to its relevance to the emerging neuroscience of consciousness. In this article, we review the existing

literature on the neuroscience of lucid dreaming, including electrophysiological, neuroimaging, brain lesion, pharmacological and brain stimulation studies. Additionally, we review recent studies that illustrate how lucid dreaming can be used as a methodology in the cognitive neuroscience of consciousness. Finally, we present strategies to measure lucid dreaming both physiologically and with questionnaires, and discuss procedures to investigate lucid dreaming in the sleep laboratory.

2. Electrophysiology of lucid dreaming

As noted in the introduction, the first physiological studies of lucid dreaming began in the late 1970s and early 1980s. These pioneer works established that lucid dreams occur in REM sleep, characterized by all of the EEG features of REM sleep according to the Rechtschaffen and Kales (1968) sleep scoring criteria (Hearne, 1978; LaBerge et al., 1981c). LaBerge (1980b) also observed that lucid dreams are associated with increased physiological activation, as measured by increased phasic activity (e.g., increased REM density). Autonomic nervous system arousal (e.g., heart rate, respiration rate, and skin potential) were also found to be elevated during lucid REM sleep compared to non-lucid REM sleep (LaBerge, Levitan and Dement, 1986). Additionally, lucid dreams were found to occur in REM sleep periods later in the night (LaBerge et al., 1986). These findings suggest that lucid dreaming is associated with increased cortical activation (LaBerge, Nagel, Taylor, Dement and Zarcone, 1981a), which reaches its peak during phasic REM sleep. In addition to physiological markers of phasic activity, lucid REM sleep was found to be associated with h-reflex suppression (Brylowski, Levitan and LaBerge, 1989), a spinal reflex that is reliably suppressed during REM sleep (Hodes and Dement, 1964). Together these results indicate that lucid dreams occur in activated periods of REM sleep, as opposed to, for example, a state that is intermediate between waking and REM sleep.

These findings also raise the further question of whether lucid REM sleep is associated with localized activation of specific brain regions or changes in specific frequencies of neural oscillations compared to non-lucid REM sleep. In this section, we will review EEG studies that have attempted to address this question. As will be discussed below, while these studies represent important first steps toward measuring the electrophysiological changes associated with lucid dreaming, all of them have interpretive issues and most suffer from low statistical power. As a result, there is considerable discrepancy among findings. Below we group and discuss results based on the regional EEG band power changes reported to be associated with lucid REM sleep dreaming.

2.1 EEG studies of lucid REM sleep dreaming

2.1.1 Central and posterior alpha

Ogilvie and colleagues conducted some of the first studies to examine EEG spectral changes during lucid REM sleep. In an early case study, Ogilvie, Hunt, Sawicki and McGowan (1978) compared two lucid REM sleep epochs to six non-lucid REM sleep epochs and found an increase in the percentage of alpha band (8-12 Hz) power in a single central EEG channel. A follow-up study examined a larger group of ten participants for two nights in the sleep laboratory (Ogilvie, Hunt, Tyson, Lucescu and Jeakins, 1982). Participants were awakened during the two periods of highest alpha power and two periods of lowest alpha power from a central EEG electrode during REM sleep, after which they were asked several questions about their lucidity, "pre-lucidity" (i.e., thoughts pertaining to dreaming without becoming lucid), and degree of dream control. Unfortunately, statistics for the number of lucid dreams reported by participants were not documented in this study, nor whether there was a significant difference between conditions in the number of lucid dreams. Instead, a composite measure was constructed by collapsing across pre-lucidity, lucidity and control, which was significantly elevated in the high alpha trials. Therefore, the number (if any) of lucid dreams that were captured by this procedure is unknown (see LaBerge (1988b) for further discussion).

Subsequent research has not supported the hypothesis that lucid REM sleep is associated with increased alpha activity. For example, in a follow-up study Tyson, Ogilvie and Hunt (1984) found that only pre-lucid but not lucid dreams significantly differed in alpha activity compared to non-lucid REM sleep. In another study of eight frequent lucid dreamers using a similar experimental design, Ogilvie, Hunt, Kushniruk and Newman (1983) observed no difference in the number of lucid dreams following awakenings from periods of high or low alpha activity. Furthermore, in a replication attempt of the original case report that observed an increase in alpha during lucid REM sleep (Ogilvie et al., 1978), LaBerge and colleagues found no significant differences in alpha power at the same central channel (C3) from a single subject (LaBerge, 1988b). Finally, a later follow-up case study analyzed EEG spectral power in five lucid REM sleep epochs compared to non-lucid REM sleep periods, and observed no differences in alpha power (Ogilvie, Vieira and Small, 1991). Together this evidence does not support a reliable association between alpha power and lucid dreaming. However, the limited spatial coverage of EEG montages in these studies (in several cases consisting of only

one EEG channel) makes it unclear to what extent these results can be generalized to other brain areas.

2.1.2 Parietal beta

Holzinger, LaBerge and Levitan (2006) examined EEG spectral changes during lucid REM sleep in a group of eleven participants who reported prior experience with lucid dreaming. Six out of the eleven participants succeeded in becoming lucid in the sleep laboratory, and some during multiple REM periods, for a total of 16 signal-verified lucid dreams. The authors found increased power in the low beta frequency range (13-19 Hz) in parietal electrodes for lucid compared to non-lucid REM sleep. This study has the advantage of analyzing a larger number of lucid REM sleep periods. However, a limitation is that EEG signals were only evaluated at four electrodes (F3, F4, P3, P4). Consequently, localized changes in EEG spectra may have been missed by the low spatial resolution. Furthermore, due to technical limitations, the online low-pass filter for the EEG recordings in this study was set at 35 Hz, and therefore changes in higher-frequency activity were unable to be evaluated.

The potential functional significance of increased low beta band power in parietal areas to lucid dreaming is not understood. Holzinger et al. (2006) speculated that the increased beta power in the parietal EEG could reflect the understanding of the meaning of the words "This is a dream." As discussed below, recent neuroimaging studies have linked parietal regions to several other cognitive functions associated with lucid dreaming, including self-reflection (Kjaer, Nowak and Lou, 2002; Lou et al., 2004), episodic memory (Berryhill, Phuong, Picasso, Cabeza and Olson, 2007; Wagner, Shannon, Kahn and Buckner, 2005) and agency (Cavanna and Trimble, 2006), suggesting several other possible interpretations of the results. Neural oscillations in this frequency range were originally thought to reflect sensorimotor behavior; however, evidence now suggests that oscillations in this range could play a role in cognitive processing as well as facilitate large-scale neural integration (e.g., Donner and Siegel, 2011; Engel and Fries, 2010). If these results can be replicated in future studies, one hypothesis is that the increased oscillatory activity in the beta band could reflect a mechanism of integration between parietal regions and other areas, which, in some way still to be understood, helps facilitate lucid dreaming. However, it is also possible that differences in sensorimotor behavior between lucid and non-lucid REM sleep could

boost brain rhythms that overlap with this frequency range (Koshino and Niedermeyer, 1975; Pfurtscheller, Stancak and Neuper, 1996; Vanni, Portin, Virsu and Hari, 1999). Additional research is needed to clarify these findings.

2.1.3 Frontolateral gamma

Mota-Rolim et al. (2008) and Voss, Holzmann, Tuin and Hobson (2009) reported increased gamma band (40 Hz) power in frontolateral scalp electrodes during lucid compared to non-lucid REM sleep in three participants each. However, besides an unsuccessful replication in patients with narcolepsy (Dodet et al., 2014), interpretation of these results are restricted by several experimental limitations, including the small sample size (LaBerge, 2010). Importantly, caution is warranted in interpreting these findings given that, as briefly discussed by Voss et al. (2009), scalp measurement of cortical gamma, particularly when selectively localized in the frontal and periorbital regions, may be confounded with mircrosaccades. Ocular myogenic artifacts, which occur during both saccades and microsaccades and are distinct from the artifacts associated with corneo-retinal dipole offsets, may confound scalp measurement of gamma activity. One type of such artifacts is referred to as the saccadic spike potential (SP), which occurs due to contraction of the ocular muscles during both saccades and microsaccades (Yuval-Greenberg and Deouell, 2009; Yuval-Greenberg, Tomer, Keren, Nelken and Deouell, 2008). The influence of the SP artifact on gamma power was overlooked for a number of years; however, the need to account for this artifact on scalp measurement of induced gamma activity has now been thoroughly documented (e.g., Hipp and Siegel, 2013; Keren, Yuval-Greenberg and Deouell, 2010).

Voss et al. (2009) corrected for eye movement artifacts using regression of the EOG signal (Gratton, Coles and Donchin, 1983) and by computing current source densities (CSD) in addition to scalp potentials. However, regression-based correction procedures are insufficient to remove the SP artifact, and while the CSD derivation attenuates the SP artifact at posterior channels, it is not sufficient to remove it at anterior scalp locations (Keren et al., 2010; Yuval-Greenberg and Deouell, 2009). As Keren et al. (2010) state: “In conclusion, SCD [CSD] seems to be effective in attenuating the SP effect at posterior sites. However at sites anterior to Cz and closer to the orbits efficacy gradually decreases, preserving the temporal and spectral signature of the SP and its amplitude relative to baseline.” (p. 2258). The influence of the SP artifact on gamma

band power in the comparison of lucid to non-lucid REM sleep is particularly relevant given that, as noted above, lucid REM sleep has been associated with increased phasic activation and higher eye movement density (e.g., LaBerge, 1990).

It is important to note that the need to account for the SP artifact does not preclude a potential association between increased frontal gamma power and lucid REM sleep. Indeed, given the link between gamma band power, local field potentials (LFPs) and the blood-oxygen-level dependent (BOLD) signal (Lachaux et al., 2007; Nir et al., 2007), if the transition from non-lucid to lucid REM sleep involves activation or recruitment of additional frontal brain regions (a plausible hypothesis, also in light of the findings of Dresler et al. (2012); see *Neuroimaging of lucid dreaming* below), regional increases in gamma power might be predicted. However, the spatial topography and frequency localization of any such effects are likely to be strongly influenced by the correction and removal of the SP artifact.

With the aforementioned considerations in mind, more research is needed to clarify whether the increase in frontolateral gamma power during lucid REM sleep observed by Mota-Rolim et al. (2008) and Voss et al. (2009) reflects ocular myogenic or neural activity. Furthermore, future studies evaluating the relationship between lucid REM sleep and gamma activity from sensor-level EEG, particularly at anterior electrodes, need to control for the SP artifact in order for the results to be interpretable. Several methods have been shown to be suitable for removal this artifact, including direct identification and removal of contaminated data by rejecting overlapping windows of time-frequency transformed data or data correction using independent component analysis (Hipp and Siegel, 2013). A study that evaluates the effect of direct removal and/or correction of the SP artifact on gamma power during lucid contrasted with baseline REM sleep would be an important addition to the literature. In summary, studies that rigorously control for myogenic artifacts, and in particular the SP artifact, are needed before conclusions can be drawn regarding the relationship between lucid REM sleep and frontal gamma activity.

2.1.4 Fronto-central delta

Dodet, Chavez, Leu-Semenescu, Golmard and Arnulf (2014) evaluated changes in EEG band power during lucid REM sleep in a group of narcoleptic patients. Given that

narcoleptic patients often report a high rate of lucid dreams (Rak, Beitinger, Steiger, Schredl and Dresler, 2015), they are a potentially useful population for cognitive neuroscience studies of lucid dreaming. In the experiment, while both control and narcoleptic patients reported achieving lucidity and performing the LRLR signals during overnight and afternoon nap recordings, only the eye signals of the patients during the nap recordings could be unambiguously identified. This is likely at least partially attributable to the specific instructions used for making the eye movement signal (see Section 9 below for discussion of this issue). Despite this, the study succeeded in recording 14 signal-verified lucid dreams during naps from seven narcoleptic patients. The main finding was that EEG power was reduced in the delta band during lucid REM sleep at frontal and central electrodes. The study also reported that the coherence between several electrodes was reduced in lucid compared to non-lucid REM sleep in delta, theta, beta and gamma bands, but these differences are difficult to interpret since no statistics were reported for this analysis and no corrections for multiple comparisons were made.

A limitation of the Dodet et al. (2014) study is that EEG signals were only evaluated at six electrodes, and only in frontal and central scalp regions (Fp1, Fp2, F7, F8, C3, C4). Parietal electrodes were not included in the EEG montage, and occipital channels reportedly could not be evaluated due to noise. Thus, it is possible that local changes in EEG spectra in posterior regions, such as parietal or occipital areas, were missed due to the limited electrode montage.

The finding of lower delta activity during lucid REM sleep is in line with previous observations that lucid dreams tend to occur during periods of increased cortical activation (LaBerge, 1990). Specifically, slow waves, reflected by delta (~0.5-4 Hz) power in the EEG, are associated with neuronal down states ("off" periods) in which neurons are hyperpolarized (Steriade, Timofeev and Grenier, 2001). Decreased delta power (EEG activation) therefore reflects recovery of neural activity. While the bi-stability between "on" and " off" periods is a central feature of non-REM sleep, slow wave activity has also been observed in REM sleep (Baird et al., 2018b; Funk, Honjoh, Rodriguez, Cirelli and Tononi, 2016). Neuronal down states have also been linked to the loss of consciousness during both anesthesia and sleep (Purdon et al., 2013; Tononi and Massimini, 2008), which is hypothesized to be related to the breakdown of causal interactions between bi-stable neurons (Pigorini et al., 2015; Tononi, Boly, Massimini

and Koch, 2016). Therefore, one potential explanation is that this finding reflects reduced bi-stability and increased causal interactions between cortical neurons in these areas during lucid REM sleep. Notably, reduced delta power in posterior cortex has been found to be associated with dreaming as opposed to dreamless sleep in both REM and NREM sleep (Siclari et al., 2017). An intriguing speculation based on these results is therefore that this reduction in delta power also extends to frontal regions during lucid REM sleep dreaming. However, it remains to be seen whether these findings can be replicated and whether the results generalize to non-clinical populations.

2.2. General discussion of EEG studies of lucid REM sleep

In summary, EEG studies show substantial disagreement regarding the spatial and spectral changes associated with lucid dreaming. As reviewed above, different studies have observed an increase in central or posterior alpha, parietal beta, frontolateral gamma or a reduction in frontocentral delta during lucid compared to baseline REM sleep. Aside from the general uncertainty in the results of some studies due to low statistical power, these discrepant results might be partially explained by the use of limited electrode montages and evaluation of different EEG frequency bands. In the spatial domain in particular, many studies have used less than six scalp electrodes, in several cases only covering some scalp regions but not others, precluding analysis of EEG activity in regions in which significant effects were found in other studies. For instance, the study by Dodet et al. (2014) did not include electrodes in parietal or occipital regions, precluding the possibility to replicate the findings of increased parietal beta by Holzinger et al. (2006). Studies by Ogilvie et al. (1978, 1982) evaluated only a single central EEG channel (C3). These non-overlapping spatial montages limit the comparison of results across some of these studies.

Another factor that might contribute to the discrepant findings is the fact that lucid dreaming can be achieved and executed in different ways. For example, the observed changes in the EEG during lucid REM sleep might depend in part on the degree of vividness, working memory, emotional tone, self-consciousness, attention and insight, which could vary across individuals as well as specific dreams. Relatedly, different subjective experiences and contents during lucid dreams plausibly have their own neurobiological substrates (Mota-Rolim, Erlacher, Tort, Araujo and Ribeiro, 2010), just as in non-lucid dreams (Siclari et al., 2017). Changes in brain activity during lucidity

may also partly depend on how experienced the lucid dreamer is. For instance, lucid dreams of less experienced individuals may often be more ephemeral and involve less control over dream content, while more experienced lucid dreamers may be more likely to have longer and more stable lucid dreams, as well as the capacity to exert greater amounts of control. This might lead to a more distinct signal in the EEG for experienced lucid dreamers on the one hand, but also presumably less neural activity related to the effort needed to maintain the state (neural efficiency). In line with this, Dodet et al. (2014) suggested that the mental effort needed to achieve and sustain lucidity might be reduced in narcoleptic patients, who may access the lucid REM sleep state with less effort.

These comments should not be taken to indicate that there is not a consistent neurobiology of lucid dreaming. However, it does suggest that analysis of the EEG spectral changes associated with lucid dreaming used in previous studies may need to be optimized for detecting more subtle and localized effects. All studies reviewed above have measured the average power over a given spectral band and region over comparably long time intervals. However, it is possible that lucid dreaming is associated with spectral changes that can only be detected by a better time resolved analysis, such as time-frequency analysis, that may be overlooked by averaging over large windows in time or frequency space.

Furthermore, these considerations emphasize the need for more careful assessment of the phenomenology of lucid dreams. In this regard, we would like to note that it is plausible that there are at least two different neural signatures associated with lucid dreaming. The first captures what might be termed the “moment of lucidity”—that is, the transient moment of meta-awareness in which one has the metacognitive insight that one is currently dreaming (Schooler, 2002). The second captures potential sustained differences in brain activity between lucid and non-lucid REM sleep dreaming. This second neural signature is unlikely to be a signature of meta-awareness per se, as during lucid dreams individuals do not continuously engage in metacognitive reflection on their state of consciousness. Rather, this second signature captures the “state-shift” in consciousness that occurs from non-lucid to lucid dreaming, with enhanced volition, episodic memory and accessibility of metacognition (Dresler et al., 2014; Spoormaker, Czisch and Dresler, 2010). Changes in these aspects of cognition in the shift to lucidity have been hypothesized to reflect an overall change in the conscious experience of

being a *cognitive subject* (Windt and Metzinger, 2007). Both the physiological correlates of the moment of lucidity as well as overall differences in brain activity between lucid and non-lucid dreams are interesting research targets. However, these research targets are at least conceptually distinct, a point that has, in our view, not received adequate attention in the research literature. To our knowledge, no studies have yet evaluated differences in brain activity with EEG or functional magnetic resonance imaging (fMRI) specifically associated with the moment of lucidity, and this remains an interesting question for future work.

Larger sample sizes are needed in future studies to achieve adequate statistical power. One way to approach this would be to undertake more extensive population screening for high-frequency lucid dreamers. For example, through mass surveys, thousands of potential participants could be screened and the top few percent reporting the highest lucid dream frequency could be selected for training before undergoing sleep laboratory recordings. As we discuss below, new techniques for lucid dream induction also have the potential to enable efficient collection of larger datasets.

Overall, studies with higher statistical power, better assessment of phenomenological content, higher spatial resolution EEG montages, and more sophisticated analysis of the EEG signal will be needed to address these issues and shed light on the conflicting findings of EEG studies of lucid dreams conducted to date. Studies using high-density EEG would also be valuable, enabling both higher temporal as well as higher spatial resolution analysis of neural oscillatory activity. Source modeling of such data could also potentially be informative for localizing changes in neural oscillations associated with lucid dreaming to specific cortical areas, though it remains unclear whether methods for source localization will be able to produce a valid specification of the generators relevant for lucid dreaming. In particular, the generators might be distributed widely in the brain and active concurrently.

Another interesting question with respect to the electrophysiology of lucid dreaming is whether there are possible sleep pattern traits that are associated with lucid dreaming. It would be informative to investigate whether there are common sleep patterns seen in frequent lucid dreamers compared to non-frequent lucid dreamers. For example, do frequent lucid dreamers tend to have more phasic REM sleep, or more fragmented REM sleep with a higher number of transitions (especially gradual transitions) between REM

sleep and waking? As far as we know, no study has investigated lucid dreaming with this approach; therefore, studies addressing these questions would be valuable.

2.3 EEG studies of lucid dreaming during non-REM sleep

The activated EEG of REM sleep was originally thought to be exclusively associated with dreaming (Antrobus and Antrobus, 1967; Dement and Wolpert, 1958), while the low-frequency activity of non-REM (NREM) sleep was thought to be associated with the absence of dreaming. Subsequent research has shown, however, that participants report dreams or related forms of sleep mentation in up to 70% of awakenings from NREM sleep (Siclari et al., 2017; Siclari, LaRocque, Postle and Tononi, 2013; Stickgold, Malia, Fosse and Hobson, 2001). NREM dreams tend to be less emotional and visually vivid, as well as more thought-like (Cavallero, Cicogna, Natale, Occhionero and Zito, 1992; Hobson, Pace-Schott and Stickgold, 2000). Research suggests that lucid dreams, on the other hand, are predominantly a REM sleep phenomenon (LaBerge et al., 1986; LaBerge et al., 1981c). However, this does not imply that lucid dreams cannot occur during NREM sleep. There have been several reports of lucid dreams during NREM sleep stages N1 (transition from wake state to sleep) and N2 (consolidated light sleep), although in many of the published cases it is uncertain whether the lucid episode occurred in an unambiguous stage of NREM sleep (Dane and Van de Caslte, 1984; LaBerge, 1980b, 1990; LaBerge et al., 1981c; Stumbrys and Erlacher, 2012).

In one study, LaBerge (1980b) recorded polysomnography from a single subject who reported frequently experiencing lucid dreams at sleep onset. The participant rested quietly while drifting to sleep and upon falling asleep was awakened and asked for a dream report. Forty-two dream reports were collected over three nights, 25 of which the participant reported as a lucid dream, and all of which reportedly occurred during stage N1. However, the dream reports were mostly short "dreamlets", thus it is plausible that these N1 lucid dreams could differ phenomenologically from REM sleep lucid dreams. Furthermore, none of these lucid dreams were verified with eye movement signals. In another study, two participants reported lucid dreams upon spontaneous awakening from N1 and N2 (LaBerge et al., 1981c). In the N2 instance, the participant reported

only a brief moment of lucidity just before waking up. Furthermore, the participant did not make eye movement signals to time-stamp the moment of lucidity, and it is therefore difficult to ascertain whether the moment of lucidity occurred in the process of awakening. The N1 case was also ambiguous: while in this case the participant reported making eye movements to signal lucidity, the signals could not be verified on the polysomnogram.

In a study on the effects of posthypnotic suggestion on lucid dreaming, Dane and Van de Caslte (1984) tested hypnotically susceptible females with no prior experience in lucid dreaming. Importantly for the present discussion, lucid dreams were reported following awakening from both REM and NREM sleep. Five lucid dreams were reported in total from stage N2, but in all cases the LRLR eye signal occurred after arousal/awakening, and thus none of these could be objectively confirmed. However, several N1 lucid dreams were confirmed by LRLR signaling. This study was thus the first to provide objective evidence for lucid dreaming during stage 1 NREM sleep. However, as noted above, how these N1 dreams compare phenomenologically to REM sleep lucid dreams remains unclear.

Stumbrys and Erlacher (2012) reported two potential cases of lucid dreams during NREM with eye signaling. However, due to the study protocol, the experimenters could only collect dream reports the following morning. In the first case, the participant reported the next morning making an eye movement signal in a lucid dream early in the night, but given the long amount of time between the report and the signal there is uncertainty whether the report corresponds to the observed signal during NREM sleep. Furthermore, while the two 30-second polysomnography epochs for sleep scoring preceding the eye signal appear to be unambiguous N2 sleep, the EEG dynamic shifts during the 30-second epoch containing the eye-signal to lower amplitude activity without apparent spindles or K-complexes. Without knowing the stage of the epochs following the eye signal, which was not reported in the study, it is possible that the eye signal occurred in a transitional sleep stage. In the second case reported by Stumbrys and Erlacher (2012), the eye signals occurred with some signs of arousal and the participant had no memory of executing the eye signals the following morning. These data therefore provide ambiguous evidence for signal-verified lucid dreams during NREM sleep.

In three further case reports of eye movement signaled NREM lucid dreams, one case was reported to occur in N1 and two cases in N2 visually scored sleep stages (Mota-Rolim et al., 2015). The first case was scored as N1, since an increase in theta (4-7 Hz) and a decrease in alpha (7-14 Hz) power was observed in more than half of the 30 second scoring epoch, meeting the AASM criteria for classification of stage N1 sleep. The other two cases were scored as occurring during N2 episodes since they had spindles and K-complexes in the 30 second scoring epoch with the eye signals. These data thus replicate signal-verification of N1 lucid dreams and provide preliminary evidence for signal-verified N2 lucid dreams.

Together, these results suggest that although most lucid dreams occur during REM sleep, they can also occur during NREM sleep. However, additional studies providing objective evidence of NREM lucid dreams confirmed by eye-signaling, particularly in N2 sleep, are needed. Currently there are no reports of lucid dreams recorded during NREM stage 3 (N3), also known as deep sleep, or slow wave sleep. While there are intriguing reports of practitioners of both Transcendental Meditation and Tibetan Dream Yoga claiming to have developed the ability to maintain a type of lucid awareness throughout the entire sleep cycle, including also states of "lucid dreamless sleep" (Gackenbach, Cranson and Alexander, 1986; Mason and Orme-Johnson, 2010; Wallace, 2013; Windt, Nielsen and Thompson, 2016), these claims have not been corroborated with physiological measures. However, several studies have found a relationship between meditation practice and lucid dreaming (e.g., Baird, Riedner, Boly, Davidson and Tononi, 2018c; Gackenbach et al., 1986; Mota-Rolim et al., 2013; Stumbrys, Erlacher and Malinowski, 2015), which could be due to changes in REM sleep patterns induced by meditation practice and/or to neurocognitive changes associated with meditation practice (e.g., increased mental control or meta-awareness).

3. Neuroimaging of lucid dreaming

As noted, dream-like mental activity can be observed during all sleep stages. However, REM sleep dreams tend to be more vivid, emotional, bizarre, and more often include a narrative structure (Cavallero et al., 1992; Hobson et al., 2000). These phenomenological characteristics have been suggested to be associated with the neural activation and deactivation patterns observed during REM sleep (e.g., Nir and Tononi, 2010). For example, higher visual areas show increased regional cerebral blood flow during REM sleep compared to both wakefulness and slow wave sleep (Braun et al., 1998), which is in line with the visuospatial experiences that are common during REM sleep dreaming (e.g., Windt, 2010). Additionally, the amygdala, medial prefrontal cortex and anterior cingulate cortex show increased regional cerebral blood flow during REM sleep (Braun et al., 1997; Maquet et al., 1996). All of these brain areas have been implicated in emotional processing, mirroring the intense emotions that can be experienced in REM sleep dreams. In contrast, the anterior prefrontal cortex (aPFC) and parietal cortex, including the inferior parietal lobule and precuneus, show low regional cerebral blood flow during normal REM sleep (Braun et al., 1997; Maquet et al., 1996). Deactivation of these regions has been postulated to underlie the diminished insight into the global state of consciousness and restricted volitional control typical of non-lucid dreaming (e.g., Hobson and Pace-Schott, 2002; Nir and Tononi, 2010).

3.1 Neuroimaging lucid REM sleep dreaming

At the current time, there is only one fMRI study of lucid REM sleep, and it is a case study (Dresler et al., 2012). Two separate signal-verified lucid dreams were recorded from a single subject during EEG-verified REM sleep inside the MRI scanner. Compared to non-lucid REM sleep, lucid REM sleep showed increased fMRI BOLD signal in a number of cortical regions, including the superior frontal gyrus, aPFC, medial and lateral parietal cortex, inferior/middle temporal gyri and occipital cortex (Figure 2a). Interestingly, several of these regions, particularly the parietal regions and frontal pole, are areas that, as noted above, consistently show reduced regional cerebral blood flow during non-lucid REM sleep compared to wakefulness (Nir and Tononi, 2010).

Evidence linking frontopolar cortex to lucid dreaming is consistent with a role of this region in metacognition and self-reflection. For example, research has found that aPFC shows increased activation during self-reflection on internal states, such as the evaluation of one's own thoughts and feelings (Christoff, Ream, Geddes and Gabrieli, 2003; McCaig, Dixon, Keramatian, Liu and Christoff, 2011). Individuals can also learn to voluntarily modulate activity in aPFC through a metacognitive awareness strategy (McCaig et al., 2011). Furthermore, inter-individual variance in metacognitive ability has also been linked to aPFC gray matter volume (Fleming, Weil, Nagy, Dolan and Rees, 2010) and aPFC resting-state functional connectivity (Baird, Smallwood, Gorgolewski and Margulies, 2013). Finally, patients with damage to this region frequently display metacognitive deficits such as inability to monitor disease symptoms or accurately appraise their cognitive functioning (Joseph, 1999; Schmitz, Rowley, Kawahara and Johnson, 2006), which might be compared to the lack of metacognitive insight into the state of consciousness characteristic of non-lucid REM sleep dreams (Dresler et al., 2015).

Dresler et al. (2012) additionally observed increased BOLD signal during lucid dreaming in the bilateral precuneus and inferior parietal lobules (angular and supramarginal gyri). As noted above, parietal cortex and the precuneus in particular has been implicated in self-referential processing, episodic memory, and the experience of agency (Cavanna and Trimble, 2006), mirroring the increase of these cognitive capabilities during lucid dreaming. Finally, activation increases during lucid dreaming were also found in some occipital and inferior temporal regions, which are part of the ventral stream of visual processing involved in conscious visual perception (Rees et al., 2002). While these activations may seem puzzling at first sight, as non-lucid dreams are also characterized by vivid dream imagery, they are in line with reports that lucid dreams can be associated with increased visual vividness and clarity of the dream scene (e.g., Green, 1968).

There are several limitations to the study by Dresler et al. (2012) that are important to note. First, as mentioned above, the findings are based on observations from a single subject and caution is therefore warranted in generalizing from the results. Currently no group-level fMRI study of lucid dreaming has been conducted, and such a study, along with systematic replications, will be needed before firm conclusions can be drawn. Another limitation is that, as described below (see Section 7.1), the participant was

performing a task during the lucid REM sleep segment (repeated eye signaling and hand clenching). While Dresler and colleagues accounted for task activation via nuisance regression of the left and right fist clenching task, it is possible that some of the activations observed still partially reflect task execution and maintenance of attention/task-set rather than lucidity per se. One way to address this in future studies would be to contrast periods of lucid REM sleep when the participant is not performing an explicit task to non-lucid REM sleep, i.e., a "no-task, within-state paradigm" (Siclari et al., 2013).

3.2 Neuroimaging individual differences in lucid dreaming

Several studies have taken an individual differences approach to neuroimaging of lucid dreaming. While most people spontaneously experience lucid dreams infrequently**,** there is substantial variation in lucid dream frequency, ranging, by current estimates, from never (approximately 40-50%) to monthly (approximately 20%) to a small percentage of people that report lucid dreams several times per week or in some cases every night (Mota-Rolim et al., 2013; Saunders, Roe, Smith and Clegg, 2016; Snyder and Gackenbach, 1988). This variation invites the question of whether individuals who experience frequent lucid dreams show differences in anatomical or functional properties of the brain. Studies addressing this question can provide complementary evidence on the neurobiology of lucid dreaming.

In the first neuroimaging study to evaluate the relationship between lucid dream frequency and brain anatomy, Filevich, Dresler, Brick and Kühn (2015) measured whole-brain gray matter volume using voxel-based morphometry in individuals with higher ("high lucidity") vs. lower ("low lucidity") scores on a scale assessing the frequency of lucid dreams and/or dream content hypothesized to be related to lucidity. Consistent with the hypothesized connection between the metacognitive functions of aPFC and lucid dreaming discussed above, the study found increased gray matter volume in two regions of the frontal pole (BA9/10), as well as the right anterior cingulate cortex, left supplementary motor area and bilateral hippocampus in the high lucidity group. Additionally, the two identified frontopolar regions showed higher BOLD signal in the monitoring component of a metacognitive thought-monitoring task performed while awake.

A limitation of the study by Filevich et al. (2015) is that participants were not frequent lucid dreamers per se, but rather subjects from a database who scored higher relative to other participants on the scale. Lucid dream frequency for the two groups was not reported in the study, thus it remains unclear to what extent the "high lucidity" group experienced frequent lucid dreams in absolute terms. Furthermore, the composite measure of dreaming used to distinguish the two groups measured not only frequency of lucid dreams but also different dimensions of dream content. While several of these content dimensions have been found to be higher in lucid dreams (Voss, Schermelleh-Engel, Windt, Frenzel and Hobson, 2013), it is likely that several of these dimensions also co-vary more generally with dream recall and/or cognitive content in dreams unrelated to lucidity. As a consequence, as the authors note, some of the results could have been partly influenced by differences in dreaming "styles", content or dream recall.

More recently, Baird, Castelnovo, Gosseries and Tononi (2018a) evaluated a sample of high-frequency lucid dreamers who reported lucid dreams in the range of three to four times per week to multiple times per night compared to a control group who reported lucid dreams once per year or less. The frequent lucid dream group and control group were case-control matched on age, gender, and dream recall frequency. Based on the previous research reviewed above, the primary aim of the study was to investigate whether individuals who have frequent lucid dreams would show increased gray matter density and/or resting-state functional connectivity of aPFC. Consistent with this, compared to the control group, individuals who reported frequent lucid dreams showed increased resting-state functional connectivity between the left aPFC and the bilateral angular gyrus, bilateral middle temporal gyrus and right inferior frontal gyrus (Figure 2b). The frequent lucid dream group also showed decreased functional connectivity between left aPFC and bilateral insula. Whole-brain graph-theoretic analysis revealed that left aPFC had increased node degree and strength in the frequent lucid dream group compared to the control group. However, in contrast to the findings of Filevich et al. (2015), no significant differences in gray matter density were observed between groups in either a whole-brain analysis or an aPFC region-of-interest analysis.

Given the link to metacognition, it has also been suggested that lucid dreaming may be linked to large-scale networks that regulate executive control processes, in particular the frontoparietal control network (Dresler et al., 2015; Spoormaker et al., 2010). To

address this question, Baird et al. (2018a) additionally evaluated the association between frequent lucid dreaming and connectivity within established large-scale brain networks, including the frontoparietal control network. Consistent with a link between the frontoparietal control network and lucid dreaming, Baird and colleagues found that frequent lucid dreamers had increased functional connectivity between aPFC and a network of regions that showed the greatest overlap with a frontoparietal control sub-network (Dixon et al., 2018; Yeo et al., 2011). However, neither connectivity within the frontoparietal control network broadly defined through meta-analysis (or within or between any other large-scale networks), nor connectivity within frontoparietal control sub-networks, as defined through parcellation of resting-state networks, showed significant differences between the lucid dream group and the control group. The authors speculate that this could be attributed to both the partial overlap of the regions that showed increased aPFC connectivity in lucid dreamers with the frontoparietal control network. However, it is important to keep in mind that while this study did not find a significant difference in resting-state connectivity within the frontoparietal network in frequent lucid dreamers, it remains an open question whether lucid REM sleep dreams show increased connectivity within the frontoparietal control network compared to non-lucid REM sleep dreams.

Overall, the resting-state connectivity results of Baird et al. (2018a) converge with the fMRI case study of lucid REM sleep dreaming described above (Dresler et al., 2012), which found that an overlapping network of brain areas increased fMRI BOLD signal during lucid compared to baseline REM sleep, including bilateral aPFC, bilateral inferior parietal lobule (including the angular gyrus), and bilateral middle temporal gyrus (Figure 2). Together, these results suggest that increased intrinsic functional connectivity between aPFC and the angular gyrus/middle temporal gyrus—regions that, as reviewed above, show reduced activity in REM sleep (Nir and Tononi, 2010) and increased activity during lucid REM sleep (Dresler et al., 2012)—is associated with the tendency to have frequent lucid dreams. However, while the convergence between these two preliminary studies is encouraging, the paucity of neuroimaging data on this question limits strong conclusions at the current time.

3.3. Future directions for neuroimaging of lucid dreaming

Neuroimaging studies of lucid REM sleep using larger samples sizes are needed. In particular, a group-level fMRI study of lucid REM sleep dreaming using a no-task, within-state paradigm is perhaps the most important next step in this line of research. In addition to evaluating activation and deactivation as revealed by changes in BOLD signal, it would also be informative to evaluate differences in functional connectivity in such a study. For example, as noted, one question that arises from the above neuroimaging findings is whether there could be increased connectivity within the network of regions identified in Dresler et al. (2012) and Baird et al. (2018a) during lucid contrasted with non-lucid REM sleep dreaming. Furthermore, building on the individual differences results, in future work it would also be interesting to evaluate whether high frequency lucid dreamers show increased functional connectivity and/or higher BOLD signal in these brain areas during baseline REM sleep. If so, this could suggest that it may be possible to bias the brain toward increased metacognitive awareness of dreaming during REM sleep, for example, as discussed in the next sections on induction of lucid dreams, through techniques to increase activation of these regions.

In line with these remarks, it has been suggested that not only regional activation of frontoparietal brain areas but also connectivity between these regions could be important for lucidity to emerge during REM sleep (Spoormaker et al., 2010). Indeed, while activation in these frontal and parietal regions has been linked to key functions associated with lucid dreaming, including metacognition, as discussed above, regional activations and metabolic increases in these regions have also been observed during states of global unconsciousness and subliminal information processing. For instance, subliminally presented no-go stimuli during a response inhibition task activate frontoparietal cortices in the absence of awareness (van Gaal, Ridderinkhof, Scholte and Lamme, 2010). Moreover, loss of consciousness during the tonic phase of generalized tonic-clonic seizures is associated with a transient increase rather than decrease in metabolism in frontoparietal cortex (Blumenfeld, 2008; Engel, Kuhl and Phelps, 1982).

Recent findings have suggested that a potentially more sensitive marker of unconscious states may be reduced connectivity between frontoparietal areas, particularly from frontal to parietal regions. For example, several neuroimaging studies of patients in a persistent vegetative state or under different categories of general

anesthetics have shown a specific impairment of the backward connectivity from frontal to parietal regions (Boly et al., 2011; Boly et al., 2012; Lee et al., 2013). These findings converge with theoretical work and computational modeling (Tononi, 2011), which has suggested a link between consciousness and effective connectivity within a neural architecture, or the capacity of a set of neural elements to exert causal influence over other neural groups in a system. At present, it is unclear whether this reduction of top-down frontoparietal connectivity is linked to changes in global brain activity, alterations in primary consciousness (i.e., subjective, phenomenal states of seeing, hearing or feeling), or whether it could relate to self-awareness (i.e., explicit conscious awareness of oneself and one's state). A common interpretation of these results is that top-down frontoparietal connectivity is a marker of global loss of consciousness, including primary consciousness (e.g., Mashour, 2014). However, given that the reduction in top-down connectivity has also been observed under ketamine (Lee et al., 2013), during which patients often report vivid dream-like experiences, it is plausible that the reduction of frontoparietal connectivity could instead indicate loss of self-awareness.

Cognitive neuroscience studies of lucid dreaming are uniquely placed to contribute to this question because the comparison of lucid REM sleep to non-lucid REM sleep is perhaps the only contrast that allows for a direct comparison between the global loss and recovery of reflective consciousness independently of global shifts in primary consciousness, arousal or vigilance state. Thus, the question of whether lucid REM sleep is associated with altered connectivity between frontal and parietal cortices has implications for several broad questions in the cognitive neuroscience of consciousness. Future studies evaluating changes in effective connectivity during lucid REM sleep dreaming, and in particular changes in top-down frontoparietal connectivity, would be valuable.

4. Brain lesions and lucid dreaming

In the neurological literature, to our knowledge only one paper has reported changes in lucid dreaming as a result of neurological insult or brain lesions (Sagnier et al., 2015). The paper describes two case reports of young patients who reported lucid dreams following unilateral ischemic stroke to the left mediodorsal thalamus. The first patient was a 26-year-old female with a left anterior and mediodorsal thalamic stroke in an area supplied by the premamillary artery, as revealed by MRI scans. She reported frequent lucid dreams in the early morning hours, along with increased nightmares and nocturnal awakenings. She also reported that her lucid dreams mostly involved the hospital and medical staff that she encountered during her hospitalization, and included catastrophic events such as helicopter crashes and hyper-aggressive behaviors. The second patient was a 36-year-old male with a left mediodorsal thalamic stroke in the area supplied by the paramedian artery as revealed by MRI scans. He reported frequent lucid dreams following the stroke, which also tended to occur in the early morning hours, along with increased nocturnal awakenings, but without an increase in nightmares. Lucid dreams subsided after one month for both patients.

Lucid dreams have not previously been reported following either unilateral or bilateral thalamic stroke. However, loss of neurons in the anterior and dorsomedian thalamic nuclei that occurs in familial fatal insomnia is associated with loss of nocturnal sleep as well as oneiric 'intrusions' during wakefulness (Montagna, 2005; Raggi, Cosentino, Lanuzza and Ferri, 2010). Furthermore, hypersomnia and irregular sleep are frequently reported following paramedian thalamic stroke (Hermann et al., 2008; Luigetti et al., 2011). These clinical features could be manifestations of disruption of the intralaminar and midline thalamic nuclei located in the mediodorsal thalamus, which are part of the brain's arousal network (Van der Werf, Witter and Groenewegen, 2002). Thus, one possibility is that lucid dreams in these patients could have partly resulted from increased or abnormal functioning of the brain's arousal systems during sleep, which is consistent with the reported increase in nocturnal awakenings. Both patients also reported that their dreams contained highly anxious and emotional content, which could reflect abnormal connectivity between these thalamic nuclei and limbic structures with which they are densely connected (Van der Werf et al., 2002). The highly emotional or disturbing content may have also contributed to lucid dreaming, as these types of experiences could induce individuals to question whether the explanation for

such surprising or frightening experiences is that they are dreaming (LaBerge and Rheingold, 1990).

A systematic study of the incidence of lucid dreams following thalamic strokes is lacking. Particularly given that the lucid dreams subsided after a relatively short duration (one month) for both patients, it is possible that lucid dreaming as a consequence of thalamic strokes has been under-reported and/or overlooked. More generally, a large-scale study of the occurrence of lucid dreaming in neuropsychological cases has not been undertaken and would be a welcome addition to the literature. The converse case, of a brain lesion causing loss of lucid dreaming in an individual who regularly experiences lucid dreams has also to our knowledge not been reported. Such a case would likely be very unusual, particularly considering the small percentage of individuals who experience lucid dreams spontaneously with high frequency. However, if such a case were identified it could be informative to the neurobiology of lucid dreaming.

5. Pharmacological induction of lucid dreaming

A main target of research is to develop methods to make the lucid dream state more accessible. Indeed, reliable techniques to induce lucid dreams are needed for it to be effectively used in both clinical and scientific applications. Evidence suggests that lucid dreaming is a learnable skill (LaBerge, 1980a) that can be developed by training with various induction strategies (LaBerge and Rheingold, 1990; Price and Cohen, 1988; Stumbrys, Erlacher, Schädlich and Schredl, 2012). These include training in prospective memory techniques (LaBerge and Rheingold, 1990), which can be further aided by application of external sensory cues (LaBerge and Levitan, 1995; LaBerge, Levitan, Rich and Dement, 1988; LaBerge, Owens, Nagel and Dement, 1981b) and/or interrupting sleep with short periods of wakefulness (Aspy, Delfabbro, Proeve and Mohr, 2017; LaBerge, 1980a; LaBerge, Phillips and Levitan, 1994; Stumbrys et al., 2012). Within cognitive neuroscience, studies have evaluated pharmacological as well as non-invasive brain stimulation approaches to lucid dream induction. In this section, we review studies that have taken a pharmacological approach to lucid dream induction and in the next section we review electrical brain stimulation studies.

5.1 Effects of Acetylcholinesterase inhibitors (AChEIs) on lucid dreaming

As discussed above, lucid dreaming is associated with increased cortical activation (LaBerge et al., 1981a), which reaches its peak during phasic REM sleep. Given the relationship between phasic REM sleep and lucid dreaming, as well as the role of Acetylcholine (ACh) in REM sleep regulation (e.g., Amatruda, Black, McKenna, McCarley and Hobson, 1975; Velazquez-Moctezuma, Shalauta, Gillin and Shiromani, 1991), agents acting on the cholinergic system have received particular interest. In an initial pilot study, LaBerge (2001) evaluated the effect of donepezil (Aricept), an Acetylcholinesterase inhibitor (AChEI), on lucid dreaming in a small group of participants (N=10) who reported prior experience with lucid dreaming. On each night, participants received either 0 mg (placebo), 5 mg, or 10 mg of donepezil, with the dose order counterbalanced across the three nights of the experiment. Nine of the ten participants (90%) reported at least one lucid dream on donepezil, while only one participant reported a lucid dream on the placebo dose.

Following on these results and additional pilot research, LaBerge, LaMarca and Baird (2018b) conducted a double blind, placebo-controlled study in a large group of participants (N=121) with high dream recall and an interest in lucid dreaming. The first goal of the study was to quantify the size and reliability of the effect of AChEI on lucid dreaming. The second goal was to test the effectiveness of an integrated lucid dream induction protocol that combined cholinergic stimulation with other methods, including sleep interruption and the Mnemonic Induction of Lucid Dreams (MILD) technique, which trains participants to use prospective memory to induce lucid dreams (LaBerge, 1980a; LaBerge and Rheingold, 1990). Participants were randomly assigned counterbalanced orders of three doses of galantamine (0 mg=placebo, 4 mg, and 8 mg), an AChEI that is readily accessible, fast acting and has a mild side effect profile. On three consecutive nights, participants awoke approximately 4.5 hours after lights out (after approximately the 3rd REM cycle), recalled a dream, ingested the capsules and stayed awake for at least 30 minutes. Participants then returned to sleep practicing the MILD technique. After each subsequent awakening, participants rated their dreams on a range of variables including lucidity, recall, vividness, bizarreness, complexity, affect, cognitive clarity, metacognition and control. Full reports of lucid dreams were also collected.

Galantamine was found to significantly increase the frequency of lucid dreaming in a dose-related manner. Increased incidence of lucid dreaming was observed for both 4 mg (27% of participants) and 8 mg (42% of participants) doses compared to 14% of participants for the active placebo procedure (which included sleep interruption and MILD). Galantamine was also found to be associated with significantly increased sensory vividness and environmental complexity, which might be expected given the general intensification of REM sleep, and associated dreaming, triggered by cholinergic stimulation (Riemann, Gann, Dressing, Müller and Aldenhoff, 1994).

Another recent double blind, placebo-controlled study conducted by Sparrow, Hurd, Carlson and Molina, 2018) also tested the effect of galantamine on lucid dreaming. 35 participants completed an eight-night study that tested the effect of 8 mg of galantamine paired with 40 minutes of sleep interruption (termed “Wake-Back-to-Bed” (WBTB) in the study). The study additionally tested combining galantamine with middle-of-the-night meditation and the imaginary reliving of a distressing dream (termed meditation and dream reliving or MDR; Sparrow, Thurston and Carlson, 2013). The study included

pre- and post-baseline nights and six conditions: 1) WBTB; 2) WBTB + placebo; 3) WBTB + galantamine; 4) MDR; 5) MDR + placebo; and 6) MDR + galantamine. MDR conditions matched the 40 minutes of sleep interruption in the WBTB conditions. Lucid dreams were measured with self-reports on the dream lucidity scale (DLS; Sparrow et al., 2013), which in this study included three categories: 0=non-lucid, 1=pre-lucid (which included either "questioning things in the dream without actually concluding that you were dreaming" or "doing things that are ordinarily impossible to do"), or 2=lucid.

Both galantamine conditions (WBTB + galantamine; MDR + galantamine) significantly increased self-ratings of lucidity on the DLS compared to the other conditions. However, no significant difference was observed between WBTB + galantamine and MDR + galantamine. The number of participants who had a lucid dream in each condition was not reported, only the conditional means for the DLS, which reflects the effect collapsed across both pre-lucid and lucid dreams. We therefore contacted the authors for this information in order to compare the results of this study to the study by LaBerge et al. (2018b). 9% of participants reported a lucid dream in the WBTB + placebo condition and 11% in the MDR + placebo, whereas 40% of participants reported a lucid dream in the WBTB + galantamine and 34% in the MDR + galanatamine (S. Sparrow, personal communication, December 17, 2018). Overall, therefore, these results are comparable to the effect of 8 mg galantamine observed by LaBerge and colleagues.

5.2 Effects of L-alpha glycerylphosphorylcholine (α-GPC) lucid dreaming

In contrast to the positive findings for AChEIs, a double blind randomized placebo-controlled study found no significant effect of 1200 mg of the ACh precursor L-alpha glycerylphosphorylcholine (α-GPC) on the frequency of lucid dreams in 33 participants with varying degrees of lucid dreaming experience (Kern, Appel, Schredl and Pipa, 2017). One interpretation of this result is that, in contrast to AChEIs, α-GPC is not effective for inducing lucid dreams, perhaps due to differences in the neurobiological effects of choline—an ACh precursor—and AChEIs. However, participants in this study appear to have received no training in mental set, such as recalling and attending to dreams in an effort to become lucid. Thus, an alternative, not mutually exclusive, interpretation is that training in at least the minimal mental set for lucid dream induction

is needed for pharmacological (and other) interventions to effectively increase the frequency of lucid dreams.

5.3 General discussion of pharmacological induction of lucid dreaming

Overall, these data provide strong initial evidence that cholinergic enhancement with AChEls, and galantamine in particular, facilitates a state of the brain favorable to lucid dreams. However, a limitation of all pharmacological studies on lucid dreaming performed to date is that they were not conducted in a sleep laboratory and there was therefore no validation of lucid dreams with eye-signaling methods. It will be important to follow up these results with sleep laboratory studies to objectively validate lucid dreams with polysomnographic recording. Such studies would also be valuable to investigate the physiological effects of galantamine on the brain during REM sleep, and which effects are predictive of lucidity. Given the high success rate of lucid dreams in the combined induction protocol with cholinergic stimulation reported by LaBerge, LaMarca and Baird (2018) and Sparrow et al. (2018), studies of galantamine have the potential to enable efficient collection of large sample sizes in electrophysiological and neuroimaging studies of lucid dreaming.

The mechanism by which AChEls facilitate lucid dreams remains unclear. There are several, not mutually exclusive, possibilities, including increasing REM sleep intensity/phasic activation, influencing the brain regions/networks associated with lucid dreaming, and influencing cognitive processes associated with becoming lucid. In general, it is known that AChEls inhibit the metabolic inactivation of ACh by inhibiting the enzyme acetylcholinesterase (AChE), leading to accumulation of ACh at synapses. Furthermore, ACh and its agonists as well as AChE and its antagonists are involved in the generation of REM sleep (Amatruda et al., 1975; Gillin et al., 1985; Velazquez-Moctezuma et al., 1991). For example, evidence suggests that REM sleep is controlled by cholinergic neurons in the brainstem (Baghdoyan, 1997; Hernandez-Peon, Chavez-Ibarra, Morgane and Timo-Iaria, 1963), and studies have observed that microinjection of the ACh agonist carbachol in the pontine area of the brainstem results in REM sleep in both humans and animals (Amatruda et al., 1975; Baghdoyan, 1997). Administration of galantamine has been associated with increased phasic activity and shortened REM latency (Riemann et al., 1994). The increased frequency of lucid dreams associated with AChEls could therefore plausibly be related to its effects on cholinergic receptors

during REM sleep, leading to longer, intensified REM periods with increased phasic activity, which, as noted above, has been found to be associated with lucid dreams (LaBerge et al., 1981a).

Beyond intensification of REM sleep/phasic activation, AChEls might also directly act on cognitive processes associated with lucidity and their neural underpinnings. One key question is whether AChEIs could facilitate lucid dreaming through increasing activation within the network of frontopolar-temporo-parietal areas observed in the neuroimaging studies of Dresler et al. (2012) and Baird et al. (2018a). The relationship between cholinergic modulation and frontoparietal activation is complex and depends on the task context and population under study (see Bentley, Driver and Dolan, 2011 for a review). However, pro-cholinergic drugs in general tend to increase frontoparietal activity in conditions in which these areas show low baseline activation, which is thought to reflect increased attentional-executive functions (Bentley et al., 2011). Given that frontoparietal activity is typically suppressed during REM sleep (Braun et al., 1997; Maquet et al., 1996), it is plausible that AChEIs could increase activation within this network during REM sleep dreaming.

Notably, AChEls are also prescribed to manage the cognitive symptoms of Alzheimer's disease (Koontz and Baskys, 2005). Theoretically, another way that AChEls could facilitate lucid dreams is therefore through their effect on memory. For example, AChEIs could enhance the ability to remember to recognize that one is dreaming (a form of prospective memory), which is the core of the MILD technique for lucid dream induction that participants engaged in during the study by LaBerge, LaMarca and Baird (2018b). However, evidence for cognition enhancing effects of AChEls in healthy subjects, particularly for single doses, is sparse (Dresler et al., 2018), rendering the theoretical possibility of a memory-mediated effect on dream lucidity unlikely. Overall, it remains unclear whether galantamine also exerts a direct influence on cognitive processes associated with lucidity, and the MILD technique in particular, or whether it merely optimizes the physiological conditions for such techniques.

One last possibility is that AChEIs could influence lucidity indirectly by affecting other neuromodulators. For example, evidence suggests that AChEls also increase systemic norepinephrine and dopamine (Cuadra, Summers and Giacobini, 1994; Giacobini, Zhu, Williams and Sherman, 1996). It is therefore possible that the increase in lucid dreams associated with AChEI might instead be directly linked to aminergic

modulation that occurs as a result of the increases in ACh. Additional research will be needed to understand how the neuromodulatory changes caused by AChEIs stimulate lucid REM sleep dreaming. Studies using Positron Tomography (PET) or pharmaco-fMRI would be valuable to address this issue.

Several other pharmacological substances have been suggested to increase the frequency of lucid dreams, including various types of supplements and drugs (e.g., Yuschak, 2006). For instance, recreational drugs such as alcohol, cocaine and cannabis have been reported to be associated with lucid dreaming. These substances suppress REM sleep (Roehrs and Roth, 2001; Schierenbeck, Riemann, Berger and Hornyak, 2008), which leads to a phenomenon referred to as REM rebound, in which longer REM sleep periods occur following REM sleep suppression (Vogel, 1975). Intensified, prolonged REM sleep as a result of REM rebound could potentially increase lucid dream frequency, particularly in individuals predisposed to have lucid dreams or with prior experience in lucid dreaming. Another example is LSD, since there is some evidence that it can prolong REM sleep periods at some doses (Muzio, Roffwarg and Kaufman, 1966), which could potentially be favorable to lucid dreaming. To our knowledge, however, no studies have systematically evaluated whether these substances, or other substances not reviewed here, increase the incidence of lucid dreams. Furthermore, it is prudent to remain cautious about such claims given the placebo effect. Placebo-controlled studies will be essential to substantiate any purported effects of pharmacological substances on lucid dreaming.

6. Induction of lucid dreams with transcranial electrical brain stimulation

Two studies have attempted to induce lucid dreams through transcranial electrical stimulation of the frontal cortex during REM sleep. Stumbrys, Erlacher and Schredl (2013b) tested direct current stimulation and Voss et al. (2014) tested alternating current stimulation. We review each of these studies in turn.

6.1 Effects of frontal transcranial direct current stimulation on lucid dreaming

In an investigation of the effect of transcranial direct current stimulation (tDCS) on lucid dreaming, Stumbrys et al. (2013b) applied either tDCS or sham stimulation (counterbalanced across nights) over a frontolateral scalp region in 19 participants. Stimulation was delivered through two pairs of electrodes with anodes at positions F3 and F4, respectively, and cathodes at the supraclavicular area of the same side. Stimulation was applied during all REM sleep periods except the first for two consecutive nights in the sleep laboratory. In total, 109 stimulations were performed, after which participants were awakened to complete a dream report.

Compared to sham stimulation, tDCS resulted in a small numerical increase in self-ratings of the unreality of dream objects as assessed by the Dream Lucidity Questionnaire (DLQ; Stumbrys et al., 2013b). Post-hoc analyses revealed that this effect was seen only in subjects with a high baseline frequency of lucid dreams, but not in participants with little or no lucid dreaming experience. However, tDCS did not significantly increase the number of dreams rated by judges to have a clear indication of lucidity: seven dreams in total, three from sham stimulation and four from tDCS stimulation. Furthermore, only one lucid dream in total was confirmed by eye signaling, which was in the stimulation condition. Given a difference of only one lucid dream between stimulation and sham conditions for either of these two assessment criteria, overall frontal stimulation with tDCS as tested in this study does not appear to be a reliable method for inducing lucid dreams.

6.2 Effects of frontal transcranial alternating current stimulation on lucid dreaming

A study by Voss et al. (2014) reported a more pronounced increase in lucid dreaming by applying transcranial alternating current stimulation (tACS) in the gamma frequency

band over the frontal cortex. The study tested 27 participants for up to four nights in the sleep laboratory. tACS was applied for 30 seconds after two minutes of uninterrupted REM sleep to frontolateral scalp regions at several different frequencies (2, 6, 12, 25, 40, 70 or 100 Hz or sham stimulation). Stimulation was delivered through two pairs electrodes with anodes at positions F3 and F4, respectively, and cathodes over the mastoids close to TP9 and TP10, respectively. Participants were then awakened and completed a dream report and the Lucidity and Consciousness in Dreams (LuCiD) scale (Voss et al., 2013). The LuCiD scale measures dream content on several different factor-analytically derived dimensions, including insight, control, thought, realism, memory, dissociation and affect.

The authors reported they were able to induce lucid dreams with a success rate of 58% with 25 Hz stimulation and 77% with 40 Hz stimulation. However, it is important to note how lucid dreams were classified in this study: instead of assessment of lucid dreams with eye signaling, self-report, or through statistical analysis of judges' ratings of dream reports, as in Stumbrys et al. (2013b), dreams were assumed to be lucid if subjects reported "elevated ratings (>mean + 2 s.e.) on either or both of the LuCiD scale factors insight and dissociation". While dissociation scores (i.e., "seeing oneself from the outside" or a "3rd person perspective") have been found to be increased in lucid dreams before (Voss et al., 2013), dissociation in the sense of adopting a 3rd person perspective has never been considered a defining feature of lucid dreams per se (e.g., DeGracia and LaBerge, 2000; Gackenbach and LaBerge, 1988; Green, 1968; LaBerge, 1985, 1990). It is therefore controversial to classify dreams as lucid based solely on higher ratings of dissociation.

The insight subscale corresponds more closely to the standard definition of lucid dreams adopted by other researchers as well as the general public. Mean ratings in the insight subscale increased from approximately 0.1-0.2 in the sham stimulation to 0.5-0.6 in the 25 Hz or 40 Hz stimulation conditions, similar to the effects in the dissociation subscale, which was approximately 0.9-1.2. However, the scale anchors ranged from 0 (strongly disagree) to 5 (strongly agree), indicating that, on average, in both the 25 Hz and 40 Hz stimulation conditions, participants disagreed that their dreams had increased insight. Moreover, in the original validation of the LuCiD scale, non-lucid dreams scored on average at 0.3 or 0.8 on the insight scale (depending on whether the assessment was laboratory or survey based), whereas lucid dreams scored at 3.2 or 3.5

(Voss et al., 2013). Thus, though significantly stronger in relation to sham simulation, mean responses of 0.5-0.6 in the 25 and 40 Hz stimulation conditions were much lower than the values reported for lucid dreams in the validation study, and still on the non-lucid (disagree that the dream contains insight) end of the scale spectrum in absolute values. Overall, therefore, the results of this study appear to be comparable to the tDCS finding of Stumbrys et al. (2013b) in that prefrontal tACS gamma band stimulation induced numerical increases in some measures of dream content, but does not appear to have lead to increases in the number of lucid dreams when defined in the traditional sense of being aware of dreaming while dreaming.

6.3 General discussion of induction of lucid dreaming with electrical brain stimulation

In summary, studies examining induction of lucid dreams with electrical brain stimulation (tDCS and tACS) have thus far observed intriguing effects on dream cognition, but a method for reliably inducing lucid dreams by electrical stimulation of the brain is still yet to be identified. We think this is a particularly interesting direction for upcoming work. Future studies might consider stimulating a wider number of brain areas and different types of stimulation. For example, transcranial magnetic stimulation (TMS) is another method of noninvasive brain stimulation that could be used to attempt to induce lucid dreams that has not yet been examined (Noreika, Windt, Lenggenhager and Karim, 2010; Mota-Rolim et al., 2010). In contrast to tACS or tDCS, high-frequency repetitive TMS (rTMS) stimulation sequences can be used to increase neuronal excitability in focal areas of the cortex (e.g., Hallett, 2007). Several practical challenges with application of TMS during sleep include the auditory artifacts produced by the TMS coil as well as tactile sensations on the scalp during stimulation, which may lead to awakenings. However, noise masking has been used to prevent subjects from hearing the clicks associated with TMS pulses (e.g., Nieminen et al., 2016), and scalp sensations could be decreased, for example, by reducing stimulation intensity or potentially by application of a topical anesthetic cream.

Studies of electrical brain stimulation have also thus far only tested a small part of the parameter space and there are many other stimulation protocols that appear to be worth evaluating. For example, given the neuroimaging results reviewed above, synchronous frontoparietal tACS—in which synchrony is increased between frontal and

parietal regions by simultaneous stimulation of these regions (e.g., Violante et al., 2017)—is another stimulation method that would be interesting to examine. Finally, as noted above, these methods are likely to maximize their effect when participants are trained in at least the minimal mental set for lucid dream induction, which was not done in either the study by Voss et al. (2014) or Stumbrys et al. (2013b).

7. Lucid dreaming as a methodology for the cognitive neuroscience of consciousness

Psychophysiological studies of non-lucid dreams have been used to study neural correlates of conscious experiences during sleep (Perogamvros et al., 2017; Siclari et al., 2017). However, there are significant limitations to this approach. Specifically, while methods such as "serial awakenings" are useful for contrasting the global presence vs. absence of dream experience during sleep (Siclari et al., 2017), it is an inefficient method to collect data on specific conscious contents during sleep. For instance, given that non-lucid dreamers usually have no control over their dream content, these studies typically employ a shot-in-the-dark approach, in which large numbers of sleep recordings are made and small subsets of data in which the content of interest appears by chance are extracted. Additionally, and perhaps most importantly, establishing temporally precise correlations between retrospective dream reports and physiological measurements is a substantial challenge. Lucid dreaming provides ways to overcome several of these challenges. For example, as mentioned above, lucid dreamers can conduct specific tasks within REM sleep dreams, enabling experimental control over dream content. Furthermore, as noted, participants can be trained to time-stamp the onset and offset of particular content or actions with eye movement signals, which provides a way to obtain more precise correlations between conscious experiences and physiological measurements during sleep (Dresler et al., 2011; LaBerge, 1990; LaBerge, Greenleaf and Kedzierski, 1983). In this section we briefly summarize several recent studies that illustrate how lucid dreaming can be used as a methodology in the cognitive neuroscience of consciousness.

7.1 Activation of sensorimotor cortex during dreamed movement in REM sleep

Dresler et al. (2011) used lucid dreaming to test whether a motor task performed during dreaming elicits neuronal activation in the sensorimotor cortex. Participants made a series of dreamed left and right hand clenches in their lucid dreams and marked the start and end of the sequences of clenches with LRLR eye movements. Specifically, participants clenched their left hand in the dream ten times, then signaled LRLR, then clenched their right hand in the dream ten times, then signaled LRLR, and continued repeating this sequence for as long as possible until awakening. Additionally,

participants performed an executed hand-clenching task as well as imagined hand-clenching task during wakefulness for comparison. Six participants with prior experience in lucid dreaming participated in the study. Of these, two participants succeeded in performing the task during lucid REM sleep: one under simultaneous EEG-fMRI (in two different signal-verified lucid dreams) and one under combined EEG-near-infrared spectroscopy (NIRS) recording (again in two different signal-verified lucid dreams).

Contrasting left vs. right fist clenches, increased BOLD signal in the contralateral sensorimotor cortex was observed in both lucid dreams of the fMRI experiment, as well as in waking and imagination conditions. Compared to executed hand clenches during wakefulness, however, BOLD signal increases during dreaming in contralateral sensorimotor cortex were more localized, which could indicate either weaker activation or more focal activation of hand areas only. BOLD signal fluctuations during dreaming were approximately 50% of those observed for executed hand clenches during wakefulness. Correspondingly, the NIRS data showed increased oxygenated hemoglobin and decreased deoxygenated hemoglobin in the contralateral sensorimotor cortex during dreamed hand clenching. This hemodynamic response was also observed in the supplementary motor area, which is involved in the timing, monitoring and preparation of movements (Goldberg, 1985). This differed with the fMRI data in which no significant differences in the supplementary motor area were found. Interestingly, during dreamed hand clenching, hemodynamic responses were smaller in the sensorimotor cortex but of similar amplitude in the supplementary motor area when compared to overt motor performance during wakefulness.

Overall, these data suggest that the pattern of brain activation observed during dreamed motor execution overlaps with motor execution during wakefulness. However, given the different patterns of activation, the data may also suggest that the interactions between the supplementary motor area, somatosensory and sensorimotor cortex differs between REM sleep dreaming and waking. The authors suggest that the reduced activation in sensorimotor cortex could be due in part to the lack of sensory feedback as a result of REM sleep atonia. However, given that this study consisted of two case reports—one for each imaging modality—the data should be interpreted cautiously. Clarification of the neural correlates of dreamed motor activity, as well as clarification of any differences in this network compared to overt motor performance, awaits larger-

scale group-level studies. Nevertheless, this experiment provides a proof-of-concept that neuroimaging of specific dream content can be accomplished using lucid dreaming as a methodology.

7.2 Voluntary control of central apneas during REM sleep dreaming

A similar type of experiment was recently undertaken by Oudiette et al. (2018) to examine respiratory behavior during REM sleep dreaming. Specifically, the researchers used lucid dreaming to investigate whether irregular breathing during REM sleep has a cortical origin and whether such breathing patterns can reflect the mental content of dreams. This research follows up on a study from LaBerge and Dement (1982) which observed tachypnea during volitional rapid breathing and apnea during voluntary breath holding during lucid REM sleep dreaming. Polysomnography and respiration were recorded during early morning naps from 21 patients with narcolepsy who reported frequent lucid dreams. Participants were instructed to modify their dream scenario so that it involved vocalizations or an apnea (e.g., diving under water)—two respiratory behaviors that purportedly require a cortical control of respiration (McKay, Adams, Frackowiak and Corfield, 2008). Participants signaled the onset of lucidity as well as the start and end of the respiration tasks with LRLR eye movement signals. Participants also performed the task during waking by actually executing the respiratory task as well as during waking imagination. In total, 32 lucid REM sleep naps were included in the analysis. Physiological data was scored for the presence of central apneas, which were defined as “cessation of nasal flow for more than 10 s in the absence of cyclic thoracic and abdominal movements” as well as preparatory breaths preceding the central apnea.

In 16 out of the 32 signal-verified lucid dreams, the physiological data showed the expected respiratory behavior (e.g., a central apnea, in some cases including preparatory breaths), which was appropriately marked by LRLR signals and confirmed by dream reports of becoming lucid and executing the task. In the remainder of cases, participants either failed to control the dream (N=2) or appropriately execute the LRLR signals (N=2), or there was incongruence between the report and the data (participants either did not recall performing the task (N=8) or there was no physiological evidence of the task despite a report of completing the task (N=4)). As the authors discuss, the incongruences between reports and physiological data may in part have been

attributable to the fact that reports were obtained at the end of the 30-minute nap sessions rather than awakening subjects directly after completing the task. Thus, in some cases this resulted in a long delay between the dream task and the report. Despite the presence of these ambiguous cases, overall the data show that voluntarily control of central apneas during REM sleep occurred in a majority of participants.

As the authors discuss, the pons is hypothesized to regulate cessation of breathing, which assimilates input from supra-brainstem structures and inhibits medullary respiratory neurons (McKay et al., 2008). Thus, they conclude that the fact that voluntary control of central apneas is possible during REM sleep suggests that this cortico-pontine drive is preserved during REM sleep. However, it remains unclear from this study to what extent non-lucid dreams containing this type of mental content (for example, diving under water), would result in central apneas. Indeed, as the authors note, some participants reported that they voluntarily held their breath when diving under water in the dream even though they did not feel that they had to. In general, it remains unclear whether central apneas as observed in the study are linked to the voluntary control of respiration enabled during lucid dreams in particular or whether they systematically occur in dreams with this type of content/scenario. In either case, however, the data support the conclusion that voluntarily control of central apneas is possible during REM sleep.

7.3 Smooth pursuit eye movements during visual tracking in REM sleep dreaming

One last recent example of the use of lucid dreaming as methodology in cognitive neuroscience is provided by LaBerge, Baird and Zimbardo (2018a), who addressed the question of whether smooth pursuit eye movements (SPEMs) occur during tracking of a slowly moving visual target during lucid REM sleep dreaming. Seven participants with high reported dream recall and frequency of lucid dreams participated in the study. Participants marked the moment of lucidity as well as the initiation and completion of the tracking tasks with LRLR eye movement signals. After making the LRLR signal, participants performed one of two eye-tracking tasks: circle tracking or one-dimensional movement tracking on the horizontal meridian. For each tracking task, participants followed the tip of their thumb with their gaze as they traced the pattern. Participants performed the tracking tasks in three conditions: 1) lucid REM sleep dreaming

("dreaming"), 2) awake with eyes open ("perception") and 3) tracking the imagined movement while awake with eyes closed ("imagination"). Eye movements were recorded with direct current EOG and subjected to a validated algorithm that classifies saccades, fixations and smooth pursuit eye movements (Komogortsev and Karpov, 2013).

The results revealed that intentional slow tracking of visual motion (of both circles and lines) during lucid REM sleep dreams results in SPEMs. Pursuit eye movements in REM sleep dreams did not significantly differ from pursuit during waking perception, and both were characterized by high pursuit ratios and low saccade rates. In contrast, tracking in imagination was characterized by low pursuit with frequent saccadic intrusions. A Bayesian classification model that included pursuit ratio and saccade rate discriminated both REM sleep dreaming and perception from imagination with greater than 98% accuracy.

Together, these findings help to address several broad questions within cognitive neuroscience and sleep research. First, the data provide empirical evidence for a difficult to test question that has been asked at least since Aristotle: "Are dreams more like perception or imagination?" (Nir and Tononi, 2010). Based on the smooth tracking behavior, the findings of this study suggest that, at least in this respect, the visual quality of REM sleep dream imagery is more similar to waking perception than imagination. Second, the findings help to address a longstanding question in the psychophysiology of sleep since the discovery of REM in the 1950's: whether the eye movements of REM sleep track the gaze of the dreamer—the so-called "scanning hypothesis" (for a review, see Arnulf, 2011). By demonstrating that individuals can trace circles and lines with their gaze while in EEG-verified REM sleep, which can be recorded with EOG, the data provide unique evidence that shifts in the perceived gaze direction in dreams give rise to the appropriate corresponding eye movements. This is consistent with the view that a subset of eye movements during REM sleep are linked to the direction of subjective gaze during dreams. Lastly, the results also provide a novel source of data for a central research question on the topic of smooth pursuit in humans and non-human primates that dates back at least forty years. Specifically, an enduring question has been whether a physical stimulus and/or retinal image motion is necessary to drive the neural circuitry of smooth pursuit (Spering and Montagnini, 2011). By demonstrating that sustained SPEMs can be elicited in the absence of visual input to the

cortex, as is the case during REM sleep, the findings provide strong evidence that neither a physical motion stimulus nor readout of retinal image motion are necessary for SPEMs.

7.4 General discussion of lucid dreaming as experimental methodology

Altogether, these studies illustrate the potential of lucid dreaming as an experimental methodology for the study of consciousness in general and REM sleep dreaming in particular. The fact that lucid dreamers can exercise volitional control over their actions while dreaming and conduct experiments from within EEG-verified REM sleep dreams opens up the ability to perform experiments that would otherwise be difficult or impossible to conduct. This methodology also has the potential to more efficiently target specific research questions. As discussed above, lucid dreaming enables both experimental control over the content of dreams as well as a way to establish precise psychophysiological correlations between the contents of consciousness during sleep and physiological measures in a way that is not possible in studies of non-lucid dreaming. In sum, lucid dreaming provides a methodology that allows for new ways of studying the relationship between consciousness and neurophysiological processes, and, as the above studies highlight, shows emerging potential for research on consciousness in sleep science.

8. Clinical cognitive neuroscience of lucid dreaming

Research on lucid dreaming is not only relevant to the neuroscience of consciousness but could also have clinical implications. While a detailed treatment of the clinical applications of lucid dreaming is beyond the scope of this review (see e.g., Garfield, Fellows, Halliday and Malamud, 1988), here we briefly note several interesting applications and future directions with particular relevance for cognitive neuroscience.

8.1 Lucid dreaming in the treatment of persistent nightmares

The most researched application of lucid dreaming is to treat recurring nightmares (Abramovitch, 1995; Holzinger, Klösch and Saletu, 2015; Spoormaker and Van Den Bout, 2006; Tanner, 2004; Zadra and Pihl, 1997). Lucid dreaming was included by the American Academy of Sleep Medicine in their therapy suggestions for nightmare disorder (Morgenthaler et al., 2018). Conceptually, the idea is that becoming lucid during a nightmare should allow the dreamer to realize the content of the nightmare is not real and thus has no reason to be afraid, and to be able to exert control over the dream and/or work with the psychological content of the nightmare (LaBerge and Rheingold, 1990; Mota-Rolim and Araujo, 2013). Neurocognitive models of nightmare generation suggest a hyper-responsivity of the amygdala coupled with a failure of prefrontal regions to dampen this activation (Levin and Nielsen, 2007). Studying the influence of lucidity on nightmare resolution could therefore provide an interesting opportunity to study top-down regulation of amygdala activation, perhaps through reactivation of prefrontal regions (Dresler et al., 2012).

A case study (Zadra and Pihl, 1997), and one small, controlled pilot-study (Spoormaker and Van Den Bout, 2006) have found that lucid dreaming therapy was effective in reducing nightmare frequency. Also a study of 32 patients who suffer from frequent nightmares found a slight advantage of lucid dreaming as add-on to Gestalt therapy compared to the latter alone (Holzinger et al., 2015). In contrast, a larger online study did not find any additional effect of lucid dreaming therapy as an add-on to other cognitive-behavioral techniques, such as imagery rehearsal therapy (Lancee, Van Den Bout and Spoormaker, 2010), although low power and high dropout rates (>70%) limited the scope of the conclusions. Controlled experiments with larger sample sizes are needed to further evaluate the potential usefulness of lucid dreaming for the

treatment of recurring nightmares and related disorders, as well as to examine the neural mechanisms of these interactions.

8.2 Lucid dreaming and narcolepsy

Consistent with a potential beneficial role of lucid dreaming in recurring nightmares, patients with narcolepsy report that becoming lucid provides psychological relief (Rak et al., 2015). Generally, narcolepsy patients experience longer, more complex, and more vivid dreams and more frequent nightmares than healthy subjects (Mazzetti et al., 2010). Moreover, patients with narcolepsy report higher reflective consciousness within dreams (Fosse, 2000) and accordingly a considerably increased lucid dreaming frequency (Dodet et al., 2014; Rak et al., 2015). The mechanisms for this increased lucid dreaming frequency are not entirely understood yet: nightmares often lead to the insight into the current dream state, however it is currently unclear if an increased nightmare frequency causes an increased lucid dreaming frequency in narcolepsy, or if the strongly increased frequency of REM sleep in narcolepsy affects nightmares and lucid dreaming independently. In particular sleep onset REM (SOREM) episodes appear to prompt lucid dreams (Dodet et al., 2014), which is in line with the dream lucidity-enhancing effects of the early morning "Wake-Back-to-Bed" procedure in healthy subjects. Of note, patients with narcolepsy make a clear distinction between their experiences of lucid dreaming and sleep paralysis (Dodet et al., 2014), and the increased lucid dreaming frequency in this patient group does not appear to be associated with medication use (Rak et al., 2014). It has been speculated whether, potentially as a result of a considerably increased lucid dreaming experience, narcoleptic patients might exhibit different neurophysiological correlates of lucid dreaming (Dodet et al., 2014).

8.3 Lucid dreaming as a model of insight in psychiatric conditions

Another area in which research on lucid dreaming might be applied is in psychiatric disorders in which patients suffer from lack of insight into their state. Indeed, there is considerable overlap between preliminary findings of brain regions related to insight into the dream state and brain regions impaired in psychotic patients with disturbed insight into their pathological state (Dresler et al., 2015; Mota, Resende, Mota-Rolim,

Copelli and Ribeiro, 2016). The concept of insight is becoming an increasingly important area in schizophrenia research (e.g., Baier, 2010), for example, where between 50 and 80% of patients diagnosed with schizophrenia have poor insight into the presence of their disorder (Lincoln, Lüllmann and Rief, 2007). Evidence suggests that poor insight in turn leads to more relapses, re-hospitalizations and poorer therapy success (Mintz, Dobson and Romney, 2003). With regard to dreaming, lack of insight into the current state characterizes almost all dream experiences, with the exception of lucid dreaming. Thus, lucid dreaming could potentially be used as a model for at least some cognitive dimensions of insight (David, 1990). In this context it is interesting to note that historical approaches to psychosis used the term *lucid* to denote the awareness of the patient into his or her illness (Berrios and Markova, 1998). At the current time, these ideas remain highly speculative, but this appears to us to be an area worthy of additional research.

8.4 Using lucid dreaming to establish brain activity markers of self-awareness

Another potential clinical application of research on lucid dreaming is in the development of neuroimaging-based diagnostic markers of awareness. Such measures have the potential to improve the diagnosis and monitoring of patients who are unresponsive due to traumatic injury, aphasia, motor impairment, or other physical limitations. Neurobiological studies of lucid dreaming could provide information relevant to development of these brain activity markers since, in addition to assessing a patient's capacity for primary consciousness (i.e., if a patient can see, hear or experience pain), an important clinical goal is to assess whether patients, who may nevertheless be unresponsive via behavioral assessment, are aware of themselves and their state (Laureys, Perrin and Brédart, 2007). This information is critical to making appropriate therapeutic choices and determining prognosis (Laureys et al., 2007). Research indicates that the bedside behavioral assessment of such patients is challenging and has a high rate of misdiagnosis (over 40% according to some studies) (Laureys, Owen and Schiff, 2004; Schnakers et al., 2009). Accordingly, identification of a reliable brain activity marker of a patients' capacity for self-awareness—which could be informed by studying the changes in brain activity between lucid and non-lucid REM sleep dreaming—is an important goal. Research on lucid dreaming has the potential to contribute a valuable source of data to this question since, as noted above, the contrast between lucid and

non-lucid REM sleep is perhaps the only one currently known in which a global loss and recovery of self-awareness can be contrasted within the same vigilance state in healthy individuals.

9. The measurement of lucid dreaming in cognitive neuroscience research

In this last section, we review methodological issues and strategies in the measurement of lucid dreaming in cognitive neuroscience research. We review the validation of lucid dreaming both physiologically and with questionnaires, and discuss best-practice procedures to investigate lucid dreaming in the sleep laboratory.

9.1 Eye signaling methodology

The eye signaling methodology is the gold standard in lucid dreaming research, as it allows for objective confirmation of lucid dreams through the execution of pre-agreed upon sequences of eye movements recorded with the EOG during EEG-verified REM sleep (Figure 1). While there are some types of research studies (e.g., field studies) where eye signaling is not possible or not applicable, we recommend considering this method as the standard practice for laboratory-based studies of lucid dreaming. In general, most recent studies have adhered to this convention; however, there is some confusion about how to properly instruct participants in executing the eye movements, which has resulted in studies implementing several different variations of the method. For instance, while some experimenters instruct participants to move their eyes left and right (e.g., Dresler et al., 2012), in other cases participants have been instructed to "scan the horizon" from left to right (Dodet et al., 2014).

Differences in eye signaling instructions could partly account for the varying degrees of effectiveness in objectively identifying the eye signals across studies. For example, Dodet et al. (2014) reported that while three control participants and twelve narcoleptic patients reported making the eye signal in overnight EEG recordings, none of these could be objectively identified, and only about half of reported signals could be unambiguously identified from nap recordings. While it is possible that some of these instances represent cases where participants misremembered or misreported a lucid dream, it is likely that this high rate of ambiguous eye movement signals is partly attributable to the specific instructions that were given. Similarly, while the exact instructions provided to participants was not described, Voss et al. (2009) stated that they were unable to obtain reliable eye signals using the standard signaling method and therefore attempted to develop a novel signaling method employing two sets of eye-signals separated by a pause. Again, suboptimal results could plausibly be due to the

instructions provided and the way the eye signals were executed by participants in the study.

One reason for the confusion and diverse instructions given for the eye signaling methodology is that a standardized set of instructions for making the signals has never been published. Here we report a simple set of instructions adapted from LaBerge et al. (2018a) that has been reported to yield nearly 100% correspondence between subjective reports of eye signals and objective EOG recordings. The instruction is as follows:

> *When making an eye movement signal, we would like you to look all the way to the left then all the way to the right two times consecutively, as if you are looking at each of your ears. Specifically, we would like you to look at your left ear, then your right ear, then your left ear, then your right ear, and then finally back to center. Make the eye movements without moving your head, and make the full left-right-left-right-center motion as one rapid continuous movement without pausing.*

Looking at the ears is one of the critical aspects of the instruction, as it encourages full-scale eye movements without corresponding head movements. These extreme and rapid consecutive eye movements make the signals easy to discern in the horizontal EOG time series. The signals are of higher amplitude than typical REMs, and have a distinctive shape (Figure 1) which can be identified with template matching (LaBerge et al., 2018a). To optimize execution of the eye signals, participants are given the opportunity to practice the signals in the laboratory while awake and connected to the EOG, with an opportunity to view the signals and receive feedback from the experimenter. Additionally, participants practice making the eye signals in any lucid dreams they have at home in the weeks leading up to the sleep laboratory visit, in order to gain experience with executing the signals.

9.2 Questionnaire assessment of lucid dreaming

A related issue concerns how the phenomenology of lucid dreaming should optimally be assessed in cognitive neuroscience research. Several questionnaires have been developed to assess changes in conscious experience during dreaming. For instance, the Metacognitive, Affective, Cognitive Experience questionnaire (MACE; Kahan, LaBerge, Levitan and Zimbardo, 1997; Kahan and Sullivan, 2012) was designed

to assesses the monitoring and regulation of both cognitive and affective experience during both wakefulness and sleep. In its latest form, it consists of ten items, including four self-monitoring questions, three questions on self-reflective consciousness and three questions that target self-regulatory behaviors. Another measure, the Dream Lucidity Questionnaire (DLQ; Stumbrys et al., 2013b), is designed to assess different aspects of lucidity within dreams. It consists of ten items, scored on a 5-point scale: 0 – not at all, 1 – just a little, 2 – moderately, 3 – pretty much, 4 – very much. The DLQ evaluates different types of awareness (awareness of dreaming, awareness that the physical body is asleep, awareness that dream characters and objects are not real, awareness of different possibilities) and control (deliberately choosing an action, changing dream events, dream characters, dream scenes, breaking physical laws). In a similar manner, the Lucidity and Consciousness in Dreams scale (LuCiD, Voss et al., 2013) includes eight subscales, derived from factor analysis of a sample of lucid and non-lucid dream reports, which assess various dimensions of dreaming experience and cognition: 1) Insight, 2) Control, 3) Thought, 4) Realism, 5) Memory, 6) Dissociation, 7) Negative emotion, and 8) Positive emotion.

All three questionnaires provide measures for evaluating how some content dimensions might differ across lucid and non-lucid dreams. However, as mentioned, questionnaires such as these are not sufficient to objectively establish whether a participant had a lucid dream. It is worth noting that only the DLQ in its first question ("I was aware that I was dreaming") directly queries whether the dreamer was lucid, and the MACE was never intended for this purpose. Most studies using the DLQ and LuCiD questionnaires have collected a dream report from participants in addition to the questionnaire responses. We would like to emphasize the importance of this point: Instead of relying on the inference of lucidity solely from questionnaire measures of dream content, accurate assessment of lucidity can be facilitated by, in addition to using the eye signaling method, collecting a full dream report from participants. In making the dream report, participants are typically asked to describe in as much detail as possible the narrative of the dream, including the sequence of events and any thoughts, feelings or sensations that they experienced. In dream reports for lucid dreams, the participant is also asked to include a specific description of how they became lucid in the dream (for example, by noticing an oddity of an event, action or person in the dream, which is also

known as “dream sign”) and also to explicitly note any eye signals made at the appropriate instances in the dream narrative.

Following the collection of a full open-ended dream report, further confirmation of lucidity and eye signaling can be accomplished with simple follow-up questions, including, for example: 1) “Were you aware of the fact that you were dreaming while you were dreaming?” (YES/NO), If YES: 2) “How confident are you that you became lucid?” (0-4 scale), 3) “Did you have a wake-initiated lucid dream (WILD) or a dream initiated lucid dream (DILD)?” (DILD/WILD), 4) “Did you make the eye movement signal to indicate that you became lucid?” (YES/NO), 5) “Please briefly describe how you became lucid.” These example questions are not meant to be exhaustive, but they illustrate the types of questions that in our view are useful to obtaining an accurate and thorough assessment of lucid dreams following collection of a full dream report.

9.3 Measurement of individual differences in lucid dreaming

Another related question facing researchers is how to measure individual differences in lucid dream frequency, which has been done in inconsistent ways, and could be improved in future research. A method used in several studies consists of an 8-point scale that asks participants to self-rate the frequency with which they experience lucid dreams, ranging from “never” to “several times per week” (Schredl and Erlacher, 2004; Mota-Rolim et al., 2013). While this method provides a straightforward coarse assessment of an individual’s estimated frequency of lucid dreams that has shown high test-retest reliability (Stumbrys, Erlacher and Schredl, 2013a), a limitation is that it does not measure lucid dream frequency greater than several times per week. The scale could be improved by including additional categories on the higher end of the measure, including, for example, “Every night” and “Multiple times per night.” While individuals who experience lucid dreams on a nightly basis represent a very small percentage of respondents (according to Baird et al. (2018a) approximately one in one thousand), optimally this kind of instrument would enable a researcher to distinguish respondents that have lucid dreams once or several times a week from those that have them every night or multiple times per night. Indeed, these “virtuoso” lucid dreamers represent perhaps one of the most interesting populations for cognitive neuroscience studies of individual differences in lucid dreaming (Baird et al., 2018a).

A related method is to query the number of lucid dreams reported in a given period of time (i.e., the last 6 months), which has the advantage of asking participants to report a specific number rather than a coarse estimate. In principle, this method has the potential to be more accurate and to capture increased variance. However, some participants, particularly those with high lucid dream rates, may not be able to recall all instances of their lucid dreams within the requested time interval, and may therefore resort to heuristics when answering the question, making it akin to simply asking participants to report frequency using a multiple-choice scale. Furthermore, this method may not accurately assess lucid dream frequency over longer periods of time – i.e., an individual may normally experience lucid dreams frequently, but has not experienced them as frequently during the last queried interval of time. If using this approach, it is therefore advisable to also collect additional estimates of frequency, including the most lucid dream episodes experienced in any 6-month period (e.g., LaBerge et al., 2018b).

A limitation of the above methods is that they do not measure variation in the length or degree of lucid dreams. Indeed, lucid dreams can range from a mere fleeting thought about the fact that one is dreaming followed by an immediate loss of lucidity or awakening, to extended lucid dreams in which an individual is able to engage in sequences of actions (e.g., LaBerge and Rheingold, 1990). Distinguishing between these different "levels" or subtypes of lucid dreams will likely be valuable to understanding observed differences (or lack of differences) in brain structural or functional measures associated with lucid dream frequency. Along these lines, several recent questionnaires have taken first steps to measure individual differences in specific characteristics of lucid dreaming. For example, the Lucid Dreaming Skills Questionnaire (LUSK) measures participants' frequency of several different but inter-related aspects of awareness and control in lucid dreams (Schredl, Rieger and Göritz, 2018). Another questionnaire, the Frequency and Intensity Lucid Dreaming Questionnaire (FILD) queries participants regarding the duration of their lucid dreams and various aspects of dream control, as well as the frequency with which they deliberately attempt to induce lucid dreams (Aviram and Soffer-Dudek, 2018). In addition to providing data for individual differences studies, such questionnaires could also potentially be useful in selecting participants for sleep laboratory experiments of lucid dreaming, for example by selecting participants who report high levels of dream control in addition to frequent

lucid dreams (in order to select participants who are more likely to be able to effectively make the eye signals or engage in experimental tasks during lucid dreams).

For all the above methods, important steps need to be taken to minimize measurement error, particularly to ensure that participants have a clear understanding of the meaning of lucid dreaming (Snyder and Gackenbach, 1988). These include providing participants with a written definition of lucid dreaming, asking participants to provide written examples of their own lucid dreams to ensure clear understanding, as well as additional vetting through participant interviews (Baird et al., 2018a). Ultimately, however, basing the measurement of individual differences in lucid dreaming solely on self-report is not optimal. One way to further validate participant questionnaire responses would be to attempt to physiologically validate at least one lucid dream in the sleep laboratory for each participant. While additional validations such as this could potentially be valuable to incorporate in future studies, it is important to note that the estimated frequency of lucid dreaming would still depend on questionnaire assessment. Thus, approaches such as this do not obviate the reliance on questionnaire assessment.

An intriguing, though ambitious, method for deriving a measure of lucid dream frequency not dependent on questionnaire assessment would be to utilize home-based EEG recording systems to collect longitudinal sleep polysomnography data, from which estimates of lucid dreaming frequency could be derived from the frequency of signal-verified lucid dreams collected over many nights. However, this approach would only measure the frequency of signal-verified lucid dreams, and instances in which participants achieved lucidity but did not make the eye signal due to factors such as awakening, forgetting the intention, or lack of dream control would be missed by this procedure. There are many more points that could be addressed on the topic of questionnaire assessment of lucid dreaming frequency, but an extended analysis of this issue is beyond the scope of the present review.

9.6 General discussion of the measurement of lucid dreaming in cognitive neuroscience

In summary, cognitive neuroscience studies of lucid dreaming have at their disposal a unique set of rigorous methodological tools, including in particular the eye movement signaling method, which allows for the objective validation of lucid dreaming as well as

precise time-stamping of physiological data. However, refinement of the instructions given to participants, as described above, could help further increase the reliability of the technique. Accurate phenomenological assessment of lucidity has been more mixed. In sleep laboratory studies using the eye signaling method, phenomenological reports are important but less critical due to the presence of an objective marker of lucidity. In contrast, in studies without eye signaling, accurate phenomenological assessment of whether an individual was lucid becomes essential, and inadequate or ambiguous measurement of lucidity can undermine the interpretability of the results. Questionnaire measures such as the DLQ (Stumbrys et al., 2013b) or LuCiD scale (Voss et al., 2013) by themselves do not provide an unambiguous assessment of whether an individual had a lucid dream. Cognitive neuroscience research on lucid dreaming would greatly benefit from the further development of improved questionnaire measures for the validation of lucidity and the cognitive and experiential changes that accompany it, as well as further development of standardized measures for quantifying both the frequency and degree of lucidity. Overall, this brief discussion highlights the need for a set of standard operating procedures for both the phenomenological and objective sleep laboratory assessment of lucid dreaming.

10. Conclusion

Despite having been physiologically validated for approximately four decades, the neurobiology of lucid dreaming is still incompletely characterized. Most studies conducted to date have relied on small sample sizes, which limits the generalizability of the findings. Not surprisingly, the results of such underpowered studies are not consistent: almost every EEG study reports changes in spectral power in a different frequency band or brain area. Neuroimaging data on lucid dreaming is even sparser. Currently, there is only one fMRI study contrasting lucid and non-lucid REM sleep and it is a case study. Nevertheless, the results of this study converge with MRI studies that have evaluated individual differences in lucid dreaming frequency. Together, this preliminary evidence suggests that regions of anterior prefrontal, parietal and temporal cortex are involved in lucid dreaming. The involvement of these brain regions in metacognitive processes during the waking state is also in line with these findings.

A primary goal is to develop reliable strategies for making lucid dreaming more accessible. As reviewed above, several studies have explored methods for non-invasive electrical stimulation of the brain as well as pharmacological approaches to lucid dream induction. Electrical stimulation of prefrontal brain areas has resulted in statistically significant but weak increases of dream "insight" ratings, but so far it has not resulted in significant increases in the frequency of lucid dreams. Currently it remains too early to tell how effective (if at all) electrical stimulation of the frontal cortex, or other brain areas, could be for lucid dream induction. Pharmacological induction with agents acting on the cholinergic system, in particular the AChEI galantamine, has shown promising results; however, these findings need to be replicated systematically, and lucid dreams objectively confirmed with polysomnography. Other approaches to lucid dream induction not discussed in the current review, and that do not directly target neural mechanisms, such as cognitive/psychological approaches, also appear promising (Stumbrys et al., 2012). For example, advances in the research of targeted memory reactivation via olfactory or acoustic stimuli during sleep (e.g., Oudiette, Antony, Creery and Paller, 2013) might lead to new strategies for lucid dream induction, together with continued research on stimulating lucid dreams with visual or auditory cues (LaBerge and Levitan, 1995; LaBerge et al., 1988; LaBerge et al., 1981b).

In conclusion, additional studies with larger sample sizes, for example large-scale group-level high-density EEG, MEG, and concurrent EEG/fMRI studies, will be

important next steps toward characterizing the neural functional changes associated with lucid dreaming. For now, a more detailed understanding of the neurobiological basis of lucid dreaming remains an open question for ongoing research. Lucid dreaming shows promise as a useful methodology for psychophysiological studies of REM sleep, with potential applications in both clinical and basic research domains. Perhaps the largest potential of research on lucid dreaming is that it provides a unique method to investigate the neurobiology of consciousness, which remains one of the largest lacunas in scientific knowledge.

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Competing interests

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Figure 1

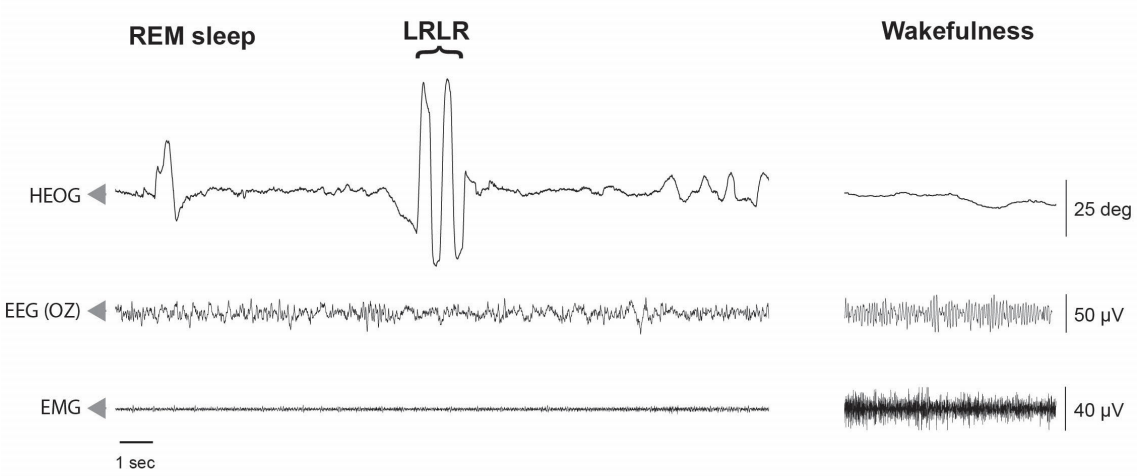

Fig 1. Lucid REM sleep eye movement signaling paradigm. Exemplary left-right-left-right-center (LRLR) eye movement signal during polysomnographcally-verified REM sleep. Participants signal when they realize they are dreaming by rapidly looking all the way to the left (as if looking at their ear) then all the way to the right two times consecutively then back to center without pausing. The LRLR signal is readily discernable in the HEOG, which exhibits a distinctive shape of four consecutive full-scale eye movements of higher amplitude compared to typical REMs. Note high-frequency electroencephalogram (EEG) with theta rhythm (~5 Hz) and lack of alpha at OZ as well as minimal electromyogram (EMG) amplitude due to muscle atonia characteristic of REM sleep (left) compared to wakefulness (right).

Figure 2

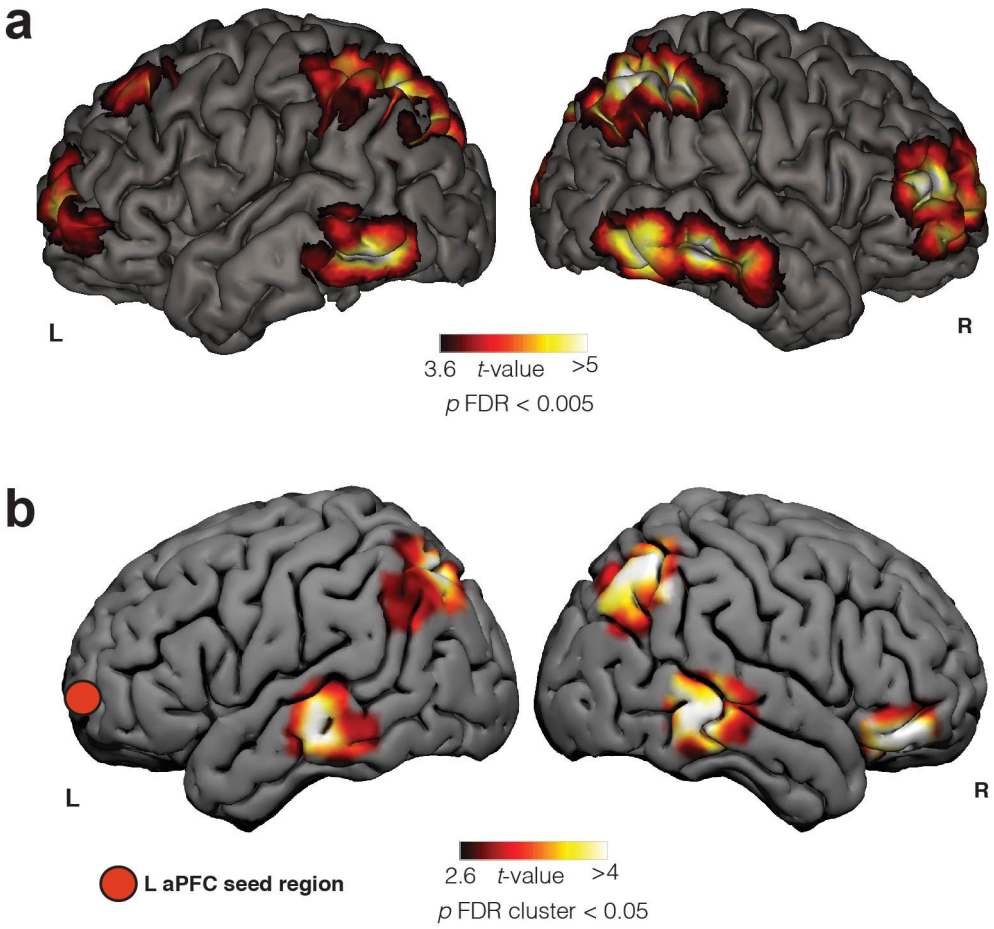

Figure 2. a) Blood-oxygen-level dependent (BOLD) activation in fMRI case study of lucid dreaming (Dresler et al., 2012). Clusters show regions with significantly increased BOLD signal during lucid REM sleep (pFDR < 0.005) in the left lateral hemisphere view (left) and right lateral hemisphere view (right). Increased activity was observed in anterior prefrontal cortex (aPFC), medial and lateral parietal cortex, including the supramarginal and angular gyrus and inferior/middle temporal gyrus during lucid REM sleep contrasted with non-lucid REM sleep. **b) Seed-based resting-state functional connectivity differences between frequent lucid dreamers and controls (Baird et al., 2018a).** To estimate connectivity, spherical regions-of-interest were defined in aPFC based on the peak voxel reported in Dresler et al. (2012) (red circle). Frequent lucid dreamers had increased resting-state functional connectivity between left aPFC and bilateral angular gyrus, bilateral middle temportal gyrus and right inferior frontal gyrus. All clusters are significant at p<0.05, corrected for multiple comparisons at the cluster level.