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Neurobiology and clinical implications of lucid dreaming ☆

Sérgio A. Mota-Rolim *, John F. Araujo

Instituto do Cérebro - Universidade Federal do Rio Grande do Norte, Av. Nascimento de Castro 2155, Bairro Nova Descoberta, CEP 59056-450, Natal, RN, Brazil
Departamento de Fisiologia, Centro de Biociências - Universidade Federal do Rio Grande do Norte, Bairro Lagoa Nova, Caixa Postal 1506, CEP 59078-970, Natal, RN, Brazil
Laboratório do Sono, Hospital Universitário Onofre Lopes - Universidade Federal do Rio Grande do Norte, Av. Nilo Peçanha 620, Bairro Petrópolis, CEP 59.012-300, Natal, RN, Brazil

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ABSTRACT

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.

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

* Corresponding author at: Universidade Federal do Rio Grande do Norte, Instituto do Cérebro, Av. Nascimento de Castro 2155, Bairro Nova Descoberta, CEP 59056-450, Natal, RN, Brazil. Tel.: +55 (84) 3215 3409x218; fax: +55 (84) 3211 9206.
E-mail address: sergioarthuro@neuro.ufrn.br (S.A. Mota-Rolim).

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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 delirious 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 1
Differences between psychosis and lucid dreaming.

	Psychosis	Lucid Dreaming
Concept	Dreaming during wake	Waking during dreams
Phenomenology	Pathological	Physiological
Neurobiology	Generalized hypofrontality	Localized hyperfrontality

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