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Picture representation during REM dreams: A redox molecular hypothesis

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ABSTRACT

A novel molecular hypothesis about visual perception and imagery has recently been proposed (Bókkon, 2009; BioSystems). Namely, external electromagnetic visible photons are converted into electrical signals in the retina and are then conveyed to V1. Next, these retinotopic electrical signals (*spike-related electrical signals along classical axonal-dendritic pathways*) can be converted into synchronized bioluminescent biophoton signals (*inside the neurons*) by neurocellular radical reactions (*redox processes*) in retinotopically organized V1 mitochondrial cytochrome oxidase-rich visual areas. The bioluminescent photonic signals (*inside the neurons*) generated by neurocellular redox/radical reactions in synchronized V1 neurons make it possible to produce computational biophysical pictures during visual perception and imagery. Our hypothesis is in line with the functional roles of reactive oxygen and nitrogen species in living cells and states that this is not a random process, but rather a strict mechanism used in signaling pathways. Here, we suggest that intrinsic biophysical pictures can also emerge during REM dreams.

1. Introduction

It was suggested that the brain can operate by means of pictures (*holograms*) during informational processes by biophotons, and the properties of the homeothermic state make the development of explicit memories in our brain possible (Bókkon, 2003, 2005, 2006). In addition, it was proposed that phosphenes are intrinsic perceptions of induced bioluminescent photon emissions (*e.g., via external electric or magnetic fields, etc.*) or spontaneous enhanced bioluminescent photon emissions in cells in various parts of the visual system (Bókkon, 2008; Bókkon and Vimal, 2009). We have put forward a new molecular hypothesis about the natural biophysical substrate of visual perception and imagery (see Fig. 1) (Bókkon, 2009; Bókkon and D'Angiulli, 2009; Bókkon et al., 2009). This hypothesis describes that external electromagnetic visible photons are converted into electrical signals in the retina and are then conveyed to the V1 area (*primary visual cortex* or *striate cortex that is the first station of the visual pathway to receive an integrated signal from the two eyes*). These V1 retinotopic electrical signals can subsequently be converted to controlled bioluminescent photon signals by mitochondrial and cellular redox reactions, which make it possible to produce biophysical pictures in retinotopically organized mitochondrial cytochrome oxidase-rich visual areas during visual perception and imagery. Here, we suggest that intrinsic biophysical pictures can also emerge in retinotopic visual areas during REM (Rapid eye movement) dreaming.

2. REM Sleep

REM sleep is a behavioral state characterized by the activation of cortical and hippocampal EEGs (electroencephalogram), rapid eye movements and muscle atonia. Although rapid eye movement is a prominent feature of REM sleep (also called paradoxical sleep), its origins and functional significance are still poorly understood in humans. Most dreams occur during REM sleep but when they are not often detected during other sleep stages some criteria of REM sleep are present (Takeuchi et al., 2001).

During paradoxical sleep, REMs are closely related to the occurrence of so-called ponto-geniculo-occipital (PGO) waves, *i.e.*, prominent phasic activities recorded throughout the brain but predominantly in the pons (P), the lateral geniculate bodies (G) and the occipital cortex (O). PGO waves are a fundamental part of REM sleep. They begin as electrical pulses from the pons, then move to the lateral geniculate nucleus (LGN) and finally end up in the primary visual cortex of the occipital lobe. The appearance of these waves is most prominent in the period right before REM sleep, and they are assumed to be intricately involved with eye movement during wake and sleep cycles. However, there is debate in dream research as to whether PGO waves or cortical arousal during sleep underlie the biological mechanisms of dreaming. Our cur-

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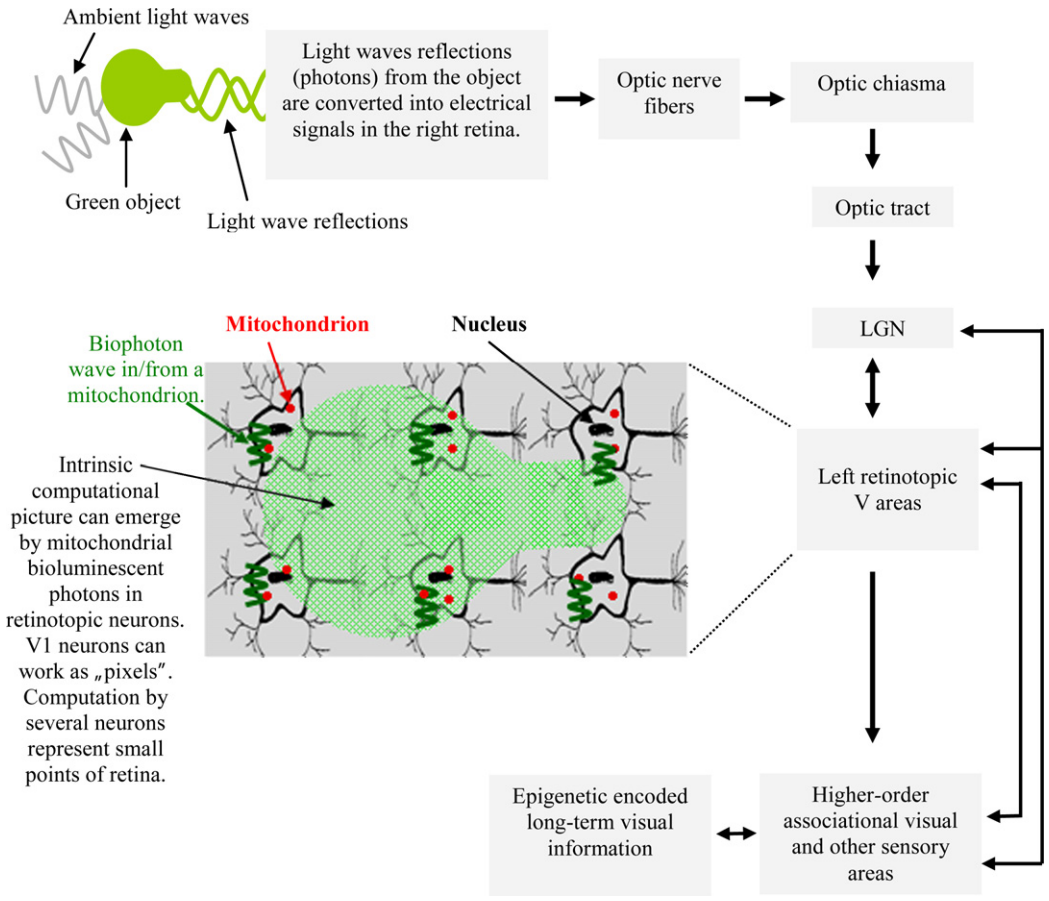

Fig. 1. Redox molecular hypothesis about the natural biophysical substrate of visual perception and imagery. During visual perception, light photons from object are converted to electrical signals in the retina and are then conveyed to V1. Retinotopical V1 electrical signals can be converted into bioluminescent photons in V1 neurons by mitochondrial and cellular radical/redox processes. So, we can get an intrinsic computational picture about object within retinotopical V1. This hypothetic model is limited to a static object with a color. In reality, computation of different kinds of visual information (motion, contrast, size, orientation, spatial frequency, texture type) is much more complex process. Long-term visual memory is not stored as pictures but as epigenetic encoded information, which is regulated mostly by mitochondrial free radicals and cellular redox processes. This epigenetic encoded DNA information can be rewritten or retrieved by mitochondrial free radicals and cellular redox processes during visual imagery. (For interpretation of the references to color in the artwork, the reader is referred to the web version of the article.)

rent research moves toward combined brainstem/forebrain models of sleep cognition (Stuart and Conduit, 2009; Pace-Schott et al., 2003).

REM sleep is classified into two categories: tonic and phasic events. The tonic events include postural muscle atonia and desynchronized EEG. The phasic events include PGO waves, phasic interruptions of respiration (a pause in diaphragm activity that lasts ~20–100 ms), rapid eye movements, and muscle twitches (Hunt et al., 1998; Nelson et al., 1983). According to Hong et al. (1995), the same cortical areas are involved in eye movements in REM sleep and wakefulness, suggesting that REM eye movements are saccadic scans of targets in the dream scenes. Cantero et al. (1999) hypothesized that REM and waking brain states use the same general mechanisms to produce mental images.Serotonin, histamine and norepinephrine neurotransmitters that are used during waking hours, are decreased during non-REM sleep and stop altogether in REM (McCarley, 2007). This neurotransmitter shutdown causes REM atonia, a state in which the motor neurons are not stimulated and thus the body's muscles do not move. In contrast to cells that use serotonin, norepinephrine and histamine, most dopaminergic neurons do not appear to alter their discharge rate across the sleep cycle (Miller et al., 1983; Shouse et al., 2000).

Acetylcholine plays a major role in regulating behavioral arousal and cortical electroencephalographic activation. Cholinergic neurons of the basal forebrain supply the neocortex with acetylcholine. Cortical acetylcholine release is greatest during waking and REM sleep and is reduced during non-REM sleep (Vazquez and Baghdoyan, 2001).

Neurotransmitter-mediated excitation and inhibition of brainstem cells is important for the regulation of wake-fullness and REM sleep (Datta, 2000; Thakkar et al., 1998). REM sleep is accompanied by a selective increase in GABA (gamma-aminobutyric acid) release, but not by a change in glutamate or glycine release in the dorsal raphe (Nitz and Siegel, 1997; Kaur et al., 2001). The most widely accepted belief for the regulation of REM sleep emphasizes the reciprocal inhibitory interactions between monoaminergic and cholinergic neurons in the pontine brainstem (Hobson, 1992). In particular, cholinergic agonists promote REM sleep and muscarinic antagonists and monoamines inhibit REM sleep. However, this belief has been questioned because in addition to the aforementioned cholinergic and aminergic neuronal populations, some additional neurotransmitter systems participate in the control of REM sleep (Mallick et al., 2001).

3. REM Dreams as Cognitive-like Processes

REM dreams can be cognitive-like processes. Hobson interpreted synchronization in the gamma frequency as a reflection of our cognitive processing during dreams and states of wakefulness (Hobson et al., 1998). If the same stimuli conditioned during wakefulness were used for the duration of REM, memory-formation can be enhanced (Hars et al., 1985). Classical conditioning can be

achieved during REM sleep and can be developed after sleep (Maho and Bloch, 1992). We are able to see our dreams from first-person perspective, indicating that dreams a feature of consciousness. REM dreams are not random processes because they bear a high amount of narrative. Dream imagery is not generated by chaotic activation of the forebrain, but rather by specific forebrain mechanisms with dreams and complex cognitive processes (Solms, 2000).

4. Visual Imagery and REM

REM sleep EEG readings are similar to EEGs during wakefulness (Lliná and Ribary, 1993). During phasic-REM, there is an increase in gamma power and a concomitant decrease in theta, alpha and beta EEG activities (Jouny et al., 2000). During phasic-REM, the spectral power of the background alpha activity is decreased over occipital brain regions (Cantero et al., 2000). Alpha activity (8–13 Hz) attenuation in occipital regions is an electrophysiological index of cortical activation associated with waking visual imagery and visual attention. Thus, there is active visual processing caused by the complex mental imagery during phasic-REM. It is probable that the generation of visual imagery shares general mechanisms during waking and REM sleep. Braun et al. (1998) suggested that the brain mechanisms underlying REM sleep are visual association cortices and their paralimbic projections, operating as a closed system dissociated from the regions of the visual hierarchy, which mediate interactions with the external world. Visual imagery during REM sleep, in comparison to wakefulness, can occur through the inverse pathway of visual information processing (Cantero et al., 1999; Ogawa et al., 2006).

5. Extrastriate and Striate (V1) Cortex Activation During REM Dreams

Although numerous studies have revealed activation of the visual cortex during REM sleep (Braun et al., 1998; Peigneux et al., 2001), there is controversy in the functional imaging literature about whether there is V1 activation during REM. According to Igawa et al. (2001), in spite of the deactivation of striate cortex (V1) during REM sleep, the activated areas of the visual cortex are broader during REM sleep than under visual stimulation during wakefulness. The controversy about whether there is activation of V1 during REM dreams may be due to different tasks, baseline conditions and analysis methods used in the different studies. It seems that visual imagery during REM sleep uses the same (or very similar) neural systems as those used in wakefulness (Cantero et al., 2000). However, according to the latest event-related fMRI (functional Magnetic Resonance Imaging) analyses, the pontine tegmentum, ventroposterior thalamus, primary visual cortex (V1), putamen and limbic areas are activated in association with REMs (Hong et al., 2009; Miyauchi et al., 2009).

6. Brain Temperature, Mitochondrial Activity, Oxygen and Glucose Utilization During REM Dreams

There are increases in oxygen and glucose utilization, brain temperature, local brain blood flow and neuron activity during REM (Shvets-Ténéta-Gurii et al., 2001). REM sleep metabolic rates are as high as those seen in wakefulness. The mean levels of mitochondrial cytochrome oxidase activity and blood volume during REM sleep significantly exceed those during waking and slow-wave sleep (Vern et al., 1988). Mitochondrial oxidative phosphorylation dominates in waking and REM sleep, while aerobic glycolysis dominates during non-REM sleep (Shvets-Ténéta-Gurii et al., 2003).

7. Neural Activity-dependent Biophoton Production

Ultraweak bioluminescent photons (or biophotons) are continuously emitted by living cells without any excitation (Kataoka et al., 2001; Van Wijk, 2001; Isojima et al., 1995; Kobayashi et al., 1999a,b; Brizhik, 2008; Yoon et al., 2005) Bioluminescent biophotons are originated from the diverse free radical reactions such as lipid peroxidation, mitochondrial respiration chain, oxidation of tyrosine and tryptophan residues in proteins, *etc.* (Nakano, 2005; Thar and Kühl, 2004).

A great deal of experiments provided strong evidence that ROS (reactive oxygen species) and RNS (reactive nitrogen species) and their derivates act as fundamental signals during physiological (*pathophysiological*) processes in cells and the brain (Dröge, 2002; Knapp and Klann, 2002; Valko et al., 2007; Tong, 2007). Since free radicals can act as secondary messengers, and the bioluminescent photons are originated from various free radical reactions, it means that biophoton emission of cells may also be a controlled process.

There is a direct correlation between the biophoton intensity and neural metabolic activity in rat hippocampal slices (Isojima et al., 1995). A few experimental data have demonstrated neural activity-dependent biophotonic activity in rats' brain. Namely, according to in vivo experiments, the biophoton emission from a rat's brain is associated with cerebral energy metabolism, EEG activity, and oxidative metabolism (Kobayashi et al., 1999a). Biophoton emission from the brain slices depended on temperature and oxygen concentrations. The increase of biophoton activity is related to the membrane depolarization induced by a high concentration of K^+, and the decrease of biophoton activity is related to the elimination of extracellular Ca^{2+} and the suppressed neural activity by tetrodotoxin a voltage-dependent sodium channel inhibitor in rat's hippocampus slices (Kataoka et al., 2001). In addition, pulsed electric excitation of frog sciatic nerve is reported to induce photon emission (Artem'ev et al., 1967).

In humans, some data suggested that the change of biophoton intensity is related to the consciousness, meditation, and conditions of acupuncture meridians (Dobrin et al., 1969; Kim, 2002; Van Wijk et al., 2005, 2006) suggesting that biophotons could play an important role in the functions of nervous system. However, very recently, Sun et al. (2010) experimentally demonstrated that biophotons (ultraweak bioluminescent photons) can conduct along the neural fibers.

These research data suggest that biophotonic or bio-electronic activities seem not to be an independent biological phenomenon in the nervous system. Thus, we can conclude that neuronal biophoton emission is in direct relationship with neural activity and neurobiochemical processes. Since regulated electrical (*redox/free radical*) signals of neurons could be converted into regulated biophoton signals, external information can emerge not only as electrical signals but also as biophotonic (*ultraweak optical signal*) signals in the brain.

8. Biophysical Picture Representation During Visual Perception and Imagery

There is a long-standing imagery debate in cognitive sciences involving two rival hypotheses: Kosslyn's pictorial theory (*analog or depictive*) (Kosslyn, 1994; Gluck et al., 2007) versus Pylyshyn's tacit knowledge (*symbolic or linguistic*) (Pylyshyn, 2002, 2007) interpretation. Pylyshyn asserts that activation of early visual areas during visual mental imagery is epiphenomenal. Although there is growing evidence that visual perception and visual imagery share common neural substrates in the brain (Borst and Kosslyn, 2008; Slotnick et al., 2005; Klein et al., 2004; Kosslyn et al., 1995), the visual imagery

debate is still unresolved and there is no convincing biophysical substrate that meets the constraints and available evidence on reverse hierarchies as applied to imagery and visual cognition.

We have recently proposed a biophysical mechanism for imagery and visual cognition (Bókkon, 2009; Bókkon and D'Angiulli, 2009). Namely, objects in the visual field are directly represented in the visual cortex by congruent patterns of bioluminescent photons generated by radical processes in retinotopically organized mitochondrial cytochrome oxidase-rich neural networks of V1 (*our hypothesis is represented schematically in* Fig. 1). An important implication is that there is no *homunculus*[1] in our head but that the biophysical image representation is processed as signals to be sent to, and subsequently interpreted by, higher order areas of the brain. Our proposed mechanism also suggests that the phosphene phenomenon is due to the intrinsic perception of induced or spontaneously increased bioluminescent photon emission of cells in various parts of the visual system (Bókkon, 2008). Phosphene lights may be nothing but natural bioluminescent photons originating from retinotopic mitochondrial cytochrome oxidase-rich visual areas. Nevertheless, if it can be verified and proved that perception of cortical induced phosphenes is due to bioluminescent photons, intrinsic regulated biophotons in the visual system can serve as a natural biophysical substrate of visual perception and imagery.

9. Retinal Phosphenes

It was proposed that phosphenes are intrinsic perceptions of induced (*e.g.*, *via external electric or magnetic fields*, *etc.*) or spontaneous enhanced bioluminescent photon emissions in cells in various parts in the visual system (Bókkon, 2008; Bókkon and Vimal, 2009). Recently, Narici et al. (2009) confirmed our prediction (Bókkon, 2008) about retinal phosphenes during space travel. According to Narici et al., phosphenes in space travel are due to the free radicals and chemiluminescent photons induced by ionizing radiation. In other words, free radicals induced by ionizing radiation (*cosmic particles*) can create bioluminescent photons by retinal lipid peroxidation. These photons are then absorbed by the photoreceptors, modify the rhodopsin molecules (*rhodopsin bleaching*) and initiate the photo-transduction cascade, resulting in the sensation of phosphene lights.

We should see that visual circuits that are normally involved in the detection of visual perception are also responsible for the generation of the phosphenes. However, because retinal and cortical induced phosphenes must have a common molecular biophysical basis, the experiments done by Narici et al. can serve as the first step toward our biophysical (*bioluminescent*) picture hypothesis (Bókkon, 2009; Bókkon and D'Angiulli, 2009).

10. Emergence of Biophysical Pictures From Long-term Visual Memory

In biology, the term epigenetics refers to heritable changes in phenotype (*appearance*) or gene expression caused by mechanisms other than changes in the underlying DNA (deoxyribonucleic acid) sequence (*epigenetics refers to regulation of chromatin structure through direct methylation of DNA or post-translational modification of histone proteins, including methylation, acetylation, and phosphorylation*). It is suggested that long-term memory can be coded at the level of modified DNA molecules (*transcription-dependent long-term memory formation*) (Feng et al., 2007; Reul and Chandramohan, 2007; Levenson et al., 2004). However, cognitive functions are performed by complex elements whose function is not restricted to the generation of electrical potentials and transmission of signals to other neurons. Arshavsky (2006) argues that the performance of cognitive functions is based on complex cooperative activity of complex neurons that are carriers of "elementary cognition".

Roubertoux et al. (2003) presented direct evidence for the involvement of mitochondrial DNA in cognitive function. Mitochondria have a fundamental role in maintaining genomic stability and controlling nuclear processes (Desler et al., 2007). Mitochondrial function can be controlled by interactions between nuclear and mitochondrial genes (Roubertoux et al., 2003; Chen et al., 2006). It seems that regulated mitochondrial free radical production can modulate gene expression processes and rewrite nuclear epigenetic information.

According to our molecular biophysical concept (Bókkon, 2009; Bókkon and D'Angiulli, 2009), long-term visual memory is not stored as pictures but as epigenetic encoded information, which is mostly regulated by free radicals and cellular redox processes. Visual perception can perform iterative processes between top-down (*activated long-term epigenetic visual information*) and bottom-up (*from retinal map*) processes in the condition that perceived image and activated long-term visual epigenetic information have similar picture convergence. This epigenetically encoded long-term visual information can be rewritten or retrieved by mitochondrial free radicals and cellular redox processes. We must mention that the emergence of intrinsic dynamic pictures in retinotopic areas by regulated bioluminescent photons is an extremely complex process in reality. In addition, intrinsic biophysical pictures (*images*) are not rigid objects; we can alter images *ad-lib*, making it possible for the visual system to generate irrationally assembled pictures and scenes.

11. Hypothesis: Emergence of Redox-related Biophysical Picture Representation During REM Dreams

We have previously seen that physiological and psychological processes of REM sleep are similar to waking visual imagery. The EEG pattern during REM sleep is basically indistinguishable from that during wakefulness and is characterized by low amplitudes and higher frequencies.

It is well known that the V1, V2 visual areas and many extrastriate visual cortical areas, including V3 and V4, are organized in a retinotopic manner in primates (Kaido et al., 2004; Martínez et al., 1999). V1 and V2 have a detailed map of the retina and hence are said to be topographically (*retinotopically*) well organized. There are several visual areas beyond V1 and V2 known as the prestriate cortex, which have larger receptive fields and cruder topographic organizations. However, V1 and V2 can represent all the principal submodalities of vision such as color, form, motion and depth. V1 and V2 contain similarly scaled retinotopic maps of the visual field and have comparable surface areas (Sincich et al., 2007).

There is active visual processing caused by the complex mental imagery during phasic-REM. The activated areas of the visual cortex become broader during REM sleep compared to visual stimulation during wakefulness (Igawa et al., 2001). According to latest studies, not only extrastriate areas but also V1 are activated during REM (Hong et al., 2009; Miyauchi et al., 2009). Since most dreams have strong visual content (*in non-congenitally blind people*) we can claim that we are visually aware during our dreams. The neurons that represent our visual awareness should be activated when we see something, when we imagine it with closed eyes or when we are asleep and dream about the same thing. We have to emphasize, although our visual imagery can be a consciously directed

[1] The concept of a *homunculus* (Latin for "little man", sometimes spelled "*homonculus*") is frequently used to demonstrate the functioning of a system. In the scientific sense of an unknowable prime actor, it can be viewed as an entity. Who looks at the images in the brain? If we suppose a homunculus, our visual imagery is attended by the occurrence of seeing with the mind's eye.

process, we can also imagine absurd scenes like those seen during REM.

REM dream pictures, similarly to visual imagery, can originate from long-term visual memories by iterative processes (*see more detail about long-term visual memories in* Section 10). However, Jouvet (1998) suggested an idea similar to our hypothesis. He said that there is an iteration process at the DNA level during sleep that maintains and programs hereditary behavior. During REM dream pictures (*same as visual imagery*), long-term epigenetic encoded visual memory is activated and retrieved by neuro-redox processes in a complex manner, which makes it possible to create biophysical pictures in the retinotopically organized mitochondria-rich neurons of visual areas.

It is not an accident that prominent phasic activities are recorded throughout the brain, but primarily in PGO areas. PGO waves initiate as electrical signals from the pons, then move to the LGN, and finally end up in the V1. Consequently, PGO waves can activate retinotopic areas such as LGN, V1, V2, and other extrastriate areas during REM. This allows for the emergence of active intrinsic visual processing, which is produced by the complex mental imagery during phasic-REM.

It was also found that during REM, there are increases in oxygen and glucose utilization, brain temperature, local brain blood flow and neural activity. Moreover, mitochondrial oxidative phosphorylation dominates in paradoxical sleep and waking, and the mean levels of cytochrome oxidase activity during REM sleep significantly exceed those during waking and slow-wave sleep. However, mitochondrial oxidative phosphorylation is one of the main sources of bioluminescent photons generated by regulated redox reactions (*see in* Section 7). Spatiotemporal distributions of mitochondria can create spatiotemporal dynamic patterns of free radicals as well as bioluminescent photons in retinotopic visual neurons.

These findings and observations can support the hypothesis that visual neuro-activation patterns (*spike-related electrical signals along classical axonal-dendritic pathways*) can be converted into synchronized bioluminescent photonic signals (*inside the neurons*) by intra-cellular radical reactions, which can produce intrinsic biophysical pictures in retinotopically organized mitochondrial cytochrome oxidase-rich neurons of V1 and V2 during REM dreams (Fig. 2).

12. Redox-related Biophysical REM Dream Pictures as Sources of Visual Hallucination

It has been proposed that mental status abnormalities are wakeful dreams caused by a sleep and dream-associated disorder (Kavanau, 2001; Saucerman, 1997). According to polysomnographic studies, there is a relationship between visual hallucinations and REM sleep in patients with Parkinson's disease (Nomura et al., 2003). During REM sleep, activation of the visual cortex represents dream-related brain activity (Igawa et al., 2001). The psychological, electrophysiological, blood flow, pharmacological and neurochemical processes of the dream state are very similar to the characteristics observed in schizophrenia (Gottesmann, 2006).

During schizophrenic visual hallucination, there is a loss of control of cortical functioning, including deficits in serotonin, noradrenalin and dopamine mechanisms, which let dream-like pictures break into the waking consciousness. In a recent paper in Progress in Neurobiology (Gottesmann and Gottesman, 2007) several relationships between REM sleep neurobiological criteria and psychiatric diseases are presented. This study also cited the author's concept (Bókkon, 2006) that in patients with schizophrenia, the waking protection process fails and holographic-like dream pictures generated by biophotons erupt into the waking consciousness.

Several independent lines of evidence support a dysfunction of mitochondria in schizophrenia, including mitochondrial hypoplasia and dysfunction of the oxidative phosphorylation system, as well as altered mitochondrial-related gene expression (Ben-Shachar and Laifenfeld, 2004). However, our biophysical picture theory is based on the activity-dependent ultraweak photons of coordinated energetic processes of the mitochondrial network, which can generate intrinsic pictures in the retinotopically organized cytochrome oxidase-rich visual areas.

Furthermore, there is accumulating evidence of dysregulation of antioxidants, redox processes and neurotransmitter (dopamine, serotonin, and noradrenalin) processes in schizophrenia (Abi-Dargham, 2007; Arnsten, 2004; Ohara, 2007; Fendri et al., 2006). However, these dysregulated neurotransmitter processes also have fundamental importance in our hypothesis. For example, neurotransmitters can regulate redox and free radical processes (Noh et al., 1999; Munoz-Castaneda et al., 2006; Liu and Mori, 1993; Andorn and Pappolla, 2001; Boggess and Martin, 1975; Yang et al., 2006; Yasunari et al., 2000) and, as a result, can also control the ultraweak photon emission in the brain and visual areas.

There is tight coupling between mitochondrial organization-movement-activity and synaptic activity (Mattson, 2007). According to the latest experiments, both dopamine and serotonin can regulate mitochondrial movement and determine the distribution of mitochondria in neurons (Chen et al., 2007, 2008). Therefore, information processes are directly linked to mitochondrial redox/energetic and free radical processes within neuronal cells during perceptions and representations.

Visual hallucinations can be due to deafferentation and disintegration of certain visual structures, which leads to an increase in excitability of deafferented neurons (Burke, 2002). This deafferentation can be associated with an increase in spontaneous activity and synchronization of nerve discharges. Thus, hallucination may be considered as a local paroxysm in certain visual structures. Local paroxysms can create dream-like pictures by bioluminescent photons in the retinotopically organized cytochrome oxidase-rich visual areas. These unregulated dream-like pictures by bioluminescent photons can then break into the waking consciousness (Gottesmann and Gottesman, 2007; Bókkon, 2005).

13. Testing the Hypothesis

If it can be proved that conscious cortical retinotopic phosphene lights are bioluminescent (bio)photons, then intrinsic regulated biophotons can serve as a natural biophysical (redox) substrate not only of visual perception and imagery but also of REM dream pictures. However, it is especially difficult to directly test the present hypothesis because the real biophoton intensity within neurons and other cells can be significantly higher than one would expect from the measurements on ultraweak bioluminescence, which is generally measured macroscopically several centimeters from the tissue or cell cultures. Namely, the most significant fraction of natural biophoton emission can be absorbed during natural cellular processes.

A methodology needs to be established to be able to measure in vivo and/or in vitro increases in biophoton emission in human and animal brains during phosphene induction by transcranial magnetic stimulation (TMS) or intracranial current stimulation (ICS). In vivo increases in biophoton emission should be measured in human and animal brains during REM dreams. Further investigations should test whether human subjects experience phosphenes induced by TMS during REM dreams.

In vitro increases in biophoton emission should be measured in brain slices that have been induced using TMS or ICS and similar parameters as those used for phosphene induction.

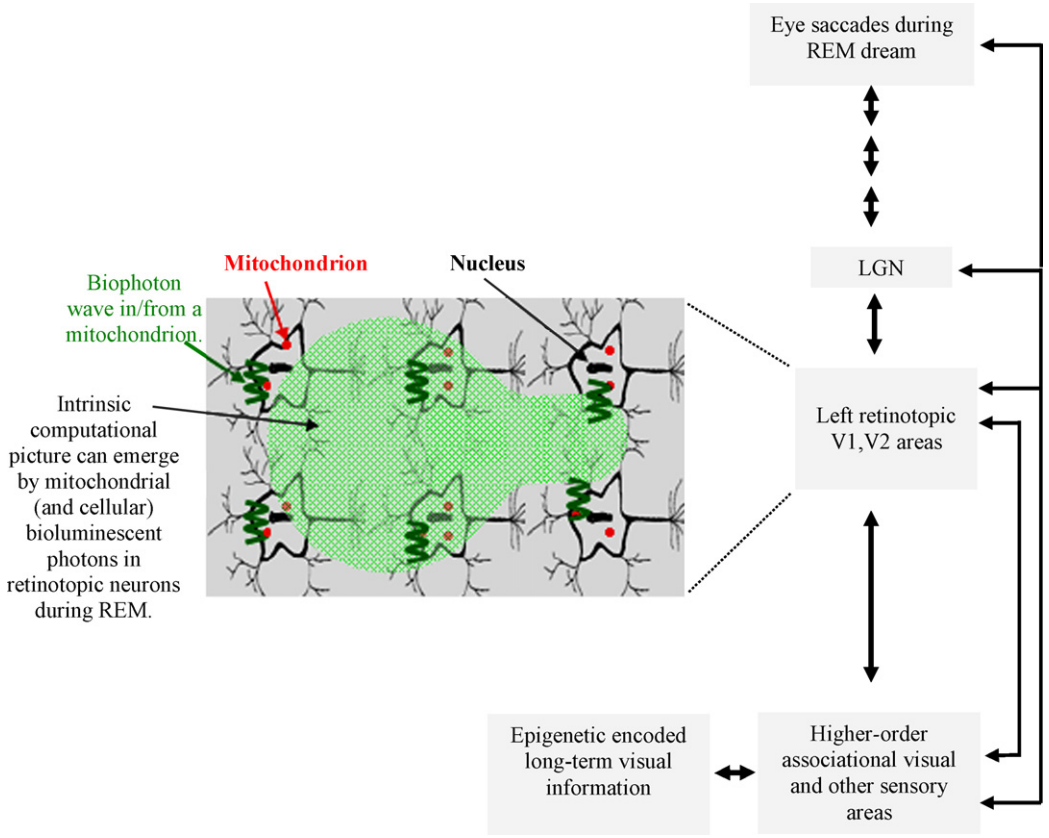

Fig. 2. Redox molecular hypothesis about biophysical picture representation during REM dreams. During REM dreams, activated top-down processes regulate the epigenetic encoded long-term visual information. Then, this epigenetic encoded long-term visual information is retrieved by mitochondrial and cellular redox processes that can re-produce biophysical picture representation of object (to some degree of accuracy) by regulated bioluminescent photons of mitochondrial networks in retinotopically organized V1and V2 neurons. Intrinsic biophysical pictures (images) are not like rigid objects but we can alter images *ad-lib*, which make it possible that the visual system can also generate irrationally assembled pictures and scenes during REM dreams.

Mitochondrial cytochrome oxidase activity is a marker of neural activity. A series of other tests should be conducted to track down cytochrome oxidase pathways and detect mitochondrial flavoprotein or NADH (Nicotinamide adenine dinucleotide) autofluorescence by testing animals in vivo at the same cortical points of phosphene induction during imagery or REM dreams.

14. Summary

In this paper we suggest that intrinsic biophysical pictures can also appear during REM dreams, similarly to visual imagery. The whole argument is summarized below:

- Physiological and psychological processes of REM sleep are similar to waking visual imagery.
- According to latest studies, not only the extrastriate areas, but also retinotopic V1 visual area can be activated during REM.
- Retinotopy is a fundamental organizing part of the visual cortex. The expanded neural representation of the fovea found in the retina and LGN is maintained in the visual cortex.
- The EEG pattern during REM sleep is basically indistinguishable from the pattern during wakefulness.
- Visual imagery during REM sleep, as compared to wakefulness, can occur through the inverse pathway of visual information processing.
- There are increases in oxygen and glucose utilization, brain temperature, local brain blood flow and neural activity during REM.
- REM dream pictures, similarly to visual imagery, can originate from long-term visual memories.
- REM dream pictures can be performed by non-linear iterative processes of top-down activated long-term visual epigenetic memory, much like visual imagery.
- An important implication is that there is no *homunculus* in our head but that the biophysical image representation is processed as signals, which are then sent to and interpreted by other parts of the brain.
- The same cortical areas are involved in eye movements in REM sleep and wakefulness, suggesting that REM saccades scan dream pictures and scenes.
- The generation of reactive oxygen and nitrogen species are precise mechanisms used in signaling pathways during physiological (pathological) processes in neurons and other cells.
- Bioluminescent photon emission originates from natural reactions of reactive oxygen and nitrogen species in various cells.
- Mitochondrial oxidative phosphorylation dominates in REM and waking, and the mean levels of mitochondrial cytochrome oxidase activity during REM sleep significantly exceed those during waking and slow-wave sleep.
- Mitochondrial oxidative phosphorylation is the main source of bioluminescent photons generated by regulated redox (free radical) reactions.
- Neuronal electrical signals can be converted into regulated bioluminescent photon (ultraweak optical signal) signals by radical and redox processes in neurons.
- There is neural activity-dependent ultraweak bioluminescent biophoton emission in the brain.
- Visual circuits that are normally involved in the detection of visual perception are also responsible for the generation of the phosphenes. Because retinal and cortical induced phosphenes

must have a common molecular biophysical basis, the experiments done by Narici et al. can serve as the first step toward our biophysical picture hypothesis.
- The majority of cells in the mitochondrial-rich areas are selective for color. Therefore, mitochondrial-rich visual areas can represent monocular sites of biophysical color processing in primate striate cortex.
- Intrinsic biophysical pictures (images) are not strict objects but can be altered *ad-lib*, which makes it possible that the visual system can produce irrationally assembled pictures and scenes during REM dreams.

These summarized facts and observations may support the hypothesis that neuro-visual activation redox patterns (*spike-related electrical signals along classical axonal-dendritic pathways*) can be converted into synchronized bioluminescent photonic signals (*inside the neurons*), which produce intrinsic biophysical pictures by regulated bioluminescent photons of mitochondrial networks in the retinotopically organized mitochondrial-rich neurons of V1 and V2 during REM dreams. We have to again emphasize that there is no *homunculus* in our head but rather a processed biophysical image representation, which is then sent to and processed by higher order areas of the brain.

We also proposed that visual hallucinations can be due to unregulated biophysical dream-like pictures by bioluminescent photons. Local paroxysms can produce dream-like pictures by bioluminescent photons in the retinotopically organized mitochondrial cytochrome oxidase-rich visual areas. Afterwards, these unregulated dream-like pictures formed by bioluminescent photons break into the waking consciousness.

It is remarkable that evolution of higher levels of complexity making intrinsic biophysical picture representation of the external visual world is possible. This process is performed by regulated redox and bioluminescent reactions as well as by the development of specific retinotopic visual system structures in the brain.

Conflict of interest

The authors report no conflicts of interest. The authors alone are responsible for the content.

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