Predictive Coding, Multisensory Integration, and Attentional Control: A Multicomponent Framework for Lucid Dreaming

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SUPPLEMENTARY INFORMATION

Table S1. Wake-like activity during sleep in different sleep disorders. Sleep disorders and pathological sleep conditions associated with wake-like cortical activity patterns during sleep. Wake-like activity during sleep appears to be a common denominator behind a wide variety of sleep disorders. The above table shows illustrative examples (for more extensive and complete reviews, especially for insomnia and NREM parasomnias, see (1–4)).

| Pathological sleep condition | Publications | Distinguishing EEG activity | Sleep stage featuring wake-like activity | Specific brain regions exhibiting wake-like activity |
|-----------------------------|--------------|-----------------------------|------------------------------------------|--------------------------------------------------|
| Sleep walking and sleep terrors | (5) | locally reduced (1-4 Hz) low frequency activity | SWS, REM, WAKE | cingulate, motor and sensorimotor cortices |
| | (6) | increased (24-30 Hz) beta power | SWS | cingulate cortex (pregenual and ventral anterior cingulate area) |
| Confusional arousals and sleep terrors | (7) | sustained local arousal (~ 25 Hz), beta activity during SWS sleep while low frequency activity in frontal associative cortices | SWS | motor, cingulate, insular, amygdalar, and temporopolar cortices |
| | (8) | lower delta and increased beta power resembling wakefulness | | ventromedial thalamus |
| Sleep paralysis | (10) | increased alpha activity during REM sleep (peak frequency of alpha activity overlapping with the alpha peak of wakefulness) | REM | Unknown |
| | (11) | increased alpha and reduced delta power | | |
Insomnia (12) reduced spectral slope indicating NREM Unknown reduced low and increased high frequency activity

(13) concomitant increase in sigma and beta power, indicating simultaneous activation of sleep-protective and arousing mechanisms, respectively

NREM

(14) arousals and sleep fragmentation REM during REM sleep

Nocturnal disorder (15, 15, 16) Increased arousals and high frequency (beta and gamma) activity NREM Unknown

(17) Increased high-alpha power REM

SWS – Slow Wave Sleep, REM – Rapid Eye Movement Sleep, NREM – Non-Rapid Eye Movement sleep

**Table S2. Further areas for lucid dream research: key assumptions and research agenda**

| Assumption | Observations and/or supportive evidence | Research agenda |
|------------|-----------------------------------------|-----------------|
| The processing of bodily signals (interoceptive processing) is enhanced in LD | Frequent otolithic experiences such as flying, floating, out of body experiences | Further studies on the individual differences in LD frequency in relation to individual differences in interoceptive processing |
| | Increased vestibular sensitivity in frequent lucid dreamers | To study interoceptive processing (e.g., heartbeat evoked potentials) in lucid versus non-lucid dreams |
| | Meditation increases awareness of bodily signals and is linked to the frequency of LD | To investigate if interoceptive stimulation (e.g., vestibular stimulation) may elicit LD |
| LD is characterized by increased precision weighting at lower levels of the processing stream | Lucid dreamers are able to detect and respond to inputs coming from the sensory periphery | To investigate the role of attentional control (e.g., shifting between exteroceptive versus interoceptive focus) in relation to LD frequency |
| | Lucid dreamers are able to perform intentional eye movements (motor commands) | To study cortical responses to sensory (e.g., acoustic) stimuli during lucid versus non-lucid dreaming |
| Fluctuations in sleep-and arousal regulation modulate the processing of sensory afferents facilitating sensitivity to prediction | Several LD induction techniques facilitate sleep state-changes and sleep fragmentation | Data driven EEG analyses of lucid versus non-lucid dream experiences |
Future directions

Lucid dreaming across the lifespan: Developmental aspects

The multicomponent framework of lucid dreaming may accommodate the somewhat surprising observations of increased lucid dream frequency in children (18) and its reduction in older age groups (19). Whereas the prefrontal and frontoparietal networks (still immature in young children) may be involved in lucid dreaming, our model suggests that increased sensitivity to bottom-up signals may also facilitate lucidity. Young children are more capable to automatically extract low-level, raw probabilities (i.e., statistical rules) of the environment (and hence to detect violations of such probabilities) indicating dominantly bottom-up, model-free processing of sensory experiences compared to older individuals (20, 21). On the other hand, cognitively controlled, model-based learning is gradually taking place after around 12 years of age (22). Interestingly, this shift in learning strategies corresponds to the gradual drop of lucid dream experiences from puberty to young adulthood (18). Susceptibility to low-level bottom-up inputs producing prediction error signals may play a major role in triggering lucid dream experiences in young children. Moreover, young children spend considerable time in REM sleep, and are prone to state dissociations, and parasomnias. Due to higher arousal thresholds young children are protected from abrupt awakenings (1), reducing the chance that active inference during dreaming will be associated with arousals and awakenings. In contrast, older individuals spend less time in REM sleep, and due to reduced sleep regulation are more susceptible to arousals (23, 24) and sleep fragmentation limiting the possibility to reach lucidity during sleep. Taken together, the probabilistic, model-free learning period of young children may provide a reflexive attitude towards their (true) mental state (of dreaming), where higher arousal threshold can stabilize the (lucid) dream experience. Coming to adulthood, the development of prefrontal and frontoparietal regions can provide top-down attentional control to stabilize the lucid dream state, but reducing its occurrence beforehand: In
adulthood, prediction errors may be often explained away by schemes acquired during model-based learning, and rarely have the chance to (truly) update the state of consciousness itself (from wakefulness, to dreaming). Pre-lucid dreams can provide examples for stereotypical actions and state interpretations (e.g., "I recognized that I am dreaming, so I went to tell it to others" or "I understood that I can fly because my body died and now, I live as a spirit"). However, it should be pointed out that studies on dreaming in young children are scarce and to some extent questionable on methodological grounds (25). In addition, the assessment of lucid dream frequency in different age groups is only based on retrospective self-reports that are prone to memory biases. Therefore, future studies applying prospective data collection procedures or signal-verified lucid dream assessments are warranted to systematically investigate the frequency and nature of lucid dream experiences in children.

**Lucid dreaming and psychopathology: relevance for psychosis**

Several authors argued that reduced executive and self-reflective functions (related to hypofrontality) in dreaming resemble psychosis (26–28); they suggested that lucid dreaming is the opposite of psychosis: a state of increased self-reflection and insight due to increased frontal activity (28, 29). Nevertheless, these assumptions are challenged by empirical data indicating weak but consistent associations between lucid dream frequency and proneness to psychosis (30, 31), and similar rates of lucid dream frequency in psychotic patients compared to non-psychotic controls (32). In the context of the present multicomponent framework, we may speculate that impaired sensory gating in psychotic states (33, 34) facilitates the processing of bottom-up sensory afferents during sleep. Accordingly, signs of reduced sensory gating in psychotic conditions were evidenced not only in wakefulness but also during sleep (35, 36). Moreover, whereas non-psychotic individuals can easily monitor and automatically distinguish internally generated experiences (e.g., imagination during mind wandering) from veridical perception, psychosis-prone individuals often have difficulties in discriminating externally and internally generated mental representations. The frequent experience of intrusive, vivid, perceptual experiences of ambiguous origin, and the effort to discriminate hallucinations from veridical perception may “train” psychosis-prone individuals to question the sources of dreaming. (For a detailed description of the utility of the PC framework in psychosis see 115, 116).
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