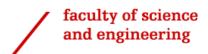
# Lucid Dreaming as Possible Therapy for Psychosis

Bachelor Thesis - Chronobiology

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#### **Abstract**

Lucid dreaming is a state of consciousness in which individuals become aware that they are dreaming and can possibly control the content or events in their dream. The awareness and control in the external world is a lack in psychosis. This review investigates whether lucid dreaming can serve as therapy for psychosis. Lucidity is trainable and can be self-induced, so it is an opportunity to study the brain basis of consciousness. Lucid dreams usually occur in REM sleep and are very intense, but signals to create detailed images are not strong enough. Experimental studies of lucid dreaming are difficult, because it is not easy to arouse lucid dreams in laboratory settings. Methodologies to investigate lucid dreaming are the use of questionnaires, non-invasive methodologies and neuroimaging techniques.

In electroencephalography (EEG), lucid dreaming can be indicated with eye movement signals. Functional magnetic resonance imaging (fMRI) shows a clear difference in activation of multiple brain areas, in particular the prefrontal cortex (PFC) increases in activity during lucid dreaming. Psychotic episodes are characterized by a hypo-function of the PFC. A great diversity of brain areas are involved both in insight deficits in psychosis and lucid dreaming. The most common brain areas seem to be the prefrontal, medial parietal and cingulate cortex. Further experimentation is required to investigate whether the feeling of control during lucid dreaming can be converted to the feeling of better control during psychosis and therefore can help as possible therapy.

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## **Abbreviations**

DLPFC Dorsolateral prefrontal cortex

EEG Electroencephalography

EMG Electromyography
EOG Electrooculogram

fMRI Functional magnetic resonance imaging

PET Positron emission tomography

PFC Prefrontal cortex
POT Scalp potentials
REM Rapid eye movement

tDCS Transcranial direct current stimulation
TMS Transcranial magnetic stimulation

WEC Waking with eyes closed

#### Introduction

Dreaming shows many peculiarities, with delusional thoughts, a bizarre plot, and a complete lack of insight into the fact that we are dreaming. During lucid dreaming, subjects also experience delusional thoughts and bizarre plots, but they become aware of their dreaming state, and they are able to control the dream. Patients with psychotic episodes are more familiar with normal dreaming compared to lucid dreaming. They also suffer with delusional thoughts and accept bizarre experiences as real. To investigate whether lucid dreaming can serve as therapy for psychosis it is important to improve insight in both lucid dreaming and psychosis. In addition, the relationship between dream perception and brain activity should be explored. In this paper, lucid dreaming and psychosis as phenomenon and the neural correlates between lucid dreaming and psychosis will be described. Also the benefits and limitations for the use of lucid dreaming as therapy for psychosis will be discussed.

### **Lucid dreaming**

Lucid dreaming is a state of consciousness in which individuals become aware that they are dreaming and can possibly control the content or events in their dream. To define lucid dreaming as above, there are several criteria. The awareness of the dream state is the starting point to label the dream as lucid dream and is therefore one of the criteria (Gillespie, 1983). Another criterion of lucid dreaming is the possibility of controlling the dream content (van Eeden, 1913). It is thus unclear how lucid dreaming is exactly defined (Erlacher, Schredl, Watanabe, Yamana, & Gantzert, 2008; A. Hobson, 2009; Noreika, Windt, Lenggenhager, & Karim, 2010; Voss, Holzmann, Tuin, & Hobson, 2009). A meta-analysis shows a proportion of 55% of individuals who have experienced at least one lucid dream in their lifetime and 23% reports experiencing lucid dreaming once a month or more. This suggests that the capacity for lucid dreaming is widespread. Nationality does not appear to be an influencing factor in lucid dreaming incidence and can occur everywhere (Saunders, Roe, Smith, & Clegg, 2016). Saunders et al. (2016) also implies that as people age they are increasingly more likely to report having lucid dreams.

LaBerge proved that lucid dreaming is a learnable skill (La Berge, 1980). A way to learn lucid dreaming is telling yourself, before going to sleep, to recognize that you are dreaming by noticing the events of the dream (Voss et al., 2009), but there is a theoretical approach to check lucidity in dreams. The study of Levitan et al. (1993) is based on the idea that the perceptual experiences in dreams arise out of activity in the same brain areas that produce perceptual experience in waking. Therefore, people find it difficult to distinguish dreaming from waking experience. If a brain area is not in a state conducive to the desired experience, physiological constraints on dream perception might occur. For example, because the visual cortex is not active enough during dreams, it might be hard to make a dark dream light. Their study was designed to assess how successful people would be at performing well-defined tasks, such as a light switch task and a mirror task, in lucid dreams. In the light switch task, participants were asked to find a light in their dream, switch it on and off and see what happens. In the mirror task, participants were during dreaming asked to find a mirror and observe their reflection in the mirror. They had to move their hand to their face, watch what their reflection would do and walk through the mirror. These tasks represented a variety of types of influence, ranging from things that are easy to achieve in waking and impossible to achieve in waking. To ensure that participants have done the task once before, they were instructed to try each task in waking prior to attempting the task in lucid dreams. The most notable result is the reluctance of the mirror reflections to show normal images and their fascinating instability in lucid dreams. Selfimage is a psychologically loaded thing, with very complex internal representations, which may account for the strange images. The instability points up the most leading difference between dreaming and waking, because dreams do change. Interesting is whether the perceptual instability results from the lack of confirmed sensory input from the physical senses or from a state of brain peculiar to rapid eye movement (REM) sleep (Levitan & LaBerge, 1993).

Lucid dreaming is useful in experiments, because experienced lucid dreamers can remember presleep instructions and therefore mark their lucid dreams with eye movement signals during the experiment (LaBerge, 2000) that are reflected in the electrooculogram (EOG) (Erlacher & Schredl, 2008). Because lucidity is trainable and can be self-induced, it is an opportunity to study the brain basis of consciousness but also demonstrates how an intervention on a voluntary basis can change those states.

#### **REM sleep and lucid dreaming**

Lucid dreams usually occur in REM sleep (Erlacher, 2005) and arise from non-lucid dreams (LaBerge, 2000). Recent EEG data shows that lucid dreaming is associated with reactivation of brain areas which are normally deactivated during REM sleep (Dresler et al., 2012). To prevent the sleeping body to act out the dreamed action, the brainstem contains a mechanism that makes sure the sleeping body is paralyzed throughout REM sleep (Jouvet, 1979). Despite the paralysis is the brain very active during REM sleep, therefore dreams in that stage are more intensive in quality and quantity (Niedermeyer & da Silva, 2005). The typical phenomenal characteristics of dreaming have frequently been associated with neural activation patterns observed during REM sleep. Higher visual and motor areas show stronger metabolic activity during REM sleep (Braun et al., 2007; Maquet et al., 1996). That is in line with visuomotor hallucinations as the hallmark of typical dreaming (J. A. Hobson, Pace-Schott, & Stickgold, 2000). Also the medial prefrontal cortex, anterior cingulate cortex and amygdala shows increased activity during REM sleep (Braun et al., 2007; Maquet et al., 1996). All these brain areas are involved in emotional processing and mirroring emotions experienced in dreams (J. A. Hobson et al., 2000).

Braun et al. used positron emission tomography (PET) and H<sub>2</sub><sup>15</sup>O to measure cerebral activity and to evaluate regional interrelationships within visual cortices and their projections during REM sleep. REM sleep was associated with selective activation of extra striate visual cortices and an attenuation of activity in the primary visual cortex. Extra striate activity was also associated with a marked reduction of activity in frontal association areas including the dorsolateral prefrontal cortices. The results of Braun et al. suggest that because increased activity in extra striate areas is coupled to decreased activity in the primary visual cortex, firing of the extra striate back-projections may in fact be actively inhibited during REM sleep. So the spontaneous generation of visual images that occurs during REM sleep may be associated with isolated activation of the extra striate cortices. All results together suggest that pathways that mediate the transfer of information between visual cortices and the limbic system may be active during REM sleep. Pathways that mediate transfer of visual information to prefrontal association cortices are not active during REM sleep. REM sleep may represent a state in which the brain develops selective activation of a perceptive network, which is dissociated from primary sensory and association areas at either end of the visual hierarchy that moderate interactions with the external world (Braun et al., 2007).

The prefrontal cortex (PFC) becomes active during lucid dreaming (Holzmann et al., 2014; S. A. Mota-Rolim, Erlacher, Tort, Araujo, & Ribeiro, 2010; Voss et al., 2009). An increase in frontal activity should contribute to lucidity during dreaming (A. Hobson, 2009). Lucid dreaming is characterized by the complete awareness of the true state of the sleeping subject (La Berge, Nagel, Dement, & Zarcone, 1981). Despite this wake-like reflection, lucid REM sleep contains all defining markers of REM sleep (La Berge et al., 1981) and basal dream features such as hallucinations (Windt, 2010). The study of Levitan et al. (1993) found that visual perception during lucid dreaming causes perceptual instability. This can now be confirmed by the study of Braun et al (2007). They showed that transfer of visual information to the PFC is not active during REM sleep. The primary visual cortex is still active, but signals are not strong enough to create detailed images during lucid dreams.

### Subjective and objective methodologies of lucid dreaming

Spontaneous lucidity is quite rare and arises commonly in home settings. Therefore experimental studies of lucid dreaming in the laboratory are difficult because they are not easily transferable. The experiment of Voss et al. (2009) used a group of 20 subjects who claimed that they regularly experienced lucid dreams. It was found that only three subjects achieved, each only one time out of five nights, lucidity in the laboratory. They must therefore conclude that lucid dreaming is fragile and not easy to study in the laboratory.

Finding a good methodology to cover all criteria for defining lucid dreaming is difficult. The most obvious methodology is the use of questionnaires (Ribeiro, Gounden, & Quaglino, 2016). Ribeiro et al. (2016) investigated experimentally whether the type of interrogation formulation influences lucid dreaming. They compared two types of questionnaires widely used in the literature: the first questionnaire contained a definition of lucid dreaming and a frequency question as used by Schredl and Erlacher (2004). The second questionnaire contained two separate questions on two specific lucid dreaming dimensions, one concerned dream control and the other targeted the frequency of dream awareness. A series of questions were common to both questionnaires. There was no significant difference between lucid dreaming frequencies across methodologies (Ribeiro et al., 2016). This could be due to other factors such as forms of parasomnia (Dodet, Chavez, Leu-Semenescu, Golmard, & Arnulf, 2015; Schredl & Erlacher, 2004), age (Stephan, Schredl, Henley-Einion, & Blagrove, 2012), cultural representations toward dream experience (Erlacher et al., 2008) or the dependence of retrospective measurement on memory and meta-cognitive capacity (Aspy, 2016; S. a Mota-Rolim et al., 2013).

A much-discussed topic is the use of noninvasive methodologies such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS)(George & Aston-Jones, 2010). To induce lucidity during REM sleep, brain stimulation methods have been suggested as promising techniques (Holzmann et al., 2014; Stumbrys, Erlacher, & Schredl, 2013). If these techniques turn out to be successful, they might as a tool to improve insight in the psychotic state, or they help to generalize increased insight across altered states of consciousness by indirectly induce lucidity during REM sleep.

One of those brain stimulation methods is tDCS (George & Aston-Jones, 2010) and is perhaps one of the simplest ways of directly stimulating the brain. tDCS involves passing a weak direct current through the brain between two electrodes, from the anode to the cathode. The current causes cortical excitability in the target region. It seems that different brain areas with different cellular composition, layering and morphology, response in their own way to direct current stimulation. Besides that, at least 50% of the current is lost to the surrounding tissue because the human brain is a poor conductor of electricity (George & Aston-Jones, 2010).

Because it is an excitatory method and might possibly increase prefrontal activity, it could work as therapy for psychosis, which has a hypo-function of the PFC (Laruelle, 2014). In studies of tDCS on motor cortex, the current induces excitation or inhibition and can last for several minutes to an hour. Whether these changes can endure for weeks or months remain to be determined.

Another brain stimulation method is TMS, a relatively new technique (George & Aston-Jones, 2010). TMS involves inducing an electrical current within a brain area using pulsating magnetic fields that are generated outside the brain. It has been invented for clinical treatments for depression (Zangen, Roth, Voller, & Hallett, 2005). The typical treatment is a 20-40 min session, 5 days a week. The patient reclines in a chair without moving. The device is held securely against the head while the patient is awake and alert and can discharge on and off for several minutes. This is done with a frequency less than 1Hz for an inhibitory effect or a frequency greater than 1Hz for an excitatory effect. The TMS coil generates a magnetic field impulse that reach the outer layers of the cortex (Davey, Epstein, George, & Bohning, 2003), reactivate descending fibers and electrical impulses cause a depolarization. The initial use of daily TMS to treat depression was based on the theory that depression involved an imbalanced relationship between PFC and limbic brain regions involved in mood regulation (George, Ketter, & Post, 1994). Coupling TMS with neuroimaging, such as fMRI,

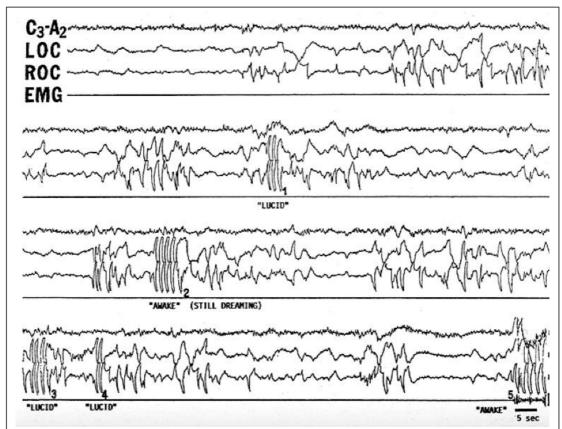
allows one to directly stimulate circuits and image the resultant changes (George et al., 2007; Siebner et al., 2009). Therefore, it could work as a therapy for patients during psychotic episodes. Further research should investigate the effects of TMS during lucid dreaming to see if TMS could induce episodes of lucid dreaming. TMS in psychotic episodes has been investigated, but the effects in lucid dreaming are still unknown. If TMS during REM sleep could induces lucid dreaming, it might improve insight in lucid dreaming from another perspective what could be valuable for the use of lucid dreaming as therapy for psychotic patients.

Different studies found a relation between lucid dreaming and an increased activity in the prefrontal cortex (PFC) (Holzmann et al., 2014; S. A. Mota-Rolim et al., 2010; Voss et al., 2009). They used different methodologies to detect this relationship, such as electroencephalography (EEG) (Voss et al., 2009) and functional magnetic resonance imaging (fMRI) (Dresler et al., 2012).

### **EEG** in lucid dreaming

With EEG it is possible to explore differences between lucid and non-lucid dreams in REM sleep. Holzinger et al. (2006) indicated a higher activation of the left parietal lobe for lucid dreams in comparison to non-lucid dreams. Therefore they concluded that the left parietal lobe is considered to be related to self-awareness and semantic understanding and therefore reflects the self-awareness of the dreaming person in lucid dreams.

In the study of LaBerge et al. (2000), subjects were asked to carry out distinctive patterns of voluntary eye movements when they realized they were dreaming. The eye movement signals appeared on the EOG during REM sleep and are evidence that subjects had become lucid (figure 1)(LaBerge, 2000). Figure 1 shows clearly when lucid dreaming is indicated with eye movement signals. The subject was trained to signal lucidity through a pattern of horizontal eye movements (left-right-left-right, LRLR) and reported through these eye movements five events of lucid dreaming. As can be seen in the figure, the EEG channel shows low voltage mixed frequency and the muscle tone in the electromyography (EMG) channel is very low (Erlacher & Schredl, 2008). This is typical for REM sleep. The eye movement signaling methodology is a powerful approach to dream research.

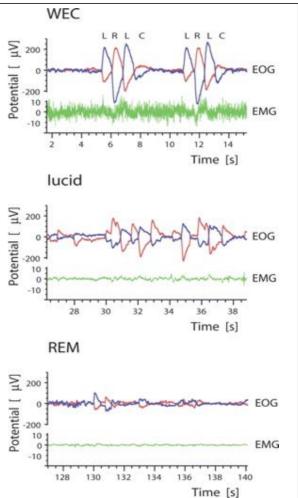


**Figure 1:** A typical signal-verified lucid dream. Channel C3-A2 represent central EEG, LOC and ROC represent left and right eye-movements, EMG represent chin muscle tone. The last 8 min of a 30 min REM period are shown. Five made eye movement signals are labeled (1-5) (LaBerge, 2000).

Voss et al. (2009) sought physiological correlates of lucid dreaming. They hypothesize that the brain must change state if the mind changes state. Three subjects participated in this experiment. They had three months of lucidity training and were able to become lucid in laboratory settings and signal lucidity through eye movements. To analyze EEG records, the study used the mean standardized power values in the following frequency bands:  $\delta$  (1–4 Hz),  $\theta$  (4–8 Hz),  $\alpha$  (8–12 Hz),  $\beta$  1 (12-16 Hz),  $\beta$  2 (16–20 Hz),  $\gamma$  1 (20–28 Hz),  $\gamma$  2 (28–36 Hz) and  $\gamma$  -40 Hz (36–45 Hz).

One figure shows recordings of three states; waking with eyes closed (WEC), lucid dreaming (lucid) and non-lucid REM sleep (REM) (figure 2). In waking and lucid dreaming typical repetitive eye movements are carried out seen in the EOG. The red and blue line refers to 2 EOG channels, one from each eye. Eyes were moved to the left (L), right (R) and back to a central (C) position. Eye movements in lucid dreaming are systematic and more pronounced than in REM sleep. Also the amplitude in REM sleep is much lesser than in lucid dreaming. Both in lucid dreaming and REM sleep, low EMG tracings are found, highlighting the muscle relaxation with no systematic variability (Voss et al., 2009).

Figure 3 shows averages for standardized power across the analyzed frequency band, based on the scalp potentials (POT) for the 3 states (WEC, lucid and REM). Power in lucid is almost the same as REM in lower frequencies and rises above REM at higher frequencies. The increase of power in lucid starts around 28 Hz and peaks at 40 Hz. Compared to WEC, power in frequency bands  $\delta$  and  $\theta$  (1-8 Hz) is increased in lucid and REM. Power in the  $\alpha$  band (8-12Hz) is notable and elevated in WEC. This increase in  $\alpha$  power is typical for WEC. Both the lower  $\alpha$  power and higher  $\delta$  and  $\theta$  activity, so between 4Hz and 8 Hz, are evidence that lucid dreaming occurs in a state of sleep. The evidence that lucid dreaming differs from REM sleep is seen in the increase in higher frequency power during lucid dreaming compared to REM sleep. Therefore lucid dreaming is a unique state of



**Figure 2:** Eye movement signals (EOG) and chin muscle activity (EMG) in WEC, lucid and REM. Eyes are moved to the left (L), right (R), and back to a central position (C) (Voss et al., 2009).

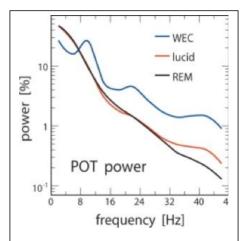
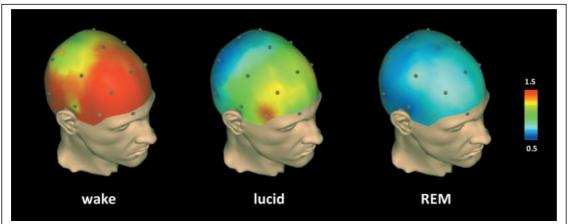


Figure 3: Grand averages for standardized power across the analyzed frequency bands, based on scalp potentials (POT). Power values are shown for all 3 states, WEC (blue), lucid dreaming (red) and REM sleep (black). Frequency resolution is 4 Hz (Voss et al., 2009).

sleep (Voss et al., 2009).

Figure 4 illustrates the topographic representation in a single subject over the right dorsolateral prefrontal cortex (DLPFC) of the overall increase in 40 Hz activities in wake, lucid dreaming and non-lucid REM sleep. Voss et al. showed that lucid dreaming constitutes a hybrid state of consciousness with measurable and definable differences from REM sleep and from waking, especially in frontal areas (Voss et al., 2009).



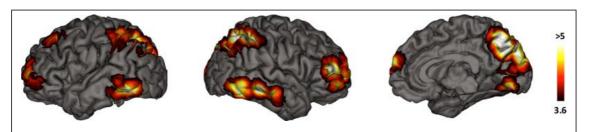
**Figure 4:** 40-Hz standardized power during WEC (left), lucid dreaming (middle), and REM sleep (right). Topographic images are based on movement-free EEG episodes and are corrected for ocular artifacts. For each state, power values are averaged across the respective episode (Voss et al., 2009).

# FMRI in lucid dreaming

FMRI is another methodology to investigate lucid dreaming. There are only a few studies applying fMRI. The head of the participant has to be kept in the exact same position while sleeping and dreaming, which is uncomfortable. During the recording session, the scans are causing a strong noise that disturbed the participant while falling asleep or wake up participants who had fallen asleep. Another reason why only a few studies applying fMRI is that it was not possible to use fMRI and EEG recordings at the same time because of the strong magnetic field produced by the fMRI (Erlacher & Schredl, 2008). Yet, fMRI studies showed that brain activation patterns are different in different sleep stages and differ from brain activation in wakefulness (Dang-vu et al., 2005). Even though it will be a challenging task for a participant to have lucid dreams during fMRI recordings, this approach gives valuable insights into the neural structures on the cortex level (Lacourse, Orr, Cramer, & Cohen, 2005).

The objective of the study of Dresler et al. (2012) was to investigate the neural correlates of lucid dreaming, based on combined EEG/fMRI recordings of night sleep and dreaming. In this study, four participants, who were experienced lucid dreamers, slept several nights in a magnetic resonance imaging (MRI) scanner under concurrent polysomnography, the standard for differentiating sleep from wake and identification of different sleep stages (Tonetti, Pasquini, Fabbri, & Belluzzi, 2009). One subject had two episodes of lucid REM sleep that was long enough to be analyzed by fMRI.

Figure 5 shows that during lucid REM sleep, dorsolateral frontopolar and prefrontal regions including the superior, middle and inferior frontal gyri, parietal regions including the precuneus, inferior parietal lobule and supramarginal gyrus, and temporal regions including the inferior and middle temporal gyri activated strongly as compared with non-lucid REM sleep. Lucid dreaming was associated with a reactivation of brain areas that are normally deactivated during REM sleep. This is in line with recent EEG data (Voss et al., 2009). The pattern of activity can explain the recovery of reflective cognitive capabilities that are the hallmark of lucid dreaming (Dresler et al., 2012).



**Figure 5:** Activity related to lucid dreaming. Color coded clusters represent areas significantly activated during lucid epochs in REM sleep: left hemisphere (left), right hemisphere (middle), and midline view (right) (Dresler et al., 2012).

### **Psychosis and dreaming**

Psychosis describes a range of conditions characterized by unusual experiences such as hearing voices, delusional beliefs and disturbances to thought and language that cause disruption to normal functioning (Peters, 2007). Psychosis is a symptom, not an illness. A psychotic or mental illness, like schizophrenia, can cause psychosis.

REM sleep is the sleep stage in which the brain is very active and cause intensive dreams (Niedermeyer & da Silva, 2005). This internally generates emotions, and perceptions show many delusional thoughts, bizarre plots and a complete lack of insight into the true state of the subject. In this regard, dreaming resembles the psychosis of mental illness. Both fail to discern self-generated perceptions from non-self-generated perceptions and they accept bizarre experiences as real (Palagini & Rosenlicht, 2011). Therefore, dreaming can indeed serve as a model for psychosis.

Between 50-80% of the patients diagnosed with schizophrenia or another mental illness, have too little insight into their disorder, meaning that they may not acknowledge their illness or the need for treatment (Lincoln, Lüllmann, & Rief, 2007). This is probably due to ineffective self-reflection processes and leads to more relapses, poorer therapy success and more rehospitalisations (Mintz, Dobson, & Romney, 2017). Therefore, the concept of insight in the psychotic state is becoming a more important area for research of psychosis (Baier, 2010).

Psychotic patients continuously experience dream-like mentation during both dreaming and waking (Llewellyn, 2013). Psychosis and dreaming share important features, such as general lack of criticism, and intrinsic sense perceptions independent of external stimulation that are associated with reduced frontal cerebral activity (Cicogna & Bosinelli, 2001). For psychotic patients a dream experience is peculiar. Subjective dream report analysis reveal a higher frequency of dreams among schizophrenic patients than in healthy people (Michels et al., 2014; Okorome Mume, 2009). They experience more hostile contents, a lower frequency of dreams in which the dreamer is the main character, and a higher proportion of strangers among the dream characters (Skancke, Holsen, & Schredl, 2014).

Psychosis-related cognitive deficits are accompanied by impairment in the ability to share thoughts when remembering a dream, leading to less connected reports than those produced by healthy subjects. These differences were more prominent for dream reports than for waking reports (B. Mota, Furtado, Maia, Copelli, & Ribeiro, 2014). The hypo-function of the PFC in psychosis resembles the reduction of PFC activity during REM sleep in healthy subjects, in comparison to the levels found in waking. Both in regular dreaming and psychosis, the hypo-function of the PFC seems to be causally related to the decreased criticism typical of both states (Dresler et al., 2015). Lucid dreaming shows increased activity in the PFC, compared with non-lucid dreaming (S. A. Mota-Rolim et al., 2010; Stumbrys et al., 2013; Voss et al., 2009).

### Lucid dreaming as a treatment for psychosis

Because lucid dreaming shows increased activity in the PFC, it has been proposed as potential therapy for psychotic patients (Dresler et al., 2015; Holzmann et al., 2014). According to this view, Mota et al. examined whether psychotic patients report lucid dreaming less frequently and with lower control ability than healthy subjects. They also expected that psychotic patients who are able to experience lucid dreaming, present milder psychiatric symptoms than psychiatric patients unable to experience lucid dreaming. Lucid dreaming features, such as occurrence, frequency, and control abilities, and psychiatric symptoms were investigated in 45 participants with psychotic symptoms and 28 healthy participants (N. B. Mota, Resende, Mota-Rolim, Copelli, & Ribeiro, 2016). Mota et al. falsify their hypotheses because psychotic patients did not report lucid dreaming less frequently than healthy subjects who are not psychotic. Also among the participants that reported lucid dreams during the experiment, psychotic patients reported lucid dreaming control more frequently than non-psychotic participants. Finally, patients who reported lucid dreaming had no reduced psychiatric symptoms, in comparison with patients who did not report lucid dreaming. Despite these results are not lifetime data, there is no support found for the notion that a psychotic patient would report less lucid dreaming than a non-psychotic patient. In this study, only 23% of the non-psychotic participants reported lucid dreaming control, in contrast with significantly large numbers among psychotic participants (67%-73%) (N. B. Mota et al., 2016). This was an unexpected result, because different studies consider that non-psychotic participants able to experience lucid dreaming show increased control of internal reality (Blagrove & Hartnell, 2000; Blagrove & Tucker, 1994), and are more frequently able to regulate emotion and cognition than non-lucid dreamers (Blagrove & Hartnell, 2000). A possible explanation is that in perspective to the external reality, a psychosis enhances the experience of the internal reality. Therefore, lucid dreamers with psychotic symptoms would be better able to control their internal reality than non-psychotic lucid dreamers.

# Brain areas involved in psychosis and lucid dreaming

Dreaming and psychosis share important neurophysiological features (Gottesmann, 2005). Both present essential sense perceptions independent of external stimulation are associated with a lack of criticism concerning the bizarreness of these experiences (Cicogna & Bosinelli, 2001). This idea stems from the decrease in PFC activity that characterizes both REM sleep and psychosis (Dresler et al., 2015; Holzmann et al., 2014). The parallel between insight in dreaming and psychosis forms a good basis for further research. If this parallel is correct, neural correlates of insight into the psychotic state should largely overlap with neural correlates of dream insight (Dresler et al., 2015). Shedding light on the neural mechanisms underlying impaired insight in psychotic patients is a hot topic for an increasing number of approaches of treatment.

Depending on the type of study, several brain areas are involved in psychosis. Neuroimaging studies confirmed an association of the PFC with impaired insight in psychotic patients, but also parietal areas are relevant in this regard (Kumar et al., 2013). A difference in an fMRI study could be the use of a self-evaluation task (Bedford, Surguladze, Giampietro, Brammer, & David, 2012) or self-reflection task (Meer et al., 2013). Also in task-free scans, functional differences in brain activity associated with impaired insight in psychosis have been reported (Liemburg et al., 2012). Beside these examples, there are many more types of studies (Dresler et al., 2015). Most neuroimaging studies of insight deficits in psychosis focused on structural measures. To sum up all studies with related brain areas, a rather scattered picture will arise, with a great diversity of brain areas involved both in insight deficits in psychosis and lucid dreaming. It is possible to create a more specific picture if only brain regions are considered whose association with lucid dream insight and psychotic insight has been replicated at least once with the same modality in different studies. Then, the dorsolateral and frontopolar prefrontal cortices in general and specifically the bilateral superior, bilateral middle and right inferior frontal gyri, and the bilateral medial prefrontal cortex, the bilateral anterior and

posterior cingulate cortices, the bilateral superior, the bilateral precuneus, left middle and bilateral inferior temporal cortices, and the right lingual gyrus are all brain areas involved in both lucid dreaming and psychotic insight deficits (Dresler et al., 2015). The most common brain areas seem to be the prefrontal, medial parietal and cingulate cortex.

### Correlations and limitations between lucid dreaming and psychosis

Lucid dreaming remains a rare and special phenomenon. Some research has been done, but it is still not enough to really say something about possible use in psychiatric therapies. It is known that the PFC increases in activity during lucid dreaming (Holzmann et al., 2014; S. A. Mota-Rolim et al., 2010; Voss et al., 2009), while the PFC decreases in activity during a psychotic episode (Dresler et al., 2015). The hypo-function of the PFC in psychotic patients can be improved through cognitive training (Edwards, Barch, & Braver, 2009). Metacognitive training is a good example of cognitive training and is usually a group-training program. Common misconceptions and a unilateral way to solve problems are addressed. Patients are invited to look critically at their way of dealing with problems, change these and adapt the contents of the training in daily life (Moritz & Woodward, 2006). Metacognitive training has been proven itself as successful in nightmare therapy (Spoormaker & Van Den Bout, 2006). Skilled lucid dreamers also use metacognitive training to gain their frequent insight into the dreaming state and contemplate their state of consciousness (Stumbrys, Erlacher, Schädlich, & Schredl, 2012). Teaching schizophrenia patients via metacognitive training can enhance insight in prefrontal and medial parietal functions, which might lead to enhanced insight capabilities and increase the probability to acquire lucidity during dreams, which might be valuable as therapy for psychotic patients (Stumbrys et al., 2012).

Dresler et al. (2015) wrote a valuable review about insight in dreaming and the psychotic state, and the overlap in neural correlates. Lucid dreaming is a good indicator to increase impaired insight in the psychotic state because they share unusual mental events such as delusions and hallucinations.

A big difficulty in dream research is the amount of participants. The number of studies in the field of lucid dreaming is small because it is difficult to recruit many skilled lucid dreamers (Erlacher & Schredl, 2008). To gain a more specific approach to dream insight, more research on the brain basis is needed. Therefore, larger sample sizes are needed. If different aspects of lucid dreaming could be differentially associated with their neural correlates (Voss, Schermelleh-Engel, Windt, Frenzel, & Hobson, 2013), than they could be compared with neural correlates of specific insight scales in psychotic patients.

This review emphasized the overlap between insight in lucid dreaming and psychosis and the neural correlates, but this provided evidence for the 'dreaming-psychosis model' couldn't be generalized to all aspects. The neural basis might be similar during dreaming and psychosis, but other aspects of the model will have to be tested in further studies and critical questions need to be asked.

Even though good evidence is lacking, lucid dreaming induction methods and training to become a lucid dreamer might lead to new therapeutic approaches to improve insight in psychosis and might even lead to a new therapy for patients with psychotic episodes. Experiencing lucid dreaming can enhance the feeling of control in daily life, because lucid dreamers have the possibility to control dream content during lucid dreaming (van Eeden, 1913). This issue deserves further attention to investigate whether the feeling of control during lucid dreaming can be converted to the feeling of better control during a psychotic episode. More control during a psychotic episode helps the patient to suppress symptoms such as hallucinations and delusional thoughts, and to get more insight in their true state (Amador, Strauss, Yale, & Gorman, 1991). Further experimentation is required to test this promising approach for treatment of psychosis.

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