Neurobiology and clinical implications of lucid dreaming

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A R T I C L E   I N F O

Article info

Several lines of evidence converge to the idea that rapid eye movement sleep (REMS) is a good model to foster our understanding of psychosis. Both REMS and psychosis course with internally generated perceptions and lack of rational judgment, which is attributed to a hyperlimbic activity along with hypofrontality. Interestingly, some individuals can become aware of dreaming during REMS, a particular experience known as lucid dreaming (LD), whose neurobiological basis is still controversial. Since the frontal lobe plays a role in self-consciousness, working memory and attention, here we hypothesize that LD is associated with increased frontal activity during REMS. A possible way to test this hypothesis is to check whether transcranial magnetic or electric stimulation of the frontal region during REMS triggers LD. We further suggest that psychosis and LD are opposite phenomena: LD as a physiological awakening while dreaming due to frontal activity, and psychosis as a pathological intrusion of dream features during wake state due to hypofrontality. We further suggest that LD research may have three main clinical implications. First, LD could be important to the study of consciousness, including its pathologies and other altered states. Second, LD could be used as a therapy for recurrent nightmares, a common symptom of depression and post-traumatic stress disorder. Finally, LD may allow for motor imagery during dreaming with possible improvement of physical rehabilitation. In all, we believe that LD research may clarify multiple aspects of brain functioning in its physiological, altered and pathological states.

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Introduction

Dreams and psychosis

A relation between dreams and madness was pointed out by Kant and Schopenhauer: they suggested that “a lunatic is a wakeful dreamer” and that “a dream is a short-lasting psychosis, and a psychosis is a long-lasting dream”, respectively. In accordance with both, Wundt affirmed that “we can experience in dreams all the phenomena we find in the hospice”. They all influenced Sigmund Freud, who postulated that psychosis is an abnormal intrusion of a dreaming activity into an awake state [1]. Emil Kraepelin, although one of the greatest opponents of psychoanalysis, also hypothesized the same [2]. Jung said “if we could imagine a dreamer walking around and acting his own dream as if he were awake, we would see the clinical picture of dementia praecox” [3]; the latter term, coined by Kraepelin, was later renamed schizophrenia by Bleuler. Bleuler also wrote “the modalities of thinking of schizophrenic subjects are very similar to dreaming” and that dreaming “has its own rules, and that most of the characteristics of schizophrenic thinking (particularly delusional thinking) are explained by the differences between the dreaming and the wakefulness way of thinking” [4].

There are many evidences that rapid eye movement sleep (REMS) – the mental stage mostly related to dreaming [5,6] – is a physiological experience similar to the psychotic symptoms of schizophrenia. The discovery of antipsychotic agents led to the hypothesis that the positive symptoms of schizophrenia such as psychosis correlate with increased dopaminergic activity in certain neuronal pathways [7]. Currently, pharmacological and/or genetic manipulations that increase dopaminergic transmission induce psychotic-like behaviors in animals, and are thus useful to understand the mechanisms underlying psychosis. For example, Dzirasa and colleagues [8] could not distinguish the spectral content of REMS from wake state in psychotic-like rats: both wakefulness and REMS exhibited equal levels of fast and slow oscillations' power. Therefore, REMS and wake states seem to be intermingled in animal models of psychosis. Dopamine D2 receptors in the mesolimbic pathway are activated during REMS [9], the same pathway involved in psychosis [10,11]. This could explain why dreams are
characterized by a plethora of mental experiences that resembles hallucinations [12,13], suggesting that dreaming would be a good model for psychosis [14–16]. In accordance with this hypothesis, Dzirasa and colleagues [8] showed that reducing dopamine transmission, a therapy used to treat psychosis, also suppresses REMS. REMS and schizophrenia have similar variations in the levels of noradrenaline, serotonin, acetylcholine and glutamate: both are associated with decreased levels of noradrenaline and serotonin, and increased activity of cholinergic and glutamatergic systems [14,15,17,18]. Hallucinogenic substances also suggest a link between dreams and psychosis [19]: LSD increases the frequency of ponto-geniculo-occipital waves, which are associated with the execution of brief ocular saccades during REMS, constituting therefore a possible correlate for the visual experiences induced by LSD and during dreaming [20].

Neuroimaging studies of the frontal cortex further indicate a proximity between dreams and psychosis. Schizophrenic subjects have this region severely impaired – a condition called hypofrontality [21–23]. Frontal areas also deactivate during physiological REMS, especially the dorsolateral prefrontal cortex [24–26]. Low frontal activity may reduce self-awareness and induce delusional thoughts and lack of rational judgment, which are present in both REMS and psychosis [12,13].

Lucid dreaming

As reviewed above, the bizarre experiences of dreams, along with neurochemical, electrophysiological and neuroanatomical observations suggest that psychosis would be a pathological dreaming during the wake state, as hypothesized by Kant, Schopenhauer, Wundt, Freud, Kraepelin, Jung and Bleuler, among others. In Greek mythology, Nyx, the goddess of night, gave birth to Hypnos, who represented sleep. Hypnos had a sister called Lyssa – the madness goddess – and fathered Morpheus, the god of dreams [27]. Thus, links between dreams and psychosis appear since the old ages. However, during lucid dreaming (LD), subjects become aware of dreaming during the dream, an exception to the idea that dreaming is necessarily an experience concurring with lack of rational judgment.

The term LD was coined by Van Eeden in 1913 [28] to denote a dream in which “... 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”. Decades later, Laberge and colleagues, in a scientifically controlled setup, instructed subjects to perform ocular movements (e.g. two consecutive left–right turns) if they became lucid while dreaming [29,30]. This is possible because eye muscles are not in atony during REMS [5,6]. Laberge’s technique allowed for subsequent electroencephalographic (EEG) recordings of subjects experiencing LD [31–35].

The last two decades witnessed an unparalleled growth in our understanding of the neurobiological basis of LD. The gain of knowledge also led to many open questions. One intriguing issue is that even though LD occurs during REMS [34], most people have REMS every night without LD [36–38]. In this sense, we have recently proposed that there exists more than one kind of REMS, and that the specific kind of REMS during which LD occurs has EEG spectral features that differentiate it from non-lucid REMS [39]. In accordance, early studies provided evidence of a relationship between the level of lucidity and the overall amount of alpha band (8–12 Hz) power [32,33]. However, a subsequent study detected increased power within the beta band (13–20 Hz), restricted to the parietal region during LD [40]. Therefore, while there is evidence to suggest that LD have different EEG spectral characteristics from non-lucid REMS, it is fair to say that there is substantial disagreement with regard to the brain regions and frequency bands most activated during LD.

Hypotheses

As mentioned above, neuroimaging studies observed a decrease in frontal activity during psychosis [21–23] and REMS [24–26], which could explain the lack of rational judgment in these mental states. Considered as the executive center of the brain, the frontal lobe enhanced significantly along evolution; in humans, this lobe plays a role in self-consciousness, working memory and attention [41–43]. Here we hypothesize that LD is related to a frontal activity during REMS, which would allow the occurrence of executive functions during dreaming and facilitate lucidity.

We also suggest that LD is inversely related to psychotic symptoms. Just as some authors consider psychosis a pathological dreaming during wake, LD would be a physiological awakening while dreaming. Consistent with this, psychotic subjects have reduced activity in frontal regions, the opposite of what we hypothesized to happen during LD (Table 1).

Evaluation of the hypotheses

EEG studies on LD started with Laberge’s technique that consists of a pre-arranged series of ocular movements to indicate dream lucidity [30,32–35,40]. However, the neurobiological basis of LD remains controversial. Since the frontal lobe is associated with executive functions such as self-consciousness [41], our hypothesis is that LD is related to a frontal activity during REMs.

According to this hypothesis, we have preliminary observed that LD occurs with increased gamma activity (30–50 Hz) in frontal regions when compared to non-lucid REMS [39,44]. This is consistent with findings of Voss and colleagues [45], who detected increased gamma oscillations (~40 Hz) in frontal cortex during LD (Table 2). Using cognitive tasks and a dream diary, Neider et al. [46] observed that subjects who performed better on a task that engages the ventromedial prefrontal cortex exhibited more lucidity reports, which was not the case for a task related to the dorsolateral prefrontal cortex. However, we recently suggested that different subjective experiences during LD have different neural substrates [39]. In accordance, a motor task with hand movements performed during LD increases neuronal activity in the sensorimotor cortex [47].

Against the hypothesis that LD is associated with increased frontal activity during REMs, Ogilvie et al. [32], and Tyson et al. [33] found no specific brain region to be activated during LD; instead, the authors reported global increases in alpha power compared to non-lucid REMS. In accordance, we preliminary observed that LD correlates with increased alpha power [48], which suggests that LD could be an intermediate stage (or phase

<table>
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<th>Concept</th>
<th>Psychosis</th>
<th>Lucid Dreaming</th>
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<td>Phenomenology</td>
<td>Dreaming during wake</td>
<td>Waking during dreams</td>
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<tr>
<td>Neurobiology</td>
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<td></td>
<td>Generalized hypofrontality</td>
<td>Localized hyperfrontality</td>
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Table 1 Differences between psychosis and lucid dreaming.
because they connect the dreaming brain and the external world. Micro-arousals caused by auditory or tactile stimuli associated
by tactile (36%), visual (20%) or olfactory (17%) sensations[68].
that incubates into dreams is auditory (a voice = 47%, phone
have recently conducted indicate that the main stimulus modality
of auditory and sensory regions, instead of by the activation of
the subject to become aware of dreaming. Therefore, the possible
effects of TMS or TDCS on dreaming could be due to stimulation
occurring in the frontal regions during REMS[39,44,45], we predict that actuating
frontal regions by TMS during REMS could trigger LD.
Recent studies showed that transcranial direct current stimulation (TDCS) of the frontal cortex during sleep improves mood of
depressive patients[58], increases memory performance[59] and
the ability to solve complex tasks[60]. TMS over the frontal cortex
increases gamma activity and working memory[57]. Marshall and
colleagues first applied TDCS frontally during sleep and were
able to show enhanced memory consolidation[61,62]. Massimini
and colleagues applied TMS during REMS over motor areas and observed changes in cortical transmission within stimulated areas as well as between connected ones[63–65]. Nevertheless, to the best of our knowledge, there are no studies that employed TMS or TDCS over frontal regions during REMS.
One problem of these techniques in sleep studies is that the
noise and/or tactile sensations, although not strong enough to
wake up the subjects, could incubate into dreams or cause micro-arousals. Incubating auditory[66] or visual[67] stimulus into
REMS is a technique to induce LD, which would act as a cue for
the subject to become aware of dreaming. Therefore, the possible
effects of TMS or TDCS on dreaming could be due to stimulation
of auditory and sensory regions, instead of by the activation of
the targeted region. Preliminary results of an online survey we
have recently conducted indicate that the main stimulus modality
that incubates into dreams is auditory (a voice = 47%, phone
rings = 43%, alarm clock = 41%, house/street noise = 37%), followed by
tactile (36%), visual (20%) or olfactory (17%) sensations[68].
Micro-arousals caused by auditory or tactile stimuli associated
with TMS and TDCS could thus be an important confounding factor
because they connect the dreaming brain and the external world
[69], being capable of inducing LD by themselves.
Finally, it should be noted that EEG has important spatial limitations. A possible way to increase its spatial resolution is by associating EEG recordings with functional neuroimaging techniques. Using such approach, Dresler and colleagues recently observed increased activity in the sensorimotor cortex when subjects dreamed about executing hand movements during LD[47]. Despite these progresses, however, the main challenge in LD research still is the development of efficient strategies to induce LD[70].

Consequences of the hypotheses
We believe LD research has three main clinical implications, as we expose below:

LD may help to understand consciousness and its disturbances
Since dreams are a model to foster our understanding of psychosis, LD could be ultimately important to the study of consciousness and its disturbances[71,72]. Here we consider consciousness as a dynamic process that can be temporally divided into states. We define the states of consciousness (SC) by a subjective experience and correlated neurobiological substrate, which can be physiological, altered or pathological (Table 3).

“The false awakening” is a physiological SC in which subjects dream of getting up, using the toilet, making the bed, eating breakfast and so on, as in a normal awakening experience[73]; however, some details are usually missing or appearing in a strange way, which cues the subject to become lucid during dreaming[74]. “Daydreaming” is an experience similar to dreaming but which occurs during a physiological wake state. During daydreaming, subjects are introspective and usually fantasize about something that takes out their attention from the external world[75]. Interestingly, there is a relationship between daydreaming and psychosis[76]. It is also important to note that physiological hallucinations may occur during transitions from wake to sleep (named hypnagogic) and from sleep to wake (hypnopompic).

“Sleep paralysis” is a pathological SC in which subjects suffer frequent or intense episodes of incapacity to move after awakening; it is a dissociated state in which EEG patterns are similar to waking, and there is muscle atonia as in REMS[77]. Some have hallucinations, which – depending on cultural beliefs – is misinterpreted as incorporation of demons or alien abduction[78–81]. “Sleepwalking” disorder is characterized by repeated episodes of rising from bed during sleep and walking around, unresponsiveness, difficulty to be awakened, and amnesia of the episode. It is a dissociated state in which EEG is similar to deep sleep (slow frequency and high amplitude waves) and motor activity is similar to waking

Table 2
<table>
<thead>
<tr>
<th>Global brain</th>
<th>Parietal region</th>
<th>Frontal area</th>
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<tbody>
<tr>
<td>Alpha</td>
<td>Holzinger et al. (2006) [40]</td>
<td>Mota-Rolim et al. (2008) [44]</td>
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Table 3
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<tr>
<th>Physiological</th>
<th>Altered</th>
<th>Pathological</th>
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<tr>
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<td>Behavioral: Yoga, Hypnosis</td>
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<tr>
<td>Dreams</td>
<td>Physical (TMS/TDCS); out-of-body experience</td>
<td>Sleep paralysis and sleepwalking*</td>
</tr>
<tr>
<td>Lucid dreaming</td>
<td>Chemical: LSD, ecstasy</td>
<td>REMS behavior disorder</td>
</tr>
<tr>
<td>False awakening</td>
<td>Behavioral/chemical: Ayahuasca worship</td>
<td>Near-death experience</td>
</tr>
<tr>
<td>Daydreaming</td>
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<td>Coma</td>
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* Sleep paralysis and sleepwalking (somnambulism) are considered pathological if very frequent and/or intense.
In “REMS behavior disorder” subjects do not present typical REMS muscle atonia and perform motor activity associated with dream imagery [83]. It was recently shown that this disturbance is associated with Parkinson disease and other motor disorders [84]. “Near-death experience” courses with visions of life memories and altered sense of time due to a hypofrontality state that disinhibits the limbic system [85–87], resembling REMS and psychosis. “Coma” is another Pathological SC in which arousal levels are minimum [88,89]. Curiously, some patients returning from coma report memories of experiencing feelings and thoughts during the comatose state; this suggests that they may have kept a vestige of consciousness (as a long dream) during coma.

“Yoga meditation” is an Altered SC characterized by an eyes-closed, mentally-relaxed wake state, and enhancement of the alpha rhythm [90]. Long-term practitioners have more rapid-eye movements during REMS [91], which may be associated with the observation that meditation is correlated with LD frequency [36,68]. “Hypnosis” uses verbal suggestion to achieve a relaxed mental state similar to sleep, but with focused attention [92], and has been used for the emotional modulation of pain [93]. The “out-of-body experience” is the subjective sensation of being outside one’s own body [49–52]. Curiously, since some subjects view their own body during LD, they tend to interpret LD as an out-of-body experience [94–96]. Other Altered SCs can be achieved by the use of synthetic substances such as LSD or ecstasy (MDMA) for recreational purposes, or by natural plant compounds used in ritualistic ceremonies such as peyote (mescaline) [97] and ayahuasca (dime-tritryptamine) [98]. Interestingly, reports from these altered SC tend to resemble a dream narrative.

Given the similarities between several SCs and dreaming, LD could be a useful window of opportunity to better understand the subjective experiences and neurobiological characteristics of these states. For instance, it would be interesting to know whether the patterns of EEG activity during LD compare with those recorded during the different SCs.

**LD may be a therapy for recurrent nightmares**

Nightmares are long and terrifying dreams, usually involving threats to survival, security or self-esteem [99]. Most of the people have occasional nightmares; however, nightmares can become recurrent and cause significant distress in some cases, particularly in post-traumatic stress disorder (PTSD) or severe depression [100].

PTSD is characterized by feelings of intense fear, helplessness or horror that occur when a person experiences, witnesses or is confronted with events that involve death, serious injury or threat to integrity [99]. More than two thirds of the world population experiences a traumatic event during lifetime, and recurrent nightmares are a major symptom [101,102]. Depression, the most common mood disorder, is characterized by loss of energy or interest, difficulty of concentrating, feelings of regret or failure, and changes in appetite, among other symptoms [99]. Patients with depression also have important sleep disturbances and recurrent nightmares, which are associated with suicidality [103–105].

Psychotherapies based on inducing LD could be an effective way of treating patients with recurrent nightmares [106–111]. We believe the ability of becoming lucid during nightmare could bring about three outcomes, which are all beneficial. First, subjects may simply wake up during a nightmare, which is always a possibility during LD. Second, once aware of dreaming, and therefore of the lack of reality of the perceptual experience, subjects may naturally lose their fear by realizing the absence of real threats. Third, subjects experiencing LD could change dream context, in a way of transforming the nightmare into a neutral or even a pleasant dream. Nevertheless, despite these perspectives, the development of therapeutic strategies to treat recurrent nightmares based on LD also depends on efficient techniques for LD induction [70].

**LD may assist the rehabilitation of motor disorders**

Mental simulations of motor skills increase actual performance in behavioral tasks: repeated imagination of muscle contraction increases muscular strength per se [112], and mental simulations improve learning of motor skills [113] and sports performance [114]. Interestingly, motor skills can be acquired without awareness of the learning process [115]. These observations suggest that being capable of performing imaginary movements during LD could influence actual motor skills during the wake state. In this way, patients with physical disabilities could potentially practice motor tasks during LD and evaluate whether dream rehearsal decreases motor symptoms. Motor training during LD could also be used by normal subjects to improve their physical skills [116]; in fact, performing a motor task during LD increases activity in sensorimotor cortex [47].

**Conclusions**

Dreams simulate past experiences as well as future expectations [68,117], and are associated with wishes [1], but also with fears [68,118]. These are the elementary tenets of evolution: based on past experiences we desire the pleasant, but are also afraid of taking risks. As a simulation of reality, we believe that dreams may have acquired an adaptive function [68,117].

LD is a special oneric experience in which one has conscious access to dream content, being able to control or even stop it. The neural underpinnings of LD are yet to be better understood, but the findings reviewed here, along with the derived hypothesis, suggest that LD research is a fertile field for increasing our knowledge of multiple aspects of brain functioning.

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**References**


