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Dream experiences and the neural correlates of perceptual consciousness and cognitive access

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This paper approaches the debate whether perceptual consciousness requires cognitive access from the perspective of dream studies, and investigates what kind of findings could support the opposing views of this debate. Two kinds of arguments are discussed, one that claims that the hypoactivity of the dorsolateral prefrontal cortex in rapid eye movement sleep is directly relevant, and another that proposes that locating the neural correlates of dream experiences can indirectly inform the debate. It is argued that under closer reflection, neither the classical claim about dorsolateral prefrontal cortex hypoactivity nor the more recent emphasis on general posterior hot zone activity during dreaming stand up to scrutiny. White dreaming is identified as the phenomenon that, nevertheless, holds the most promise to have an impact on the debate. Going beyond the topic if studying dreams can contribute to this debate, it is argued that cognitive access is not a monolithic phenomenon, and its neural correlates are not well understood. There seems to be a relevant form of cognitive access that can operate in the absence of activity in the dorsolateral prefrontal cortex, and maybe also in the whole frontal region. If so, then exclusive posterior activation during conscious experiences might very well be compatible with the hypothesis that perceptual consciousness requires cognitive access.

This article is part of the theme issue 'Perceptual consciousness and cognitive access'.

1. Introduction

The debate about the relationship between perceptual consciousness and cognitive access centres around whether perceptual consciousness requires cognitive access, i.e. whether a perceptual state can only be conscious if its content becomes directly available for reporting, manipulation, rational control of action, reasoning, etc. According to what might be called the 'access view' (or access hypothesis, see [1]), the answer is yes, perceptual consciousness does require cognitive access: a subject is conscious of the content of a representational state only if that content is globally available for deployment in a wide range of cognitive tasks. According to the opposing view, which might be called the 'no-access view', the answer is no, cognitive access is not necessary for perceptual consciousness: a piece of perceptual information can become conscious even if it is not globally broadcast, but only processed by specific local processes.

This paper approaches the debate between the access and no-access views from the perspective of studying dreams. Our aim is to investigate whether and how recent findings regarding dream experiences and the dreaming brain can inform us about the prospects of the access and the no-access views. Two sets of arguments will be discussed in detail: one that claims that certain characteristics of the dreaming brain—like the hypoactivity of the dorsolateral prefrontal cortex (dlPFC)—are directly relevant for the access versus no-access debate (§2), and another one that proposes that studying dream

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experiences is a natural no-report paradigm, and as such can indirectly inform the access versus no-access debate via locating the neural correlates of consciousness (NCC) more precisely than traditional report-based paradigms do (§3).

The main conclusion of the paper, however, goes beyond the particular topic of whether dream research can provide support for the access or the no-access view. In the course of reviewing dream-related arguments favouring these views, we will show that cognitive access is not a monolithic phenomenon, and its neural correlates are not well understood. This crucially affects what impact the direct and the indirect use of dream research might have on—and, more generally, how informative recent empirical approaches studying the NCC can be with regard to—the debate between the access and the no-access views (§4).

2. A direct role of studying dreams: lack of rationality and the argument from dorsolateral prefrontal cortex hypoactivity

Can studying the dreaming brain contribute to the debate between the access and the no-access views? Some people think that it can—that dreaming provides a straightforward case for the no-access view.

(a) Dreams and rationality

In a commentary to Ned Block's original paper introducing the phenomenal (P-) consciousness/access (A-) consciousness distinction [2], Antti Revonsuo offers dreaming as a 'pure case of P-consciousness without A-consciousness [that] has all the phenomenological properties without having any of the normal functional relationships to perceptual input or external behavior' [3, p. 266]. Revonsuo argues that even if the motor output blockade was removed and thus dream experiences could drive dream enactments, this would still not be a case of rational control of action, because people who have rapid eye movement (REM) sleep without atonia often injure themselves badly during attempted dream enactment.

Note, however, that in this respect, Revonsuo's argument misses the point: dreams stem from internal signals, which are quasi-independent from actual external environmental sources, so the question is not whether dream behaviour is an adequate reaction to the actual physical environment. A more promising strategy to draw conclusions with regard to dream rationality is investigating dream bizarreness. While some studies argue that discontinuities in dreams are quite common [4,5], others find it much less characteristic [6,7]. The portion of dreams with at least one bizarre content is high (between 63 and 91%); nonetheless, dreams are only rarely totally irrational or incongruent, and around 25% of dreams are absolutely realistic [8,9]. That is, although rationality is often present in dreams in an attenuated form, it is never totally absent (see also [10, p. 275]).

Be that as it may with regard to dream bizarreness, this argument for the lack of access from the lack of rationality draws attention to a reoccurring theme of this paper: that access is not a monolithic phenomenon (see §2d and §4). Cognitive access has many different manifestations, and rational control of action is only one of them—even if it is absent, other forms of cognitive access might very well still be in operation.

(b) Argument from dorsolateral prefrontal cortex hypoactivity

The idea that dreaming can provide a direct support for the no-access view has recently resurfaced in the literature in a more sophisticated form [11–14]. According to this line of thought, the fact that dream experiences occur in REM sleep clearly shows that phenomenal consciousness does not require cognitive access because the dlPFC, which is supposed to be responsible for key functions behind cognitive access, is highly inactive during this stage of sleep.

The argument runs as follows:

(i) During REM sleep, we have conscious experiences.
(ii) During REM sleep, dlPFC activity is reduced.
(iii) The dlPFC plays a crucial role in cognitive access.

Consequence: conscious experiences do not require cognitive access.

If the argument is valid and sound, then studying the dreaming brain can have a direct impact on the access versus no-access debate by providing unequivocal support for the no-access view. In what follows, we will be concentrating on the soundness of the argument, as we think that in light of recent discoveries, the truth of two of the premises can be called into doubt. Note, however, that the validity of the argument is not guaranteed either. It hinges on whether the reduction in dlPFC activity that premise (ii) focuses on affects cognitive access as discussed by premise (iii). If the residual activity of the dlPFC that premise (ii) is compatible with is sufficient for a minimal but relevant sense of cognitive access, then the consequence will not follow. That is, even if one finds the following sections unpersuasive and is still determined with regard to the soundness of the argument from dlPFC hypoactivty, one will still have more work to do as one will need to establish that the empirical evidence supporting premise (ii) also blocks the possibility of cognitive access compatible residual dlPFC activity. Especially so, as dreams are, after all, reportable: subjects after waking from dreamful sleep are able to report their dream experiences—which, in accordance with the standard definition, seems to render dream experiences cognitively accessed.

Although there are sceptical arguments with regard to premise (i) [15–20], there is a wide consensus in the literature that dreams are indeed conscious experiences occurring during sleeping [3,11,13,21–31]. In accordance with this general consensus, in what follows we are going to grant (i) and concentrate on the other two premises of the argument.

(c) Is dorsolateral prefrontal cortex activity reduced during rapid eye movement sleep?

Premise (ii) seems to be well supported by classical psychophysical findings regarding brain activity during REM sleep. According to the mainstream of dream research, dlPFC is highly deactivated during REM sleep, while other parts of the frontal lobe become even more activated than in waking [32–36] (for reviews, see [37–39]).

This pattern of activation and deactivation is hypothesized to be responsible for the characteristic features of dream experiences and dream cognition, like the fit between the dream-actor's behaviour and the places and objects occurring in dreams, the rare presence of a well-defined purpose to globally organize the actor's actions, the lack of planning, flexibility and logical reasoning, confusions with regard to space and time, and the rare occurrence of real full-fledged episodic memory items [40–42].

However, while there is a broad consensus about the intensified activation of the medial prefrontal cortex during REM sleep, not all findings are consistent with the standard view emphasizing the hypoactivity of the dlPFC region. For example, in a positron emission tomography (PET) study, Nofzinger *et al*. [43] famously found no significant change in glucose metabolism in the dlPFC (compared to the wakeful level).

In a more recent study, Kubota *et al*. [44], using near-infrared spectroscopy, found REM-locked activation of the dlPFC during REM sleep. The study, monitoring cortical oxygenation levels, concluded that the activity of dlPFC, which decreased after sleep onset, increased again to the level of wakeful activity during REM sleep after the onset of the first REM. Similar findings have been reported based on PET [45], fMRI [46] and MEG data [47].

A very recent study relying on intracranial electrode recordings from epilepsy patients [48] provided further support for the claim that dlPFC might actually be more active during REM sleep than it was previously thought. The study found that dlPFC was dominated by bursts of beta and theta activity during REM sleep, and probably interacted with other areas as indicated by a coherence between the beta and theta oscillations observed in the dlPFC and those observed in the anterior cingulate cortex.

The possible sources of the inconsistencies between classical and newer findings regarding dlPFC activity during REM sleep are not clear yet. Important factors might be differences in the temporal resolution of the different experimental techniques [48], in the studied subject-groups, in the studied REM stage (early- versus late-night REM [43]) or in the studied REM microstates (phasic versus tonic REM [49]). It is also important to note that during REM sleep, there are periods waking from which subjects report no dream experiences [50], so findings focusing on REM sleep itself might be contaminated by data from such dreamless episodes.

In any case, recent findings raise doubts with regard to dlPFC hypoactivity during REM dreams. Consequently, the soundness of the argument from dlPFC hypoactivity becomes controversial, as its eponymous premise (ii) turns out to be questionable: contrary to the standard characterization, dlPFC seems to be more active during REM sleep than traditionally thought.

(d) Does the dorsolateral prefrontal cortex play a crucial role in cognitive access?

As it happens, the prospects for the argument from dlPFC hyperactivity are poor even for those who are not persuaded by the findings that raise doubts with regard to premise (ii), as premise (iii) seems problematic as well.

Premise (iii) is originally motivated by the global neuronal workspace (GNW) model itself that advocates the access view by defining cognitive access in terms of a global activation of a broad fronto-parietal brain network (for alternative motivation, see [51]). Dehaene *et al*. [52–57] argue that dlPFC is a central hub of the neuronal network (the anatomical substrate of the global workspace) that needs to be ignited for conscious experiences to occur. As Dehaene *et al*. put it: 'the GNW involves primarily the dorsolateral prefrontal cortex, but also additional hubs in inferior parietal cortex, mid-temporal cortex, and precuneus' [56, p. 79].

The GNW theory associates consciousness with maintaining information online independently of stimulus presence, and thus connects it to working memory (WM), the dedicated memory system that temporarily stores information and interfaces between perception, long-term memory and action [58–62]. The neural basis of WM consists of a distributed network with a core fronto-parietal part that is typically activated during most WM tasks [63], and stimulus- and task-specific sensorimotor areas that are recruited on the basis of the actual stimulus-set and actual task demands (for reviews, see [64–67]; for recent criticisms of the recruitment model, see [68–70]; see also [71–73]). The dlPFC is part of the fronto-parietal core network [63], and has been argued to be necessary for intact WM functions [64,67,74–76]. This association of dlPFC with WM operations and its anatomical features (it contains a large number of pyramidal neurons establishing long-range cortico-cortical tangential connections) are the main reasons for Dehaene *et al*. to claim that dlPFC is a central hub of the GNW [52,53].

However, while early non-human primate experiments suggested otherwise [76,77], it is now well documented by recent studies investigating WM processes that dlPFC is more involved in representing task-related than stimulus-related information. Although sustained elevated activity can be recorded in the lateral prefrontal cortex, stimulus-specific information cannot be decoded by multi-variate pattern analysis techniques [78,79]. Instead, the lateral prefrontal cortex seems to represent abstract high-level information, like object categories [80,81], task- and trial-relevant information [78,82,83] and load-dependent effects [63]. According to these studies, dlPFC is crucial for being able to act on the stimulus in a way that conforms to task requirements, but not so much crucial for maintaining stimulus-related information (for reviews, see [65,84]; for opposing findings, see [69,85]; for replies, see [71,72]).

This is problematic from the perspective of the argument from dlPFC hypoactivity as maintaining stimulus-specific information such that they are available for reporting is the relevant sense of cognitive access that the access versus no-access debate relies on [2,86–89].

Still, as one might want to argue, even if dlPFC does not play an active role in maintaining stimulus-specific information, it does provide top-down signals targeting sensory areas that might be necessary for maintaining robust sensory representations in the absence of bottom-up sensory activity. This line of thought finds support in early results, according to which cooling parts of the prefrontal cortex reduce spiking activity in the inferior temporal cortex [75] (see also [64,67,86]).

However, in the light of recent findings, dlPFC does not seem to be necessary for maintaining stimulus-specific information and making it available for reporting in this sense either. For instance, in a human lesion study, Barbey *et al*. [90] found that dlPFC lesions did not produce deficits in tasks in which participants needed to repeat a sequence of digits or a sequence of taps of different locations on a board (for more details, see §4b). Similarly, in another human lesion study, Mackey *et al*. [91] reported that focal dlPFC lesions that spared the precentral sulcus had no effect on performance in a memory-guided saccade task where participants had to remember the location of a transient stimulus and indicate it with an eye movement after a delay period. (For reviews with the same conclusion, see also [92,93].)

That is, recent human lesion studies demonstrate that dlPFC lesions do not affect the maintenance of stimulus-specific information and its availability for reporting. Since lesion studies are especially apt for refuting claims about the necessity of a given brain region for performing specific functions, these results indicate that dlPFC activity, contrary to the claim of premise (iii), is not required for cognitive access. This breaks the alleged direct link between characteristics of the dreaming brain and the no-access view.

3. An indirect role of studying dreams: localizing the neural correlate of perceptual experience

Parallel with the debate between the access and no-access views, there is a related debate regarding whether the NCC extend to the front of the brain or are confined to the back. Access-related (e.g. GNW) theories attribute critical role to frontal regions [52–56,57,94], while no-access accounts typically advocate posteriorly localized NCC [86,95–98]. In this context, it has recently been claimed that studying dreams could indirectly inform the access versus no-access debate via locating the NCC more precisely than traditional experimental paradigms do.

(a) No-report paradigms

In recent years, there has been a proliferation of research and review papers arguing that previously reported frontal NCC components are, in fact, not required for conscious experiences—they are rather only confounds that occur due to some cognitive factors evoked by the specific task requirements of classical report-based paradigms where conscious experiences are identified on the basis of subjective reports (for reviews, see [31,38,99–103]; for criticism, see [85]).

The problem with classical report-based paradigms is that they typically contrast two conditions that share the same sensory input but differ in conscious experiences and result in *distinct reports*. On the one hand, distinct reports inform the experimenters that the subjects had distinct conscious experiences; but on the other hand, the difference between the neural activities occurring during the two conditions partly corresponds to the difference in the reports, thus confounding the NCC [100,104].

The ability to report is based on factors like vigilance, attention, introspection and motor response [38,100,105]. No-report paradigms try to control for report-based confounds by creating conditions that can ensure that none of these factors are in operation. For example, Pitts *et al.* [106–108] used an inattentional blindness paradigm to divert attention from test stimuli, and relied on late reports to control for vigilance, introspection and motor response during stimulus presentation.

However, becoming alert, introspecting and attributing attentional resources non-focally are not easy to control and especially not easy to attenuate in the necessarily task-oriented settings of experimental sessions [105]. Wakeful subjects are always conscious of something: even in ‘unconscious’ trials, when a particular stimulus is rendered unconscious, subjects are conscious of many aspects of the actual experimental setting, and also of the task requirements, which generate expectations, keep subjects alert and trigger introspection and metacognition.

(b) Dreaming as a natural no-report paradigm

Studying dreams offers a unique possibility to reduce the confounding effects of such cognitive factors [31,38,101]. Dreaming subjects are not concerned about reporting their experiences, neither are they engaged with a specific task. Comparing dreaming with dreamless sleep, thus, is not just a no-report, but also a no-task paradigm. (Importantly, it is also generally considered to be a within-state paradigm that is free of the confounds generated by the radical shifts in the level of consciousness characteristic of studies comparing waking consciousness with unconsciousness [109,110].) Vigilance, introspection, attention and metacognition are naturally attenuated in dreaming [34–36,42]. From this perspective, studying the neural correlates of dream experiences can provide particularly relevant information about the NCC, and thus might indirectly contribute to the access versus no-access debate.

In a groundbreaking study, Siclari *et al.* [31], using high-density EEG recordings in a serial awakening paradigm where subjects were awakened in 15–30 min intervals [50], contrasted the presence and absence of dream experiences both in REM and NREM sleep. Comparing both high- (20–50 Hz) and low-frequency (1–4 Hz) components of the EEG signal, Siclari *et al.* found that the presence or absence of consciousness correlated with local changes in high- and low-frequency activity, irrespective of global EEG characteristics. According to Siclari *et al.*, the occurrence of dream experiences correlates with a decrease in the low-frequency EEG component localized to a so-called ‘posterior hot zone’ (low- and high-level sensory areas, precuneus, posterior cingulate, retrosplenial cortex). Consequently, Siclari *et al.* argue that, in line with the conclusions of recent no-report paradigms [38,100–102,106–108,111], their findings support the view that previously highlighted frontal NCC components were, in fact, confounds, and the true neural correlate of consciousness can be localized to posterior areas.

Note that this claim is in tension with the findings regarding dlPFC activity in REM sleep discussed above [44–48], as those findings imply some frontal activity. Indeed, despite the authors’ emphasis on the posterior hot zone [31,38,101], even Siclari *et al.* reported increased frontal activity: in cases of successful dream recalls versus having no dream experiences the high-frequency EEG component increased in areas extending outside the posterior hot zone to parts of lateral frontal cortex (including dlPFC, especially in REM) and temporal lobes (see fig. 3c in [31]). Others also reported that successful dream recall positively correlated with increased frontal activity in the theta band [112–115] that has been associated with episodic memory encoding and WM maintenance [116–119]. Moreover, according to a recent study, during dream experiences, even decreased low-frequency activity extends beyond the posterior hot zone: it has been found that successful dream recall correlated with reduced low-frequency activity not just over the posterior hot zone but also over frontal and temporal areas as well [120].

In the light of these findings implicating frontal activity during dreaming, the general conclusion that ties conscious experiences to posterior hot zone activity seems less well supported (see also [85]). Consequently, the findings uncovered by studies comparing successful dream recall with no dream experiences have no clear implications with regard to the debate between the access and no-access views on consciousness.

(c) White dreams and the perfect experiment

Before concluding that the evidence emerging from studying dream experiences is, in fact, uninformative with respect to the access versus no-access debate, one more phenomenon needs to be considered: *white dreams*. White dreams, also called contentless dreams, are reported dream experiences without the ability to recall any specific content: after awakening, subjects are certain that they were dreaming but are unable to report any details about these dreams. In serial awakening paradigms, approximately 30% of recorded answers report white dreams [31,50,121–123].

Siclari *et al*., beyond contrasting successful dream recall with having no dream experiences, also investigated white dreams and compared their neural correlates with the neural correlates of dream experiences with successful recall of details and with the neural correlates of having no dream experiences. The occurrence of white dreams, just like proper dream experiences with reportable content, correlated with decreased low-frequency EEG activity in the posterior hot zone. However, in the high-frequency range, Siclari *et al*. [31] found a significant difference between white dreams and dream experiences with reportable content: although they both correlated with increased activity in the posterior hot zone, in the case of white dreams, this activity did not extend to frontal and temporal areas.

White dreams, thus, present a case in which the neural correlate of a conscious experience is indeed localized to the back of the brain. Moreover, white dreams are also interesting from the perspective of the access versus no-access debate. Given the interpretation that during white dreams, subjects have dream experiences the details of which they then are unable to report, white dreams seem to be similar to classical cases motivating the no-access view [86,124,125]. Subjects in the Sperling paradigm, for example, insist that they have experienced the whole array of letters, yet they are unable to report details about most of the items [124].

We are not claiming here that white dreams provide a clear case for perceptual consciousness without cognitive access. We are only suggesting that if studying dreams can have any impact on the access versus no-access debate at all, then white dreams might be the most promising targets for further investigations. White dreaming is an immensely under-explored phenomenon open to many different interpretations. Typically, white dreams are interpreted as forgotten dreams [31,126] and are sidelined as not being particularly informative with regard to the nature of dreaming. Existing findings are also ambiguous. Contrasting white dreams with dream experiences with reportable content showed no difference in posterior hot zone activity in the high-frequency range, suggesting that these regions are just as active during white dreaming as during those forms of dreaming waking from which subjects are able to recall specific details (see Fig. 3b in [31]). However, when contrasted with having no dream experiences, white dreams, again, showed no difference in high-frequency posterior hot zone activity, which seems to indicate inactivity in the posterior hot zone (see electronic supplementary fig. S4*a* in [31]). (During dream experiences with reportable content, there is increased high-frequency posterior hot zone activity compared to cases in which subjects report having no dream experiences, see fig. 3a in [31]).

Nevertheless, granting that content-specific posterior regions are active during white dreaming (even if only to a lesser degree than in the case of dream experiences with reportable content, as this seems to be the most plausible interpretation of the ambiguous data presented above), and given that frontal areas associated with cognitive access are inactive, white dreaming might be the closest real-world counterpart of the hypothetical 'perfect experiment' (aiming to detach the regions responsible for phenomenal consciousness from other regions that underly cognitive access) that is often discussed in the literature on the debate between the access and no-access views [96,127–129].

4. The neural correlate of cognitive access

The access view maintains that for consciousness, direct availability for reporting, manipulation, rational control of action, reasoning, etc. is necessary. This is a monolithic conception of cognitive access that does not differentiate between these different functions. Rather this monolithic conception of cognitive access is tied to different neural signatures like dlPFC activity, the occurrence of the P3b component of the P300 event-related potential, or global ignition in a widespread fronto-parietal network [38,52–56,57,94], without investigating which phenomenon is the neural signature of which particular function.

Recent objections against the access view target these specific neural signatures, and try to show that they do not correlate with conscious experiences. For example, Frässle *et al*. [111] using a no-report condition of binocular rivalry found that most of the right dlPFC activation disappeared when subjects passively experienced rivalry without reports. They conclude that right frontal activity is not part of the NCC, but only required for report. In another set of studies, Pitts *et al*. [107,108] using an inattentional blindness paradigm and collecting late reports found that when subjects' attention was diverted and immediate reports were not required the P3 signal diminished. The study by Siclari *et al*. [31], arguing that dream experiences correlate with posterior hot zone activity fits into this trend. The emphasis on posterior hot zone activity [38,101,102] is meant to highlight that consciousness does not require frontal activity.

However, recent enquiries into the neural underpinnings of WM operations revealed that different WM functions depended on different neural substrates. These findings are highly relevant for the access versus no-access debate.

(a) Different working memory functions and the relevant sense of cognitive access

In a typical WM task, subjects are asked to retain certain information about a briefly presented stimulus throughout a delay period, and then produce a specific behavioural output that is partly determined by the information maintained, and partly by task requirements. From this, it is already evident that WM needs to temporarily store both stimulus-specific and task-specific information to be able to properly interface between perception (and/or long-term memory) and action.

Task requirements can vary widely, from simple maintenance, through active monitoring, to full-fledged manipulation [90]. *Maintenance* is the online retention of information in WM. In a maintenance task, for instance, subjects are asked to repeat a sequence of visually or aurally presented digits, or a sequence of locations tapped on a board [130]. *Monitoring* is the process of deliberately attending to information in WM. In a monitoring task, for example, a sequence of letters is visually or aurally presented, and subjects need to indicate whether the letter presented last (or one, two, etc. items before that) matches a target stimulus [131]. *Manipulating* is the rearrangement and transformation of the information maintained for goal-directed behaviour. In a manipulation task, for instance, subjects see or hear a sequence of alternating digits and letters, and then are asked to rearrange the digits in numerical order, and the letters in alphabetical order [130,132].

As it has already been emphasized (§2d), from the perspective of the access versus no-access debate, the relevant sense of cognitive access is the direct availability of stimulus-specific information, as the question is whether consciously experiencing a stimulus requires cognitive access to information about the features of the stimulus. We have also seen, that simple maintenance tasks already measure cognitive access in a relevant sense, as the information maintained becomes available for subsequent report [2,86–89].

(b) The dorsolateral prefrontal cortex and cognitive access

Given all this, the conclusion of recent human lesion studies [90–93] that dlPFC is not necessary for the maintenance of stimulus-specific information in WM (which is also supported by transcranial magnetic stimulation [133] and transcranial direct current stimulation studies [134]; see also [135]) means that in a relevant minimal (i.e. simple maintenance) sense, dlPFC activity is not required for cognitive access (see §2d).

For example, Barbey *et al.* [90] investigated the performance of subjects with dlPFC lesions, non-dorsolateral PFC lesions and no brain lesions in standard neuropsychological tests that selectively measured different aspects of WM functions. The tests administered differentiated between maintenance, monitoring and manipulating. Barbey *et al.* found that intact dlPFC was crucial only for the manipulation of verbal and spatial information in WM (left dlPFC), and for the manipulation of information in tests of arithmetic and spatial reasoning (right dlPFC). Lesions of the dlPFC had no effect on performance in maintenance and monitoring tasks: patients with dlPFC lesions could repeat a sequence of digits in order, could repeat a sequence of tapping different locations on a board in order and could indicate whether the most recent item of a visually presented sequence of letters matched a target stimulus [90].

In the light of these findings, the fact that dlPFC activation disappeared in Frässle *et al.*'s passive viewing condition [111] does not necessarily mean that the content of the subjects' experiences were not cognitively accessed, as relevant information maintenance—and in the light of the Barbey *et al.* study [90], even monitoring—could have occurred without dlPFC activity.

(c) Diminished P3b signal and cognitive access

Although in the access versus no-access literature, the P3b signal is linked to fronto-parietal activity [55], its neural generators are, in fact, debated. Some localize the source of the P3b signal to parietal–temporal regions [136], others suggest that it is generated by activity in the superior parietal lobule and the posterior part of the cingulate gyrus [137]. Wronka *et al.* [137] argue that the P3b signal is related to activation of a posterior network reflecting a comparison of the neuronal model of perceived stimulation with the attentional trace. Given this interpretation, Pitts *et al.*'s findings [107,108] imply that diverting attention disrupts such monitoring functions. Nevertheless, these findings might still be compatible with cognitive access via simple information maintenance not requiring monitoring or manipulation.

Just as dlPFC activity might reflect only one type or one aspect of cognitive access, the P3b signal might reflect only another aspect. Their disappearance, thus, does not entail the lack of cognitive access altogether as further aspects of cognitive access might still be in operation.

(d) Cognitive access and frontal activity

Similar concerns can be raised against attempts that try to use studying dreams—along with other no-report paradigms—to indirectly address the access versus no-access debate by arguing that frontal regions are not required for conscious experiences (see §3). The transfer of this conclusion to the access versus no-access debate is not straightforward. There is no immediate link between the frontal-posterior and the access versus no-access debates, because, as we have seen, inactivity of certain frontal regions does not necessarily speak in favour of the no-access position, because the frontal regions in question might not be necessary for access itself.

However, as one might try to argue, even if cognitive access is possible without dlPFC activity, it might not be possible without at least some kind of frontal activity. (For instance, Mackey *et al.* [91] argue that while intact human dlPFC is not, intact precentral sulcus is necessary for spatial WM. See also §2d.) From this perspective, studying white dreaming (see §3c) might be an interesting option for proponents of the no-access view, because during white dreams, there seem to be no local activations in frontal brain regions whatsoever. Nevertheless, even studying white dreams could turn out to be uninformative with regard to the question whether perceptual consciousness requires cognitive access, as according to some recent findings, cognitive access is possible even without any kind of frontal activity. In a human lesion study, Ivanova *et al.* [93] found that frontal areas are not necessary for simple WM tasks: only lesions in the superior and middle temporal gyri affected performance in a verbal two-back task (requiring information maintenance and monitoring) where participants were instructed to judge if a word in a continuous string of aurally presented words was the same as the one preceding it two items back. As the superior and middle temporal gyri are parts of the posterior hot zone [101], this result suggests that even findings that place the seat of consciousness in the posterior hot zone—and thus raise doubts with regard to the particular way the GNW theory implements the access view—can provide no unequivocal support for the no-access view.

5. Conclusion

The debate about the relationship between perceptual consciousness and cognitive access is motivated by a disagreement regarding whether a perceptual state can only be conscious if its content becomes cognitively accessed, i.e. directly available for reporting, manipulation, rational control of action, reasoning, etc. In this debate, existing literature relies on a monolithic conception of cognitive access that is anchored to neural underpinnings such as activity in the dorsolateral prefrontal cortex, or, more broadly, in a widespread fronto-parietal network. Studying the dreaming brain has been proposed as a possible source of information that could advance this debate by demonstrating that having dream experiences does not correlate with increased dlPFC and frontal activity. In this paper, we have argued that under closer reflection, neither the classical claim of dlPFC hypoactivity nor the more recent emphasis on general posterior hot zone activity during dreaming stands up to scrutiny. We have identified white dreaming as the phenomenon that, nevertheless, holds the most promise to have an impact on the debate on the relationship between consciousness and cognitive access. Going beyond the topic whether studying dreams can contribute to this debate, we have argued that recent findings raised doubts with regard to whether cognitive access requires dlPFC activity, or even any kind of frontal activity, as there seems to be a relevant form of cognitive access that can operate in the absence of dlPFC, and maybe also total frontal, activation. If so, then exclusive posterior activity during conscious experiences might very well be compatible with the hypothesis that perceptual consciousness requires cognitive access.

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References

1. Phillips I. 2018 The methodological puzzle of phenomenal consciousness. *Phil. Trans. R. Soc. B* **373**, 20170347. (doi:10.1098/rstb.2017.0347)
2. Block N. 1995 On a confusion about a function of consciousness. *Behav. Brain Sci.* **18**, 227–247. (doi:10.1017/S0140525X00038188)
3. Revonsuo A. 1995 Conscious and nonconscious control of action. *Behav. Brain Sci.* **18**, 265–266. (doi:10.1017/S0140525X00038395)
4. McCarley RW, Hoffman E. 1981 REM sleep dreams and the activation-synthesis hypothesis. *Am. J. Psychiatry* **138**, 904–912. (doi:10.1176/ajp.138.7.904)
5. Rittenhouse CD, Stickgold R, Hobson JA. 1994 Constraint on the transformation of characters, objects, and settings in dream reports. *Conscious Cogn.* **3**, 100–113. (DOI:10.1006/ccog.1994.1007)
6. Williams J, Merritt J, Rittenhouse C, Hobson JA. 1992 Bizarreness in dreams and fantasies: implications for the activation-synthesis hypothesis. *Conscious Cogn.* **1**, 172–185. (doi:10.1016/1053-8100(92)90059-J)
7. Revonsuo A, Salmivalli C. 1995 A content analysis of bizarre elements in dreams. *Dreaming* **5**, 169–187. (doi:10.1037/h0094433)
8. Colace C. 2003 Dream bizarreness reconsidered. *Sleep Hypn.* **5**, 105–128.
9. Domhoff GW. 2007 Realistic simulation and bizarreness in dream content: past findings and suggestions for future research. In *The new science of dreaming: content, recall and personality characteristics*, (eds D Barrett, P McNamara), pp. 1–27. Westport, CT: Praeger Press.
10. Block N. 1995 How many concepts of consciousness? *Behav. Brain Sci.* **18**, 272–284. (doi:10.1017/S0140525X00038486)
11. Rosen M. 2013 What I make up when I wake up: anti-experience views and narrative fabrication of dreams. *Front. Psychol.* **4**. (doi:10.3389/fpsyg.2013.00514)
12. Sebastián MÁ. 2014 Dreams: an empirical way to settle the discussion between cognitive and non-cognitive theories of consciousness. *Synthese* **191**, 263–285. (doi:10.1007/s11229-013-0385-y)
13. Crespin L. 2015 'Are dreams experiences?': insights from dreaming considered as a conscious experience under constraint of delayed report. *Dreaming* **25**, 118–140. (doi:10.1037/a0039157)
14. Pantani M, Tagini A, Raffone A. 2018 Phenomenal consciousness, access consciousness and self across waking and dreaming: bridging phenomenology and neuroscience. *Phenomenol. Cogn. Sci.* **17**, 175–197. (doi:10.1007/s11097-016-9491-x)
15. Malcolm N. 1959 *Dreaming*. New York, NY: Humanities.
16. Dennett D. 1976 Are dreams experiences? *Philos. Rev.* **85**, 151–171. (doi:10.2307/2183728)
17. Dennett D. 1991 *Consciousness explained*. 1st edn. Boston, MA: Little, Brown and Co.
18. Rosenthal D. 2002 How many kinds of consciousness? *Conscious Cogn.* **11**, 653–665. (doi:10.1016/S1053-8100(02)00017-X)
19. Dennett D. 2005 *Sweet dreams: philosophical obstacles to a science of consciousness*. Cambridge, MA: MIT Press.
20. Dennett,D. 2014 What is dreaming for, if anything? In *Dream consciousness: Alan Hobson's new approach to the brain and its mind* (ed. N Tranquillo), pp. 113–117. Berlin, Germany: Springer.
21. Flanagan O. 2000 *Dreaming souls*. New York, NY: Oxford University Press.
22. Metzinger T. 2003 *Being no one: the self-model theory of subjectivity*. Boston, MA: MIT Press.
23. Sosa E. 2005 Dreams and philosophy. *Proc. Addresses Am. Philos. Assoc.* **79**, 7–18.
24. Revonsuo A. 2006 *Inner presence: consciousness as a biological phenomenon*. Boston, MA: MIT Press.
25. Ichikawa J. 2009 Dreaming and imagination. *Mind Language* **24**, 103–121. (doi:10.1111/j.1468-0017.2008.01355.x)
26. Ichikawa J, Sosa D. 2009 Dreaming, philosophical issues. In *Oxford companion to consciousness* (eds T Bayne, P Wilken). New York, NY: Oxford University Press.
27. Metzinger T. 2009 Why are out-of-body experiences interesting for philosophers? *Cortex* **45**, 256–258. (doi:10.1016/j.cortex.2008.09.004)
28. Windt JM. 2010 The immersive spatiotemporal hallucination model of dreaming. *Phenomenol. Cogn. Sci.* **9**, 295–316. (doi:10.1007/s11097-010-9163-1)
29. Windt JM, Noreika V. 2011 How to integrate dreaming into a general theory of consciousness—a critical review of existing positions and suggestions for future research. *Conscious Cogn.* **20**, 1091–1107. (doi:10.1016/j.concog.2010.09.010)
30. Windt JM. 2013 Reporting dream experience: why (not) to be skeptical about dream reports. *Front. Human Neurosci.* **7**. (doi:10.3389/fnhum.2013.00708)
31. Siclari F, Baird B, Perogamvros L, Bernardi G, LaRocque JJ, Riedner B, Boly M, Postle BR, Tononi G. 2017 The neural correlates of dreaming. *Nat. Neurosci.* **24**, 171–878. (doi:10.1038/nn.4545)
32. Maquet P, Péters J-M, Aerts J, Delfiore G, Degueldre C, Luxen A, Franck G. 1996 Functional neuroanatomy of human rapid-eye-movement sleep

and dreaming. *Nature* **383**, 163–166 (doi:10.1038/383163a0)

33. Braun AR, Balkin TJ, Wesenten NJ, Carson RE, Varga M, Baldwin P, Selbie S, Belenky G, Herscovitch P. 1997 Regional cerebral blood flow throughout the sleep-wake cycle. An H2(15)O PET study. *Brain* **120**, 1173–1197. (doi:10.1093/brain/120.7.1173)
34. Hobson JA, Stickgold R, Pace-Schott EF. 1998 The neuropsychology of REM sleep dreaming. *Neuroreport* **9**, R1–R14. (doi:10.1097/00001756-199802160-00033)
35. Muzur A, Pace-Schott EF, Hobson JA. 2002 The prefrontal cortex in sleep. *Trends Cogn. Sci.* **6**, 475–481. (doi:10.1016/S1364-6613(02)01992-7)
36. Fox KCR, Nijeboer S, Solomonova E, Domhoff GW, Christoff K. 2013 Dreaming as mind wandering: evidence from functional neuroimaging and first-person content reports. *Front. Human Neurosci.* **7**. (doi:10.3389/fnhum.2013.00412)
37. Nir Y, Tononi G. 2010 Dreaming and the brain: from phenomenology to neurophysiology. *Trends Cogn. Sci.* **14**, 88–100. (doi:10.1016/j.tics.2009.12.001)
38. Koch C, Massimini M, Boly M, Tononi G. 2016 Neural correlates of consciousness: progress and problems. *Nat. Rev. Neurosci.* **17**, 307. (doi:10.1038/nrn.2016.22)
39. Siclari F, Tononi G. 2016 Sleep and dreaming. In *The neurology of conciousness* (eds S Laureys, O Gosseries, G Tononi), pp. 107–128, 2nd edn. San Diego, MA, Academic Press.
40. Schwartz S, Maquet P. 2002 Sleep imaging and the neuro-psychological assessment of dreams. *Trends Cogn. Sci.* **6**, 23–30. (doi:10.1016/S1364-6613(00)01818-0)
41. Fosse MJ, Fosse R, Hobson JA, Stickgold RJ. 2003 Dreaming and episodic memory: a functional dissociation? *J. Cogn. Neurosci.* **15**, 1–9. (doi:10.1162/089892903321107774)
42. Maquet P *et al*. 2005 Human cognition during REM sleep and the activity profile within frontal and parietal cortices: a reappraisal of functional neuroimaging data. In *Progress in brain research* (ed. S. Laureys), pp. 219–595. Amsterdam, The Netherlands: Elsevier.
43. Nofzinger EA, Mintun MA, Wiseman M, Kupfer DJ, Moore, RY. 1997 Forebrain activation in REM sleep: an FDG PET study. *Brain Res.* **770**, 192–201. (doi:10.1016/S0006-8993(97)00807-X)
44. Kubota Y, Takasu NN, Horita S, Kondo M, Shimizu M, Okada T, Wakamura T, Toichi M. 2011 Dorsolateral prefrontal cortical oxygenation during REM sleep in humans. *Brain Res.* **1389**, 83–92. (doi:10.1016/j.brainres.2011.02.061)
45. Hong C, Christian Gillin JM, Dow B, Wu J, Buchsbaum M. 1995 Localized and lateralized cerebral glucose metabolism associated with eye movements during REM sleep and wakefulness: a positron emission tomography (PET) study. *Sleep* **18**, 570–580.
46. Hong CC-H *et al*. 2009 fMRI evidence for multisensory recruitment associated with rapid eye movements during sleep. *Hum. Brain Mapp.* **30**, 1705–1722. (doi:10.1002/hbm.20635)
47. Ioannides AA, Kostopoulos GK, Liu L, Fenwick PBC. 2009 MEG identifies dorsal medial brain activations during sleep. *Neuroimage* **44**, 455–468. (doi:10.1016/j.neuroimage.2008.09.030)
48. Vijayan S, Lepage KQ, Kopell NJ, Cash SS. 2017 Frontal beta-theta network during REM sleep. *eLife* **6**, e18894. (doi:10.7554/eLife.18894)
49. Simor P, Gombos F, Blaskovich B, Bódizs R. 2018 Long-range alpha and beta and short-range gamma EEG synchronization distinguishes phasic and tonic REM periods. *Sleep* **41**, zsx210–zsx210. (doi:10.1093/sleep/zsx210)
50. Siclari F, LaRocque J, Postle B, Tononi G. 2013 Assessing sleep consciousness within subjects using a serial awakening paradigm. *Front. Psychol.* **4**. (doi:10.3389/fpsyg.2013.00542)
51. Lau H, Passingham RE. 2006 Relative blindsight in normal observers and the neural correlate of visual consciousness. *Proc. Natl Acad. Sci. USA* **103**, 18 768–18 763. (doi:10.1073/pnas.0607716103)
52. Dehaene S, Kerszberg M, Changeux J-P. 1998 A neuronal model of a global workspace in effortful cognitive tasks. *Proc. Natl Acad. Sci. USA* **95**, 14529. (doi:10.1073/pnas.95.24.14529)
53. Dehaene S, Naccache L. 2001 Towards a cognitive neuroscience of consciousness: basic evidence and a workspace framework. *Cognition* **79**, 1–37. (doi:10.1016/S0010-0277(00)00123-2)
54. Dehaene S, Changeux J. 2004 Neural mechanisms for access to consciousness. In *The cognitive neurosciences* (ed. MS Gazzaniga), pp. 1145–1157. Cambridge, MA: MIT Press.
55. Dehaene S, Changeux J-P. 2011 Experimental and theoretical approaches to conscious processing. *Neuron* **70**, 200–227. (doi:10.1016/j.neuron.2011.03.018)
56. Dehaene S, Charles L, King J-R, Marti S. 2014 Toward a computational theory of conscious processing. *Curr. Opin Neurobiol.* **25**, 76–84. (doi:10.1016/j.conb.2013.12.005)
57. Dehaene S, Changeux J, Naccache L, Sackur J, Sergent C. 2006 Conscious, preconscious, and subliminal processing: a testable taxonomy. *Trends Cogn. Sci.* **10**, 204–211. (doi:10.1016/j.tics.2006.03.007)
58. Baddeley A. 1986 *Working memory*. Oxford, UK: Oxford University Press.
59. Cowan N. 1995 *Attention and memory: an integrated framework*. New York, NY: Oxford University Press.
60. Oberauer K. 2002 Access to information in working memory: exploring the focus of attention. *J. Exp. Psychol. Learn. Mem. Cogn.* **28**, 411–421. (doi:10.1037/0278-7393.28.3.411)
61. Baddeley A. 2003 Working memory: looking back and looking forward. *Nat. Rev. Neurosci.* **4**, 829–839. (doi:10.1038/nrn1201)
62. Oberauer K. 2013 The focus of attention in working memory—from metaphors to mechanisms. *Front. Human Neurosci.* **7**, 673. (doi:10.3389/fnhum.2013.00673)
63. Rottschy C, Langner R, Dogan I, Reetz K, Laird AR, Schulz JB, Fox PT, Eickhoff SB. 2012 Modelling neural correlates of working memory: a coordinate-based meta-analysis. *Neuroimage* **60**, 830–846. (doi:10.1016/j.neuroimage.2011.11.050)
64. D'Esposito M. 2007 From cognitive to neural models of working memory. *Phil. Trans. R. Soc. Lond. B* **362**, 761. (doi:10.1098/rstb.2007.2086)
65. D'Esposito M, Postle BR. 2015 The cognitive neuroscience of working memory. *Annu. Rev. Psychol.* **66**, 115–142. (doi:10.1146/annurev-psych-010814-015031)
66. Eriksson J, Vogel EK, Lansner A, Bergström F, Nyberg L. 2015 Neurocognitive architecture of working memory. *Neuron* **88**, 33–46. (doi:10.1016/j.neuron.2015.09.020)
67. Fuster J. 2015 *The prefrontal cortex*. New York, NY: Academic Press.
68. Leavitt ML, Mendoza-Halliday D, Martinez-Trujillo JC. 2017 Sustained activity encoding working memories: not fully distributed. *Trends Neurosci.* **40**, 328–346. (doi:10.1016/j.tins.2017.04.004)
69. Xu Y. 2017 Reevaluating the sensory account of visual working memory storage. *Trends Cogn. Sci.* **21**, 794–815. (doi:10.1016/j.tics.2017.06.013)
70. Harrison WJ, Bays PM. 2018 Visual working memory is independent of the cortical spacing between memoranda. *J. Neurosci.* **38**, 1–36. (doi:10.1523/JNEUROSCI.2645-17.2017)
71. Gayet S, Paffen C, Van der Stigchel S. 2018 Visual working memory storage recruits sensory processing areas. *Trends Cogn. Sci.* **22**, 189–190. (doi:10.1016/j.tics.2017.09.011)
72. Scimeca JM, Kiyonaga A, D'Esposito M. 2018 Reaffirming the sensory recruitment account of working memory. *Trends Cogn. Sci.* **22**, 190–192. (doi:10.1016/j.tics.2017.12.007)
73. Xu Y. 2018 Sensory cortex is nonessential in working memory storage. *Trends Cogn. Sci.* **22**, 192–193. (doi:10.1016/j.tics.2017.12.008)
74. Bauer RH, Fuster J. 1976 Delayed-matching and delayed-response deficit from cooling dorsolateral prefrontal cortex in monkeys. *Q. J. Exp. Psychol. B* **90**, 293–302. (doi:10.1037/h0087996)
75. Fuster J, Bauer RH, Jervey JP. 1985 Functional interactions between inferotemporal and prefrontal cortex in a cognitive task. *Brain Res.* **330**, 299–307. (doi:10.1016/ 0006-8993(85)90689-4)
76. Funahashi S, Bruce CJ, Goldman-Rakic PS. 1993 Dorsolateral prefrontal lesions and oculomotor delayed-response performance: evidence for mnemonic 'scotomas'. *J. Neurosci.* **13**, 1479–1497. (doi:10.1523/JNEUROSCI.13-04-01479.1993)
77. Funahashi S, Bruce CJ, Goldman-Rakic PS. 1989 Mnemonic coding of visual space in the monkey's dorsolateral prefrontal cortex. *J. Neurophysiol.* **61**, 331–349. (doi:10.1152/jn.1989.61.2.331)
78. Riggall AC, Postle B. 2012 The relationship between working memory storage and elevated activity as measured with functional magnetic resonance imaging. *J. Neurosci.* **32**, 12 990–12 998. (doi:10.1523/JNEUROSCI.1892-12.2012)
79. Emrich SM, Riggall AC, LaRocque J, Postle B. 2013 Distributed patterns of activity in sensory cortex reflect the precision of multiple items maintained in

visual short-term memory. *J. Neurosci.* **33**, 6516–6523. (doi:10.1523/JNEUROSCI.5732-12.2013)

80. Meyers EM, Freedman DJ, Kreiman G, Miller E, Poggio T. 2008 Dynamic population coding of category information in inferior temporal and prefrontal cortex. *J. Neurosci.* **100**, 1407–1419.
81. Lee SH, Kravitz DJ, Baker CI. 2013 Goal-dependent dissociation of visual and prefrontal cortices during working memory. *Nat. Neurosci.* **16**, 997–999. (doi:10.1038/nn.3452)
82. Sreenivasan KK, Curtis CE, D'Esposito M. 2014 Revisiting the role of persistent neural activity during working memory. *Trends Cogn. Sci.* **18**, 82–89. (doi:10.1016/j.tics.2013.12.001)
83. Sreenivasan KK, Vytlacil J, D'Esposito M. 2014 Distributed and dynamic storage of working memory stimulus information in extrastriate cortex. *J. Cogn. Neurosci.* **26**, 1141–1153. (doi:10.1162/jocn_a_00556)
84. Postle B. 2015 The cognitive neuroscience of visual short-term memory. *Curr. Opin. Behav. Sci.* **1**, 40–46. (doi:10.1016/j.cobeha.2014.08.004)
85. Odegaard B, Knight R, Lau H. 2017 Should a few null findings falsify prefrontal theories of conscious perception? *J. Neurosci.* **37**, 9593–9602. (doi:10.1523/JNEUROSCI.3217-16.2017)
86. Block N. 2007 Consciousness, accessibility, and the mesh between psychology and neuroscience. *Behav. Brain Sci.* **30**, 481–548. (doi:10.1017/S0140525X07002786)
87. Block N. 2011 Perceptual consciousness overflows cognitive access. *Trends Cogn. Sci.* **15**, 567–575. (doi:10.1016/j.tics.2011.11.001)
88. Block N. 2014 Rich conscious perception outside focal attention. *Trends Cogn. Sci.* **18**, 445–447. (doi:10.1016/j.tics.2014.05.007)
89. Overgaard M. 2018 Phenomenal consciousness and cognitive access. *Phil. Trans. R. Soc. B* **373**, 20170353. (doi:10.1098/rstb.2017.0353)
90. Barbey AK, Koenigs M, Grafman J. 2013 Dorsolateral prefrontal contributions to human working memory. *Cortex* **49**, 1195–1205. (doi:10.1016/j.cortex.2012.05.022)
91. Mackey WE, Devinsky O, Doyle W, Meager MR, Curtis CE. 2016 Human dorsolateral prefrontal cortex is not necessary for spatial working memory. *J. Neurosci.* **36**, 2847–2856. (doi:10.1523/JNEUROSCI.3618-15.2016)
92. Szczepanski SM, Knight R. 2014 Insights into human behavior from lesions to the prefrontal cortex. *Neuron* **83**, 1002–1018. (doi:10.1016/j.neuron.2014.08.011)
93. Ivanova MV, Dragoy OV, Kuptsova SV, Yu Akinina S, Petrushevskii AG, Fedina ON, Turken A, Shklovsky VM, Dronkers NF. 2018 Neural mechanisms of two different verbal working memory tasks: a VLSM study. *Neuropsychologia* **115**, 25–41. (doi:10.1016/j.neuropsychologia.2018.03.003)
94. Dehaene S, Lau H, Kouider S. 2017 What is consciousness, and could machines have it? *Science* **358**, 486. (doi:10.1126/science.aan8871)
95. Lamme VAF, Roelfsema PR. 2000 The distinct modes of vision offered by feedforward and recurrent processing. *Trends Neurosci.* **23**, 571–579. (doi:10.1016/S0166-2236(00)01657-X)
96. Block N. 2005 Two neural correlates of consciousness. *Trends Cogn. Sci.* **9**, 46–52. (doi:10.1016/j.tics.2004.12.006)
97. Lamme VAF. 2006 Towards a true neural stance on consciousness. *Trends Cogn. Sci.* **10**, 494–501. (doi:10.1016/j.tics.2006.09.001)
98. Lamme VAF. 2010 How neuroscience will change our view on consciousness. *Cogn. Neurosci.* **1**, 204–220. (doi:10.1080/17588921003731586)
99. Haynes JD. 2015 An information-based approach to consciousness: mental state decoding. In *Open MIND* (eds T Metzinger, J Windt), p. 17(T). Frankfurt am Main, Germany: MIND Group.
100. Tsuchiya N, Wilke M, Frässle S, Lamme V. 2015 No-report paradigms: extracting the true neural correlates of consciousness. *Trends Cogn. Sci.* **19**, 757–770. (doi:10.1016/j.tics.2015.10.002)
101. Boly M, Massimini M, Tsuchiya N, Postle B, Koch C, Tononi G. 2017 Are the neural correlates of consciousness in the front or in the back of the cerebral cortex? Clinical and neuroimaging evidence. *J. Neurosci.* **37**, 9603–9613. (doi:10.1523/JNEUROSCI.3218-16.2017)
102. Storm J, Boly M, Casali AG, Massimini M, Olcese U, Pennartz C, Wilke M. 2017 Consciousness regained: disentangling mechanisms, brain systems, and behavioral responses. *J. Neurosci.* **37**, 10 882–10 893. (doi:10.1523/JNEUROSCI.1838-17.2017)
103. Sanders R, Mostert N, Lindroth H, Tononi G, Sleigh J. 2018 Is consciousness frontal? Two perioperative case reports that challenge that concept. *Br. J. Anaesth.* online first 1–3.
104. Aru J, Bachmann T, Singer W, Melloni L. 2012 Distilling the neural correlates of consciousness. *Neurosci. Biobehav. Rev.* **36**, 737–746. (doi:10.1016/j.neubiorev.2011.12.003)
105. Overgaard M, Fazekas P. 2016 Can no-report paradigms extract true correlates of consciousness? *Trends Cogn. Sci.* **20**, 214–242. (doi:10.1016/j.tics.2016.01.004)
106. Pitts MA, Martínez A, Hillyard SA. 2012 Visual processing of contour patterns under conditions of inattentional blindness. *J. Cogn. Neurosci.* **24**, 287–303. (doi:10.1162/jocn_a_00111)
107. Pitts MA, Metzler S, Hillyard SA. 2014 Isolating neural correlates of conscious perception from neural correlates of reporting one's perception. *Front. Psychol.* **5**. (doi:10.3389/fpsyg.2014.01078)
108. Pitts MA, Padwal J, Fennelly D, Martínez A, Hillyard SA. 2014 Gamma band activity and the P3 reflect post-perceptual processes, not visual awareness. *Neuroimage* **101**, 337–350. (doi:10.1016/j.neuroimage.2014.07.024)
109. Bayne T, Hohwy J, Owen A. 2016 Are there levels of consciousness? *Trends Cogn. Sci.* **20**, 405–413. (doi:10.1016/j.tics.2016.03.009)
110. Mashour GA, Hudetz A. 2018 Neural correlates of unconsciousness in large-scale brain networks. *Trends Neurosci.* **41**, 150–160. (doi:10.1016/j.tins.2018.01.003)
111. Frässle S, Sommer J, Jansen A, Naber M, Einhäuser W. 2014 Binocular rivalry: frontal activity relates to introspection and action but not to perception. *J. Neurosci.* **34**, 1738. (doi:10.1523/JNEUROSCI.4403-13.2014)
112. Marzano C, Ferrara M, Mauro F, Moroni F, Gorgoni M, Tempesta D, Cipolli C, De Gennaro L. 2011 Recalling and forgetting dreams: theta and alpha oscillations during sleep predict subsequent dream recall. *J. Neurosci.* **31**, 6674. (doi:10.1523/JNEUROSCI.0412-11.2011)
113. Scarpelli S, D'Atri A, Gorgoni M, Ferrara M, De Gennaro L. 2015 EEG oscillations during sleep and dream recall: state- or trait-like individual differences? *Front. Psychol.* **6**. (doi:10.3389/fpsyg.2015.00605)
114. Cipolli C, Ferrara M, De Gennaro L, Plazzi G. 2017 Beyond the neuropsychology of dreaming: insights into the neural basis of dreaming with new techniques of sleep recording and analysis. *Sleep Med. Rev.* **35**, 8–20. (doi:10.1016/j.smrv.2016.07.005)
115. De Gennaro L, Ferrara M. 2017 Brain correlates of successful dream recall. In *Principles and practices of sleep medicine* (eds MH Kryger, T Roth, WC Dement), pp. 523–528, 6th edn. Philadelphia, PA: Elsevier.
116. Mitchell DJ, McNaughton N, Flanagan D, Kirk IJ. 2008 Frontal-midline theta from the perspective of hippocampal 'theta'. *Prog. Neurobiol.* **86**, 156–185. (doi:10.1016/j.pneurobio.2008.09.005)
117. Nyhus E, Curran T. 2010 Functional role of gamma and theta oscillations in episodic memory. *Neurosci. Biobehav. Rev.* **34**, 1023–1035. (doi:10.1016/j.neubiorev.2009.12.014)
118. Hsieh L-T, Ranganath C. 2014 Frontal midline theta oscillations during working memory maintenance and episodic encoding and retrieval. *Neuroimage* **85**, 721–729. (doi:10.1016/j.neuroimage.2013.08.003)
119. Meyer L, Grigutsch M, Schmuck N, Gaston P, Friederici AD. 2015 Frontal–posterior theta oscillations reflect memory retrieval during sentence comprehension. *Cortex* **71**, 205–218. (doi:10.1016/j.cortex.2015.06.027)
120. Scarpelli S, D'Atri A, Mangiaruga A, Marzano C, Gorgoni M, Schiappa C, Ferrara M, De Gennaro L. 2017 Predicting dream recall: EEG activation during NREM sleep or shared mechanisms with wakefulness? *Brain Topogr.* **30**, 629–638. (doi:10.1007/s10548-017-0563-1)
121. Cohen DB. 1972 Failure to recall dream content: contentless vs dreamless reports. *Percept. Mot. Skills* **34**, 1000–1002. (doi:10.2466/pms.1972.34.3.1000)
122. Cohen DB. 1974 Toward a theory of dream recall. *Psychol. Bull.* **81**, 138–154. (doi:10.1037/h0037616)
123. De Gennaro L, Violani C. 1990 White dreams: the relationship between the failure in dream recall and degree of hemispheric lateralization. *Assoc. Study Dreams Newsl.* **7**, 7.
124. Sperling G. 1960 The information available in brief visual presentations. *Psychol. Monogr. Gen. Appl.* **74**, 1–29. (doi:10.1037/h0093759)

125. Landman R, Spekreijse H, Lamme V. 2003 Large capacity storage of integrated objects before change blindness. *Vision Res.* **43**, 149–164. (doi:10.1016/S0042-6989(02)00402-9)
126. Windt JM, Nielsen T, Thompson E. 2016 Does consciousness disappear in dreamless sleep? *Trends Cogn. Sci.* **20**, 871–882. (doi:10.1016/j.tics.2016.09.006)
127. Cohen MA, Dennett D. 2011 Consciousness cannot be separated from function. *Trends Cogn. Sci.* **15**, 358–354. (doi:10.1016/j.tics.2011.06.008)
128. Cohen M, Dennett D. 2012 Response to Fahrenfort and Lamme: defining reportability, accessibility and sufficiency in conscious awareness. *Trends Cogn. Sci.* **16**, 139–140. (doi:10.1016/j.tics.2012.01.002)
129. Fahrenfort JJ, Lamme VA. 2012 A true science of consciousness explains phenomenology: comment on Cohen and Dennett. *Trends Cogn. Sci.* **16**, 138–139. (doi:10.1016/j.tics.2012.01.004)
130. Wechsler D. 1997 *Adult intelligence test administration and scoring manual*. San Antonio, TX, USA: Psychological Corporation.
131. Owen AM, McMillan KM, Laird AR, Bullmore E. 2005 N-back working memory paradigm: a meta-analysis of normative functional neuroimaging studies. *Hum. Brain Mapp.* **25**, 46–59. (doi:10.1002/hbm.20131)
132. Wechsler D. 1997 *Wechsler adult intelligence scale-III*. San Antonio, TX: The Psychological Corporation.
133. Fried PJ, Rushmore RJ, Moss MB, Valero-Cabré A, Pascual-Leone A. 2014 Causal evidence supporting functional dissociation of verbal and spatial working memory in the human dorsolateral prefrontal cortex. *Eur. J. Neurosci.* **39**, 1973–1981. (doi:10.1111/ejn.12584)
134. Pope PA, Brenton JW, Miall RC. 2015 Task-specific facilitation of cognition by anodal transcranial direct current stimulation of the prefrontal cortex. *Cereb. Cortex* **25**, 4551–4558. (doi:10.1093/cercor/bhv094)
135. Ptak R, Schnider A, Fellrath J. 2017 The dorsal frontoparietal network: a core system for emulated action. *Trends Cogn. Sci.* **21**, 589–599. (doi:10.1016/j.tics.2017.05.002)
136. Polich J. 2007 Updating P300: an integrative theory of P3a and P3b. *Clin. Neurophysiol.* **118**, 2128–2148. (doi:10.1016/j.clinph.2007.04.019)
137. Wronka E, Kaiser J, Coenen AM. 2012 Neural generators of the auditory evoked potential components P3a and P3b. *Acta Neurobiol. Exp.* (*Wars*) **72**, 51–64.