Behavioural Brain Research 226 (2012) 592–596

Contents lists available at SciVerse ScienceDirect

Behavioural Brain Research

journal homepage: www.elsevier.com/locate/bbr

Review

How we remember the stuff that dreams are made of: Neurobiological approaches to the brain mechanisms of dream recall

Luigi De Gennaro[a,*], Cristina Marzano[a], Carlo Cipolli[b], Michele Ferrara[c]

[a] Department of Psychology, University of Rome "Sapienza", Italy
[b] Department of Psychology, University of Bologna, Italy
[c] Department of Health Sciences, University of L'Aquila, Italy

ARTICLE INFO

Article history:
Received 29 September 2011
Accepted 11 October 2011
Available online 17 October 2011

Keywords:
Dream recall
Brain damage
Intracranial recordings
EEG oscillations
Diffusion tensor imaging
Subcortical nuclei
Local sleep

ABSTRACT

Intrinsic and historical weaknesses delayed the spread of a sound neurobiological investigation on dreaming. Nevertheless, recent independent findings confirm the hypothesis that the neurophysiological mechanisms of encoding and recall of episodic memories are largely comparable across wakefulness and sleep. Brain lesion and neuroimaging studies converge in indicating that temporo-parieto-occipital junction and ventromesial prefrontal cortex play a crucial role in dream recall. Morphoanatomical measurements disclose some direct relations between volumetric and ultrastructural measures of the hippocampus–amygdala on the one hand, and some specific qualitative features of dreaming on the other. Intracranial recordings of epileptic patients also provide support for the notion that hippocampal nuclei mediate memory formation during sleep as well as in wakefulness. Finally, surface EEG studies showed that sleep cortical oscillations associated to a successful dream recall are the same involved in encoding and recall of episodic memories during wakefulness.

Although preliminary, these converging pieces of evidence strengthen the general view that the neurophysiological mechanisms underlying episodic/declarative memory formation may be the same across different states of consciousness.

Contents

1. Introduction to the neurobiological investigation on dreams

The production of dreams during sleep is a largely unexplained phenomenon of human existence, and its underlying brain mechanisms are mostly unknown. This is mainly due to the unaccessibility of dreams to a *direct* study, but only by using dream recall after spontaneous or provoked awakening from sleep. Like the Schrodinger's Cat, we can investigate dreaming only interrupting dream experience and, in this way, altering the physiological *scenario* in which dreams are produced (i.e., the electrical and neurochemical characteristics of sleep stages). Until innovative protocols – which might overcome this apparently insurmountable obstacle – will be introduced, neurobiological investigation is necessarily limited to the brain mechanisms of dream *recall*.

Almost inevitably related to the issue of this indirect access to dreaming, the study of brain mechanisms of dreams has been mainly restricted to rapid eye movement sleep (REMS), implicitly assuming that dreams are strictly dependent on its specific physiology. This assumption was essentially based on the initial finding

* Corresponding author at: Dept. of Psychology – Section of Neuroscience, University of Rome "Sapienza", Via dei Marsi, 78, 00185 Rome, Italy. Tel.: +39 06 49917647; fax: +39 06 49917711.
E-mail address: luigi.degennaro@uniroma1.it (L. De Gennaro).

0166-4328/$ – see front matter © 2011 Elsevier B.V. All rights reserved.
doi:10.1016/j.bbr.2011.10.017

that subjects awakened from REMS reported that they were dreaming in 70–95% of the cases, whereas only in 5–10% of the cases after non-rapid eye movement sleep (NREMS) awakenings [1]. In fact, the systematic review of the studies comparing dream recall from REMS and NREMS awakenings shows that the phenomenon cannot be restricted to a specific sleep state, although large REMS–NREMS differences actually do exist in dream report length [2,3] and qualitative features [4]. The assumption that dreams are dependent on (or strictly associated to) the specific physiology of REMS seems now untenable.

The consequences of the *reduction* of dreaming to a single sleep stage do not seem trivial, since it might imply that the physiology of REMS partly or completely *coincides* with that of dreaming. In such way, studying the brain mechanisms of REM sleep might have surrogated the analysis of brain mechanisms of dreaming as well as of dream recall. The weakness and the paucity of consistent evidence on this topic should be attributed overall to the indirect access to dream experience, and the improper reduction of dream generation to REM sleep.

On the other hand, the refinement of the experimental techniques over the last years allows more direct and systematic investigations on the neurobiological features and the neural basis of dreaming. In the present review, we will discuss the findings of some recent investigations which outline a coherent, albeit still incomplete, picture of how brain structures interact in dream formation. This will be done by integrating the main findings provided by neurobiological approaches focused on (A) brain lesions, (B) functional neuroimaging of cortical and subcortical structures, (C) morphoanatomical measurements of subcortical structures, and (D) "local" changes in electroencephalographic (EEG) cortical oscillations.

2. Dreaming in brain damaged patients

The so-called "neuropsychology of dreaming" has evaluated dream features in patients with selective brain lesions and provided univocal evidences that confirmed the assumption that phenomenology of dreams is closely related to the brain activity and organization. Preliminary neuropsychological observations were addressed to the brainstem, in keeping with the view that the high co-occurrence of REM sleep and dream recall implies that brainstem mechanisms are also responsible of dream generation. Nevertheless, it was reported that brainstem lesions do not necessarily abolish dreaming [5]. Accordingly, dreaming occurs in the absence of REMS in depressed patients treated with monoamine oxidase (MAO) inhibitors, which suppress REMS without affecting the intensity of NREMS or the exponential decay of SWA [6].

Indeed, historical reports of the 19th century had suggested that the complete (or nearly complete) loss of dreaming was associated with localized lesions in the forebrain and with total absence of lesions in the brainstem. These clinical reports described two patients who dreamed "almost not at all anymore" after respectively a bilateral occipital–temporal [7] and bilateral occipital [8] lesion. There was, in fact, a double dissociation, suggesting that REMS can occur without dreaming as well as dreaming can occur without REMS. Even at this stage of evidence REM sleep does not seem strictly necessary for dreaming.

A crucial involvement of cortical areas has been substantially confirmed, about one century after, by Doricchi and Violani [9] who evaluated the sites of the lesions producing cessation, alteration or maintenance of dreaming following cerebral damage. These authors re-evaluated 104 patients with brain lesions, who reported information about their dreams. Their main finding was that the presence of damages in the frontal lobes is not systematically associated with loss of dreaming, and that lesions in the parietal lobes and lesion associated with disconnective syndromes could cause a loss of dreaming without notable hemispheric asymmetry. Moreover, they reported that dream cessation after unilateral left or right damage was as frequent as after bilateral damage. Since a lesion in either hemisphere could be sufficient to cause dream loss, no simple relation between either hemisphere (i.e., unilateral brain lesions) and dreams can be posited, so that they proposed that the right hemisphere provides the core material for the dreams, whereas the left hemisphere provides the means of decoding it.

The notion that mechanisms underlying dreaming are generated and mediated by specific cortical areas was strongly supported by further re-evaluations of lesion studies allowing to outline the network of cortical areas involved in dreaming. Unilateral (or, in a few cases, bilateral) injuries in or near the temporo-parieto-occipital junction were associated to a complete loss of dreaming, suggesting that this area might be essential for dreaming itself [5]. This finding is crucial, since it has been long time recognized that the cortical network for spatial representation is centered in the inferior parietal lobe, that subserves various cognitive processes involved in waking mental imagery. As a matter of fact, patients with such damage typically show a decline in waking visuospatial abilities [10].

A further systematic relation was found for another cerebral system, since bilateral lesions in the ventromesial frontal white matter cause complete cessation of dreaming [11]. This area plays a crucial role in the interactions between the basal forebrain and limbic structures, on one hand, and many parts of the frontal cortex, on the other hand. Sparse observations showed that this lesion site coincides with the region that was targeted in modified (orbitomesial) prefrontal leukotomy and, interestingly, that an average 80% incidence of nearly complete loss of dreaming was reported in the cases of prefrontal leucotomy [e.g., 12,13]. Moreover, a damage along this system typically produces disorders characterized by reduced interest, initiative, imagination, and ability to plan ahead, while a chemical activation of this circuit (e.g., through Levodopa) stimulate not only positive psychotic symptoms but also excessive, unusually vivid dreaming and nightmares [13]. Altogether, these observations provide support to the view that also the mesocortical–mesolimbic dopamine system may have a causal role in the generation of dreams. In keeping with this hypothesis, it has been reported that lesions in the medial prefrontal cortex, the anterior cingulate cortex and the basal forebrain, caused an increased frequency and vividness of dreams often accompanied by a breakdown of the distinction between dreaming and waking cognition. This suggests that the hallucinated, disoriented and delusional quality of dream cognition may be associated with the inhibition of these structures during sleep [11]. Conversely, some parts of the brain seem inessential for dreaming sleep. Indeed, it has been shown that lesions in the dorsolateral prefrontal cortex can cause waking deficits of self-monitoring and disorders of volitional control, but do not affect dreaming [5]. This last finding is reminiscent of the attenuated volition and other executive deficiencies of dream cognition. Moreover, lesions in ventromesial occipitotemporal (visual association) cortex caused unimodal deficits of dream imagery (in association with identical deficits of waking imagery), whereas lesions in the primary visual cortex have no effect on dreams [11].

Altogether, these data showed that specific brain lesions determine specific loss or alteration of dreaming. Therefore, based on the observations of 332 clinical cases with confirmed cerebral lesions, Solms [11] proposed a nosology for the brain-lesion related disorders of dreaming identifying four major disorders of dreaming. In the syndrome termed "visual anoneira" (or nonvisual dreaming), a bilateral medial occipito-temporal lesion produces full or partial loss of dream visual imagery. The second syndrome, "global anoneira" (or global cessation of dreaming) is

characterized by a total cessation of dreaming in presence of a preserved vision; this disorder was associated to posterior cortical or deep bilateral frontal lesions. The third syndrome proposed is the "anoneirognosis" (or dream-reality confusion), due to frontal–limbic lesions. The patients affected by this disorder are impaired at distinguishing internally generated experiences, such as their dreams, from externally driven percepts. Finally, the syndrome of "recurring nightmares" was characterized by frequent nightmares with a repetitive theme. Recurring nightmares often appeared in the presence of a temporal-limbic seizure activity, with stereotyped nightmares accompanying complex-partial seizures in some cases.

Taken together, these clinical investigations suggest that neurological diseases result in specific alterations of dream experience and that, vice versa, dream disorders can also inform about their underlying neuropathological processes [14].

To summarize, the evaluation of brain damaged patients seems to support the hypothesis that dreaming is not an intrinsic function of REM sleep (or of the brainstem mechanisms that control it), but results from various forms of cerebral activation during sleep. Thus, neuropsychological evidence strongly indicates that dreaming and REM sleep can be dissociated.

3. Neuroimaging studies: functional measures

Neuroimaging techniques like Positron Emission Tomography (PET), using $H_2{}^{15}O$ measurements of regional cerebral blood flow (rCBF), have allowed to describe a functional neuroanatomy of human sleep and the possible correlates of dream features. Although consistent evidence has been provided on dream recall upon awakenings from any stage of NREM sleep [2,4], neuroimaging studies mostly focused their attention on the peculiar association between REM sleep and dreaming, in order to identify which dream characteristics are related to the patterns of cerebral activity observed during REM sleep.

Some PET studies identified networks of areas that seem responsible of the modulation of distinct characteristics of dreams. According to the lesion studies, it has been suggested that the perceptual aspects of dreams would be related to the activation of posterior (occipital and temporal) cortices during REM sleep, and that the strong activation of high-order occipito-temporal visual cortex would explain the vivid visual imagery during dreams [e.g., 15].

The emotional features in dreams would be related to the activation of amygdalar complexes, orbitofrontal cortex, and anterior cingulate cortex [e.g., 16,17,18]. Secondly, the relative hypoactivation of the prefrontal cortex would explain the alterations in logical reasoning, working memory, episodic memory, and executive functions which characterize dream reports obtained from REM sleep awakenings [e.g., 16]. Therefore, the origin of the dream emotionality would derive from the limbic activation, while the REMS frontal deactivation may cause the appearance of dream-associated executive impairment. In addition, it has been found [e.g., 15,19] a marked activity in the hippocampal formation, which seems to confirm that the activation of mesio-temporal areas during REM sleep would account for the memory content commonly found in dreams. Functional neuroanatomic studies strongly supported a metabolic distinction between the different sleep states, in fact NREM sleep showed marked decreases in activation levels compared to REM sleep. More specifically, it has been found a prominent thalamic deactivation and a concomitant regional deactivation in the pontine brainstem, orbitofrontal cortex, anterior cingulate cortex, and lateral prefrontal cortex [e.g., 15,20]. Notably, the main finding is the metabolic decline of the central core structures (brainstem, thalamus), which are known to play a role in the generation of the slow oscillations of NREM sleep [21]. In more detail, thalamic activation was found to decline significantly in concomitance with increased delta EEG activity; moreover, an additional decline resulted associated with increased spindle-frequency activity when the decrements associated with delta were subtracted [22]. This pattern of decline has been interpreted as reflecting the progressive deactivation of the reticular activating system that accompanies deepening NREMS. In conclusion, the regional pattern of deactivation in NREMS sharply contrasts with the regional activation of these same regions (i.e., thalamus, pontine brainstem, anterior cingulate cortex) in REMS, supporting the traditional notion that more story-like affect-laden dreams are attributable to REMS more than NREMS.

Although neuroimaging techniques yielded new body of evidence which helped to better describe the brain mapping of human sleep stages, the functional neuroanatomy of dreaming remains almost speculative. In fact, only the study by Maquet et al. [16] evaluated dream recall in association to PET scans (i.e., in subjects awakened by REM sleep after the collection of PET measures, and with the presence of a dream report). This means that a systematic evaluation of functional imaging measures before awakenings from different sleep stages, with and without dream recall, is still lacking.

4. Neuroimaging studies: morphoanatomical measures of subcortical nuclei

Neuroimaging measures and intracranial recordings delineate a fairly coherent picture of how two deep grey matter structures may play a pivotal role in dreaming: the hippocampus and the amygdala.

The specific activity of the hippocampus should mediate the partial reproduction of memories of events occurred during wakefulness into dream contents. The first evidence in favour of this hypothesis was provided by studies using intracranial recordings in epileptic patients, which showed that rhinal-hippocampal connectivity mediates memory formation in the waking state [23], and there is a close association between a successful recall of a dream upon awakening from a period of REM sleep and an enhancement in rhinal-hippocampal and intrahippocampal EEG connectivity during such period [24]. Both these findings suggest that the ability to memorize dreams may be related to a coordinated activity between the neocortex and the hippocampus.

The amydgala is involved in both the control of the encoding and retrieval of emotional memories and the expression of emotions during wakefulness [25]. Therefore, it may be also involved in the processing of emotionally significant memories during sleep and the emotional load in dreams might be related to its activation level. Consistent with this hypothesis, a higher amygdalar activity during REMS and NREMS [21,26] compared to wakefulness, and a bilateral amygdalar activation in subjects capable to report a dream upon awakening from REM sleep have been reported [21].

However, neuroimaging studies appear limited by their very low temporal resolution, bringing about an intrinsic difficulty in evaluating changes on a much longer time scale, as well as by the implicit (but untenable) assumption of a one-to-one coincidence of dreaming and REM sleep. On the other hand, intracranial studies may be limited by the availability of pre-surgical pharmacoresistant epileptic patients and by some cautions in the generalization of findings to normal subjects.

Several recent findings converge to indicate that the structural characteristics of some subcortical nuclei (i.e., hippocampus and amygdala) are associated with cognitive and emotional processing in waking tasks [27–29]. This "structural" approach may also represent a methodological improvement capable of overcoming some long-standing obstacles in the understanding of the neural

correlates of dreaming by "functional" approaches. Indeed, these approaches inevitably combine the low temporal resolution of PET and Functional Magnetic Resonance Imaging (fMRI) methods with an indirect access to dreaming through the collection of dream reports upon awakening from sleep. According to this view, individual volumetric and ultrastructural measures of hippocampus and amygdala, which are stable over time, may be related to similarly stable (i.e., *trait-like*) individual features of dream experience. This prediction has been confirmed by a recent microstructural analysis of Magnetic Resonance (MR) brain scans and of Diffusion Tensor Imaging (DTI) analysis of the hippocampus and the amygdala [30]. These methods allowed to measure the volume of grey matter and its microstructural alterations, as expressed by reduced cellular barriers that restrict the free diffusion of water molecules in tissues. The main results showed the presence of a dissociation between some quantitative and qualitative aspects of dream reports. The measures of inter-individual differences in the brain tissue of the hippocampus–amygdala complex were directly related to emotional load, bizarreness, and vividness of dreaming, whereas no correlation with the quantitative measures (i.e., the mean number of dreams recalled per day) of dream reports was found. More in details, bizarreness of dream reports was negatively correlated with the left amygdala volume and positively correlated with the right amygdala microstructural integrity, while a lower emotional load was correlated with a decreased microstructural integrity of the left amygdala. Weaker relationships were also reported between bizarreness and hippocampal measures.

Hence, these findings provide an indirect support to the hypothesis that the amygdalar and hippocampal structures, and their relationship with memory and emotional sources, are crucially involved in dream experience. In order to provide insightful hints into the fundamental neural correlates of dreaming in humans, future studies should detail the subcortical networks underlying the peculiar features and organization of dream experience.

5. Local (cortical) electrophysiology of sleep and dream recall

In the last decades, a large body of evidence corroborated the hypothesis that sleep is not a spatially global and uniform state. Indeed, the application of the technical advancements (i.e., multichannel sleep EEG and Fast Fourier Transform – FFT – analyses of EEG) allowed to evaluate the spatio-temporal dynamics EEG during sleep, showing that the homeostatic dynamics of EEG power in the recovery process are intrinsically local and did not involve the whole cerebral cortex to the same extent [31]. It has been reported that the main correlate of sleep intensity and the marker of sleep need (expressed by the amount of slow wave activity, SWA) has a regional modulation, since mostly affects the frontal cortex [e.g., 32,33]. Furthermore, the "local sleep theory" posits that sleep depends on past activity, so that its local features should be use-dependent. Indeed, it has been shown that experience-dependent plasticity in specific neural circuits during wakefulness induces localized changes of SWA during subsequent sleep, highlighting the presence of specific regional effects of learning and plasticity on EEG sleep measures [e.g., 34,35,36].

The framework of the local, use-dependent, theory of sleep may provide further insights into the process of dream generation, and may shed light on the relationship between episodic memories, cortical activity, and dreaming. Since dream recall regards such a peculiar form of episodic information as dream content, which is encoded in declarative memory during sleep, the retrieval of oneiric contents could share some electrophysiological mechanisms with successful episodic memory encoding of the awake brain. With respect to this general view, a large body of findings point to that cortical theta oscillations during wakefulness act to temporally order individual memory representations [37]. Intracranial recordings in humans have confirmed that significant increases in frontal theta oscillations during the encoding phase predict subsequent recall [38].

According to the hypothesis that theta oscillations plays a pivotal role in memory encoding and that wakefulness and sleep share some neurophysiological mechanisms involved in successful episodic memory encoding, Marzano et al. [39] provided electrophysiological evidence that stage-specific cortical brain oscillations in the 5 min of sleep before morning awakening (from REMS and NREMS) are predictive of a successful dream recall. In particular, higher frontal theta activity during sleep predicted subsequent dream recall upon the awakenings from REM sleep. Differently, a lower alpha oscillatory activity in correspondence of right temporo-parietal areas predicted a successful dream recall upon the awakenings from stage 2 sleep. These findings provide a direct support to the notion that the intrinsic differences in the basic electrophysiology of the NREM stage 2 and REM sleep, and their regional changes, may determine which cortical EEG oscillation (i.e., frontal theta or right temporal alpha) is predictive of dream recall upon awakening. In other words, closer times in collecting dream report lead to the larger differences.

Hence, the changes of cortical oscillatory activity during sleep localized over the frontal and temporo-occipital regions appear indicative of the involvement also in dreaming of those regions which control successful memory encoding in waking. This suggests an interdependent regulation of two neuronal circuits involved in the ability to recall a dream upon awakenings from REM and NREM sleep (the hippocampo-cortical circuit and the thalamo-cortical circuit, respectively) and makes it further plausible that the neurophysiological mechanisms underlying the encoding and recall of episodic memories remain the same across different states of consciousness.

6. Concluding remarks and future perspectives

Neurobiological evidences discussed in this review, by integrating findings from different approaches and methods, depicts a fairly coherent picture that emphasizes the continuity between cortical and subcortical mechanisms of waking and sleep cognitive activity [40,41]. Regional EEG results suggest that the electrophysiological mechanisms involved in encoding and recall of episodic memories across wakefulness and sleep are the same [39]. Brain lesions and functional neuroanatomy of healthy subjects identify two cortical systems crucial for dream generation, which are also involved in waking mental imagery and visuospatial abilities (i.e., the temporo-parieto-occipital junction) and in waking encoding and retrieval of episodic memories (i.e. the ventromesial regions of prefrontal cortex) [e.g., 5,21]. Morphoanatomy of deep grey matter structures shows direct relationships between specific qualitative features of dreaming and volumetric and ultrastructural measures of the hippocampus–amygdala complex, that is the structures involved during wakefulness in both the control of the encoding and retrieval of emotional memories and the expression of emotions [30]. Finally, intracranial recordings of epileptic patients show that rhinal-hippocampal connectivity mediates memory formation in waking as well as during sleep [23,24].

These converging pieces of clinical and experimental evidence, although not definitive, support the general view that the neurophysiological mechanisms underlying episodic/declarative memory formation remain the same across different states of consciousness and, thus, their functioning also during sleep may be studied.

Acknowledgements

This work was partly supported by the Compagnia di San Paolo, Programma Neuroscienze 2008/09 (3889 SD/sd, 2008.1300 to L.D.G), in the form of a fellow grant to C.M., and by the Fondazione Cassa di Risparmio di Bologna (Project n. 2008.2443 to C.C.).

References

[1] Dement W, Kleitman N. Cyclic variations in EEG during sleep and their relation to eye movements, body mobility and dreaming. Electroencephalogr Clin Neurophysiol 1957;9:673–90.
[2] Nielsen T. A review of mentation in REM and NREM sleep: covert REM sleep as a possible reconciliation of two opposing models. Behav Brain Sci 2000;23:851–66.
[3] Nielsen T. Chronobiological features of dream production. Sleep Med Rev 2004;8:403–24.
[4] Fagioli I. Mental activity during sleep. Sleep Med Rev 2002;6:307–20.
[5] Solms M. Dreaming and rem sleep are controlled by different brain mechanisms. Behav Brain Sci 2000;2:843–50.
[6] Landolt HP, Raimo EB, Schnierow BJ, Kelsoe JR, Rapaport MH, Gillin JC. Sleep and sleep electroencephalogram in depressed patients treated with phenelzine. Arch Gen Psychiatry 2001;58:268–76.
[7] Wilbrand H. Die Seelenblindheit als Herderscheinung und ihre Beziehung zur Alexie und Agraphie. Bergmann: Wiesbaden; 1887.
[8] Muller F. Ein Beitrag zur Kenntniss der Seelenblindheit. Arch Psychiatr Nervenkrank 1892;24:856–917.
[9] Doricchi F, Violani C. Dream recall in brain-damaged patients: a contribution to the neuropsychology of dreaming through a review of the literature. In: John S, Antrobus MB, editors. The neuropsychology of sleep and dreaming. Hillsdale, NJ: Erlbaum; 1992. p. 99–129.
[10] Kerr NH, Foulkes D. Right hemispheric mediation of dream visualization: a case study. Cortex 1981;17:603–9.
[11] Solms M. The neuropsychology of dreaming: a clinico-anatomical study. Mahwah, NJ, US: Lawrence Erlbaum Associates; 1997.
[12] Frank J. Clinical survey and results of 200 cases of prefrontal leucotomy. J Ment Sci 1946;92:497–508.
[13] Partridge M. Pre-frontal leucotomy: a survey of 300 cases personally followed for 1–3 years. Oxford: Blackwell; 1950.
[14] Schwartz S, Dang-Vu TT, Ponz A, Duhoux S, Maquet P. Dreaming: a neuropsychological view. Schweiz Arch Neurol Psychiatr 2005;156: 426–39.
[15] Braun AR, Balkin TJ, Wesenten NJ, Carson RE, Varga M, Baldwin P, et al. Regional cerebral blood flow throughout the sleep-wake cycle. An H2(15)O PET study. Brain 1997;120:1173–97.
[16] Maquet P, Peters J, Aerts J, Delfiore G, Degueldre C, Luxen A, et al. Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. Nature 1996;383:163–6.
[17] Maquet P, Franck G. REM sleep and amygdala. Mol Psychiatr 1997;2:195–6.
[18] Hobson JA, Pace-Schott EF, Stickgold R, Kahn D. To dream or not to dream? Relevant data from new neuroimaging and electrophysiological studies. Curr Opin Neurobiol 1998;8:239–44.
[19] Nofzinger EA, Mintun MA, Wiseman M, Kupfer DJ, Moore RY. Forebrain activation in REM sleep: an FDG PET study. Brain Res 1997;770:192–201.
[20] Maquet P, Degueldre C, Delfiore G, Aerts J, Peters JM, Luxen A, et al. Functional neuroanatomy of human slow wave sleep. J Neurosci 1997;17:2807–12.
[21] Maquet P. Functional neuroimaging of normal human sleep by positron emission tomography. J Sleep Res 2000;9:207–31.
[22] Hofle N, Paus T, Reutens D, Fiset P, Gotman J, Evans AC, et al. Regional cerebral blood flow changes as a function of delta and spindle activity during slow wave sleep in humans. J Neurosci 1997;17:4800–8.
[23] Fell J, Klaver P, Lehnertz K, Grunwald T, Schaller C, Elger CE, et al. Human memory formation is accompanied by rhinal–hippocampal coupling and decoupling. Nat Neurosci 2001;4:1259–64.
[24] Fell J, Fernández G, Lutz MT, Kockelmann E, Burr W, Schaller C, et al. Rhinal-hippocampal connectivity determines memory formation during sleep. Brain 2006;129:108–14.
[25] Misane I, Tovote P, Meyer M, Spiess J, Ogren SO, Stiedl O. Time-dependent involvement of the dorsal hippocampus in trace fear conditioning in mice. Hippocampus 2005;15:418–26.
[26] Nofzinger EA, Buysse DJ, Miewald JM, Meltzer CC, Price JC, Sembrat RC, et al. Human regional cerebral glucose metabolism during non-rapid eye movement sleep in relation to waking. Brain 2002;125:1105–15.
[27] Maguire EA, Gadian DG, Johnsrude IS, Good CD, Ashburner J, Frackowiak RS, et al. Navigation-related structural change in the hippocampi of taxi drivers. Proc Natl Acad Sci USA 2000;97:4398–403.
[28] Bohbot VD, Lerch J, Thorndycraft B, Iaria G, Zijdenbos AP. Gray matter differences correlate with spontaneous strategies in a human virtual navigation task. J Neurosci 2007;27:10078–83.
[29] Iaria G, Lanyon LJ, Fox CJ, Giaschi D, Barton JJ. Navigational skills correlate with hippocampal fraction anisotropy in humans. Hippocampus 2008;18:335–9.
[30] De Gennaro L, Cipolli C, Cherubini A, Assogna F, Cacciari C, Marzano C, et al. Amygdala and hippocampus volumetry and diffusivity in relation to dreaming. Hum Brain Mapp 2011;32:1458–70.
[31] Ferrara M, De Gennaro L. Going local: insights from EEG and stereo-EEG studies of the human sleep-wake cycle. Curr Top Med Chem 2011;11:2423–37.
[32] Werth E, Achermann P, Borbely AA. Brain topography of the human sleep EEG: antero-posterior shifts of spectral power. Neuroreport 1996;8:123–7.
[33] Marzano C, Ferrara M, Curcio G, De Gennaro L. The effects of sleep deprivation in humans: topographical electroencephalogram changes in non-rapid eye movement (NREM) sleep versus REM sleep. J Sleep Res 2010;19:260–8.
[34] Huber R, Ghilardi MF, Massimini M, Tononi G. Local sleep and learning. Nature 2004;430:78–81.
[35] Moroni F, Nobili L, Curcio G, De Carli F, Tempesta D, Marzano C, et al. Procedural learning and sleep hippocampal low frequencies in humans. Neuroimage 2008;42:911–8.
[36] De Gennaro L, Fratello F, Marzano C, Moroni F, Curcio G, Tempesta D, et al. Cortical plasticity induced by transcranial magnetic stimulation during wakefulness affects electroencephalogram activity during sleep. PloS ONE 2008;3(6):e2483.
[37] Nyhus E, Curran T. Functional role of gamma and theta oscillations in episodic memory. Neurosci Biobehav Rev 2010;34:1023–35.
[38] Sederberg PB, Kahana MJ, Howard MW, Donner EJ, Madsen JR. Theta and gamma oscillations during encoding predict subsequent recall. J Neurosci 2003;23:10809–14.
[39] Marzano C, Ferrara M, Mauro F, Moroni F, Gorgoni M, Tempesta D, et al. Recalling and forgetting dreams: theta and alpha oscillations during sleep predict subsequent dream recall. J Neurosci 2011;31:6674–83.
[40] Schredl M. Continuity between waking and dreaming: a proposal for a mathematical model. Sleep Hypn 2003;5:26–40.
[41] Schredl M. Dreams in patients with sleep disorders. Sleep Med Rev 2009;13:215–21.