Neural correlates of consciousness: progress and problems

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Abstract | There have been a number of advances in the search for the neural correlates of consciousness — the minimum neural mechanisms sufficient for any one specific conscious percept. In this Review, we describe recent findings showing that the anatomical neural correlates of consciousness are primarily localized to a posterior cortical hot zone that includes sensory areas, rather than to a fronto-parietal network involved in task monitoring and reporting. We also discuss some candidate neurophysiological markers of consciousness that have proved illusory, and measures of differentiation and integration of neural activity that offer more promising quantitative indices of consciousness.

Neural correlates of consciousness
(NCC). The minimum neural mechanisms jointly sufficient for any one specific conscious experience.

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doi:10.1038/nrn.2016.22
Published online 20 Apr 2016; corrected online 6 May 2016

Being conscious means that one is having an experience — the subjective, phenomenal 'what it is like' to see an image, hear a sound, think a thought or feel an emotion. Although our waking experiences usually refer to the external world, we continue to be conscious when we daydream and during those periods of sleep when we dream[1]. Consciousness only vanishes during dreamless sleep or under general anaesthesia when, from our own intrinsic perspective, everything disappears and we experience nothing[1]. Understanding the origin of consciousness, how it fits into a physical account of the universe and its relationship with the body are long-standing questions in philosophy, psychology and brain science.

It has been known for a long time that being conscious requires the proper functioning of midline brain structures and that the particular contents of an experience are supported by the activity of neurons in parts of the cerebral cortex[2]. The research strategy to identify the neural correlates of consciousness (NCC) involves relating behavioural correlates of consciousness to the neural mechanisms underlying them. There has been considerable progress in this area since the subject was last reviewed in this journal[3]. In this Review, we start by outlining some contemporary approaches used to characterize the NCC, including the no-report paradigm. This work has led to a shift in our understanding of the location of the NCC, away from a broad fronto-parietal network towards a more restricted posterior cortical hot zone. We discuss how two popular candidate physiological markers of consciousness — gamma activity and the P3b wave — have not shown any predictive power and how other promising quantitative indices of consciousness have been developed. We outline the inherent limits of such an empirical research programme. This Review focuses on visual and auditory studies; for accounts of the NCC for metacognition, body, tactile and olfactory experiences, see REFS 4–7.

Behavioural correlates of consciousness

Although experiences are private, we can usually infer that people are conscious if they are awake and act purposefully, in particular if they can report what they experience and if that report accords with what is experienced by others. In a clinical setting, simple behavioural criteria are often used to infer consciousness, such as the ability to respond to a command (for example, patients may be asked to squeeze the observer's hand twice if they feel pain). The level of consciousness[2,8] is typically assessed by assigning a rating to a subject's auditory, visual, verbal and motor functions using standardized scales (such as REF. 9). However, consciousness can be present even in the absence of reliable behavioural responses. For example, minimally conscious patients can be misclassified as being in a non-conscious, vegetative state, with significant clinical, practical and emotional consequences, if careful and repeated assessments are not carried out[10,11].

In an experimental setting, the content of consciousness is typically evaluated by verbal report or by button-press by the participant in response to a yes or no question (such as "did you see a face?"). Such reports can vary, however, as a result of criterion shifts about what counts as seen for that individual participant, especially when the content is at or near perceptual thresholds[12]. Forced-choice procedures during such report tasks[13] can establish whether a participant detects or discriminates a stimulus above chance levels, and so determine whether they are objectively aware of it. For example, patients with lesions in their visual cortex may report that they

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are unaware of a visual stimulus (a subjective measure), while performing above chance in tests requiring stimulus detection (an objective measure), a phenomenon referred to as blindsight[14,15].

A more refined way of characterizing experience is to use a perceptual awareness scale[16], which ranges from "no experience", to "brief glimpse", "almost clear image" and "absolutely clear image". Participants can also rate confidence in their judgement using a scale that runs from "completely guessing" to "fully confident", or by making an economic judgement following each response (referred to as post-decision wagering). When such report protocols are implemented judiciously, subjective and objective measures of consciousness are in concordance with each other[17]. The use of paradigms that include such confidence measures has been extended to non-human species[18–20].

Neural correlates of consciousness

The NCC are defined as the minimum neuronal mechanisms jointly sufficient for any one specific conscious percept[21,22]. There are two possible interpretations of this definition, depending on whether we are referring to the specific content of consciousness or to the overall state of being conscious.

The content-specific NCC are the neurons (or, more generally, neuronal mechanisms), the activity of which determines a particular phenomenal distinction within an experience. For example, the NCC for experiencing the specific content of a face are the neurons that fire, on a trial-by-trial manner, whenever a person observes, imagines or dreams a face, and are silent in other circumstances[22]. When the content-specific NCC neurons in this example are activated artificially — for example, by transcranial magnetic stimulation (TMS), electrical stimulation or optogenetic stimulation — the participant should see a face even if none is present, whereas if their activity is blocked, the participant should not be able to see a face even if one is present.

The full NCC, as defined here, are the neural substrates supporting conscious experiences in their entirety, irrespective of their specific contents. This is the union of the sets of content-specific NCC for all possible contents of experience. It is also important, both conceptually and empirically, to distinguish between the NCC and the background conditions for being conscious. These are factors that enable consciousness without contributing directly to its content, such as appropriate glucose and oxygen levels, an appropriate neuromodulatory milieu and afferent inputs that ensure adequate cortical excitability.

Identifying the content-specific NCC. To identify the content-specific NCC, neural activity when a particular stimulus (such as a face) is perceived is compared with neural activity when that stimulus is not perceived[23], with the sensory stimulus and the overall state of the participant kept constant under both circumstances (FIG. 1a). Classic report-based visual paradigms to identify the content-specific NCC include binocular rivalry[24], interocular suppression[25], bi-stable perception (such as the Necker cube test, face-vase test and the Mooney face test[26]) and various visual masking techniques (such as forward and backward masking[27] and metacontrast masking[28]). Such report-based paradigms have revealed that a broad fronto-parietal network is activated during visual–motor tasks that contrast perceived stimuli with invisible stimuli.

It has become clear, however, that at least part of the neural activity that co-varies with the perception of a particular conscious content reflects processes that precede or follow the experience — such as selective attention, expectation, self-monitoring, unconscious stimulus processing, task planning and reporting[29–32] — rather than the experience itself. Recent experimental paradigms have been designed to address these issues. To distinguish the true, content-specific NCC from neural activity that is merely a prerequisite for consciousness, factors that modify consciousness thresholds (such as stimulus expectation, adaptation, working memory or the allocation of attention) are varied systematically[32,33]. This can be achieved by matching performance[33], manipulating task relevance[34] or using a no-report paradigm[33,35] during which participants do not make any overt report, such as speaking or pushing a button (FIG. 1b). For example, a recent study using binocular rivalry[36] showed that eye movements and pupil dilation correlate tightly with conscious reports of perceptual dominance using a standard button-push paradigm. Eye movements and pupil dilation alone subsequently acted as proxies for the participants' reports during sessions in which the NCC were identified by functional MRI (fMRI) while the participants viewed the stimuli passively[36]. Such no-report paradigms identify a more restricted content-specific NCC, which typically includes posterior cortical areas but not the prefrontal cortex.

Identifying the full NCC. The neural mechanisms that are jointly sufficient for being conscious in a broad sense, irrespective of the particular contents of experience, are usually identified through state-based approaches. These involve contrasting brain activity when consciousness is present, typically in awake healthy participants performing no task, with brain activity when consciousness is severely diminished — for example, during dreamless sleep[37,38] (FIG. 1c), general anaesthesia[39,40] or disorders of consciousness such as coma and vegetative states (also known as unresponsive wakefulness syndromes)[41,42]. Similar to findings from studies of content-specific NCC, such studies of the full NCC often find that a fronto-parietal network is activated when one is conscious. However, it is important to note that major changes in the physiological state of the brain alter not only consciousness but also several other brain functions, such as vigilance and attention, that depend on levels of arousal-promoting neuromodulators[43].

One approach to address these confounds is to use a within-state, no-task paradigm, taking advantage of spontaneous fluctuations of consciousness. For example, during sleep, participants are disconnected from the environment and are not performing a task[44] (FIG. 1d). If awakened from the same behaviourally defined state, such as non-rapid eye movement (NREM) sleep, participants at times report that they were having conscious experiences (dreams) and at other times that they were unconscious. Activity seen by electroencephalography (EEG)

No-report paradigm
A paradigm in which trials with explicit report are included along with trials without explicit report, during which indirect physiological measures are used to infer what the participant is perceiving. This paradigm allows the neural correlates of consciousness to be distinguished from events or processes that are associated with, precede or follow conscious experience.

Hot zone
A temporo-parietal-occipital zone of the posterior cerebral cortex where the best current anatomical candidates for full and content-specific neural correlates of consciousness in the human brain are located. The content-specific neural correlates of consciousness may be any particular subset of neurons within this hot zone that supports specific phenomenological distinctions, such as faces.

Vegetative state
A disorder of consciousness that occurs in some patients with brain injury. The patients remain unresponsive and show no purposeful behaviour, but retain the ability to spontaneously open their eyes and maintain autonomic reflexes.

Content-specific NCC
(Content-specific neural correlates of consciousness). The neural substrate supporting a particular content of experience (for example, faces) whether seen, dreamt or imagined.

Transcranial magnetic stimulation
(TMS). A method of non-invasive brain stimulation, in which a magnetic field is induced by an electrical current in a coil placed onto the skull to induce neuronal activity in the underlying cortex.

Full NCC
(Full neural correlates of consciousness). The neural substrate supporting experience in general, irrespective of its specific content.

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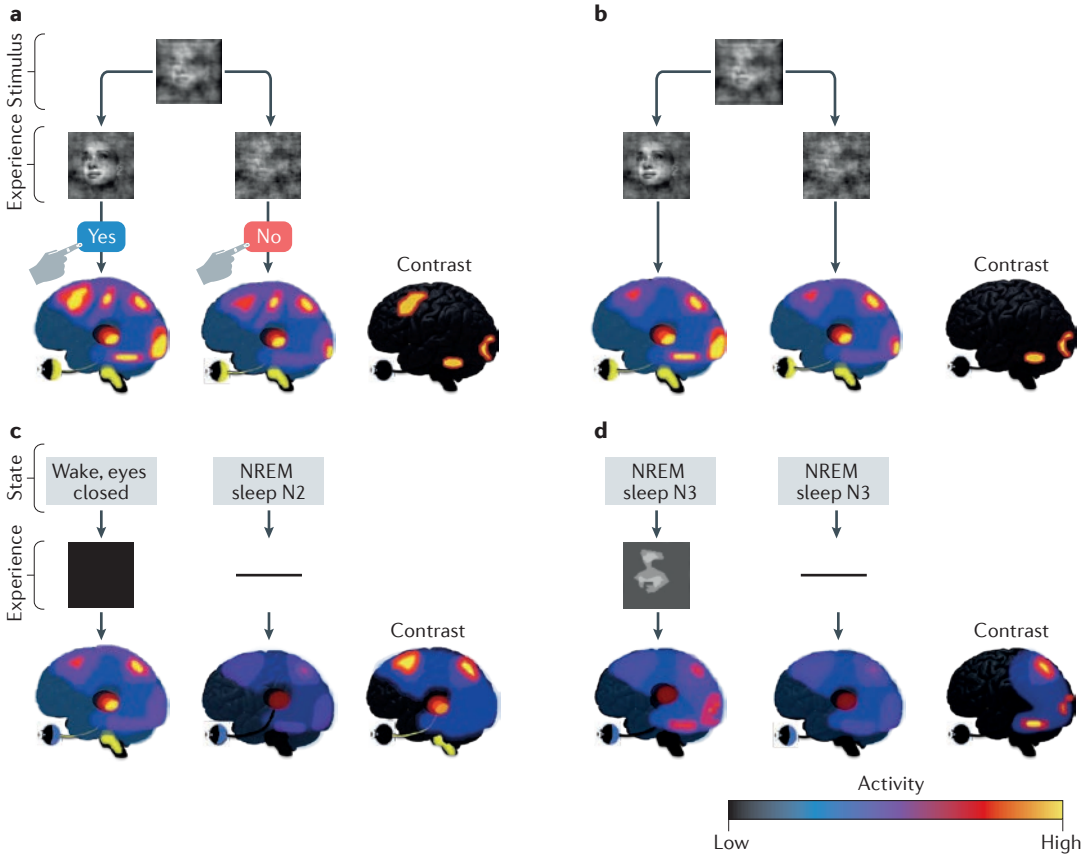

Figure 1 | **Identifying the neural correlates of consciousness.** This schematic diagram illustrates the absolute levels of brain activity across different conditions designed to identify the neural correlates of consciousness (NCC). **a** | Haemodynamic activity in a report-based paradigm in which the experience of seeing a face or other stimulus manipulations such as backward masking is contrasted with not seeing it as a result of image noise[142]. The participant presses a button to report seeing (yes) or not seeing (no) the face. This isolates the brain regions involved in conscious face recognition, a content-specific NCC, but also the regions involved in the maintenance of the face in working memory and access to the concept 'face' for decision making. Note that the background conditions for consciousness, such as an active reticular activating system, are eliminated by this contrast. **b** | Haemodynamic activity in a no-report, seen/not seen paradigm, similar to part **a**, in which the participants do not report on their percept during the trial. Instead, either retrospective reports or physiological indicators, such as eye movements or pupil dilation, are used to avoid confounds related to the act of reporting[34,36]. This allows the content-specific NCC to be isolated. The union of all such content-specific NCC constitutes the full NCC. **c** | In a between-states paradigm, the haemodynamic activity associated with the experience of seeing anything (here the experience of seeing pure black) while awake is contrasted with the activity associated with the condition of no experience that would occur during dreamless sleep[37]. This analysis identifies brain regions involved in having any conscious experience, but also regions that mediate being awake rather than being asleep. **d** | In a within-state, no-task paradigm, participants are awakened at random during sleep (stage N2) and are asked whether, before they were awakened, they were dreaming and, if so, about what. This paradigm identifies brain activity associated with having dream experiences up to 20 s before awakening[44] — a candidate for the full NCC of dreaming consciousness — and avoids confounds associated with task execution and state change. NREM, non-rapid eye movement.

Background conditions
Factors that enable consciousness, but do not contribute directly to the content of experience.

Binocular rivalry
An experimental paradigm used to evoke bi-stable percepts in which one image is presented to the left eye of the participant and a different image is presented to the right eye; rather than seeing a juxtaposition or fusion of both images, participants see one or the other object alternately.

Coma
A disorder of consciousness occurring in some patients with brain injury, in which the patient remains in an enduring, sleep-like state of immobility and unresponsiveness, with their eyes closed, from which they cannot be aroused.

during sleep (either NREM or REM sleep) preceding retrospective reports of consciousness and unconsciousness can then be contrasted within the same physiological state. Such experiments indicate that, in both NREM and REM sleep, the full NCC appear to be localized to a temporo-parieto-occipital region associated with perceptual experiences and to a frontal region associated with thought-like experiences[44]. High-frequency activity in the posterior cortical regions predicts the perceptual categories experienced during dreaming, such as faces, spatial setting, movements and speech[44]. The posterior cortical zone identified through this within-state, no-task paradigm largely overlaps with the union of cortical areas identified in studies of content-specific NCC during waking and with neuropsychological evidence. Because of this converging evidence, the posterior cortical region can be considered as a hot zone for the NCC.

Neuroanatomy

Subcortical structures. Many brain regions, despite having a large number of neurons, an intricate architecture and plentiful inputs and outputs, do not contribute directly to the content of our experiences. For example, the cerebellum has four times more neurons than the

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cortex[45], is densely connected to the rest of the brain, receives mapped inputs from several modalities and is heavily involved in input and output control[46]. Lesions of the cerebellum have little effect on consciousness and its contents, however[47]. A recent report described a woman with complete cerebellar agenesis and mostly preserved cognitive functions[48].

By contrast, brainstem lesions typically cause immediate coma[2] by damaging the reticular activating system[49] and its associated neuromodulatory systems[50]. However, neurological patients with a severely damaged cortex, but with relatively spared brainstem function, typically remain in a vegetative state. This suggests that brainstem activity is insufficient to sustain consciousness in a clinical sense[2] (but see REF. 51). Rather, it is likely that the activity of heterogeneous neuronal populations within the brainstem, hypothalamus and basal forebrain, which project diffusely to thalamic and cortical neurons and promote their depolarization, provides an important background condition for enabling consciousness by facilitating effective interactions among cortical areas[49–51]. Nonetheless, the activity of subcortical neuromodulatory systems may not be strictly necessary for consciousness if an appropriate subset of cortical regions has sufficient intrinsic activation[52]. Indeed, when awakened from NREM sleep, when the activity of most neuromodulatory systems is reduced[53], participants report having been conscious before being woken in more than one-third of instances[54].

Unilateral or bilateral lesions of the basal ganglia can produce akinetic mutism[55], an abulic, emotionless state associated with preserved tracking of visual stimuli[56] that is difficult to evaluate with respect to experiential content. In such patients, basal ganglia atrophy correlates with the loss of alertness, but not with the degree of responsiveness[57]. It is known that large, bilateral basal ganglia haemorrhagic lesions can occur without significant alteration in the level of consciousness[58]. For example, children affected by familial bilateral basal ganglia necrosis may not be able to speak, but are able to respond to sensory stimuli and communicate with their parents in other ways[59]. Similarly, adults with late onset familial dystonia and lesions throughout the basal ganglia may develop cognitive problems, which can include a frontal syndrome, but are nonetheless clearly conscious[60]. Thus, despite the many loops that link the cortex, basal ganglia and thalamus, and then go back to the cortex, and the involvement of such loops in cognitive, limbic, motor and oculomotor functions[61,62], it remains unclear whether the basal ganglia contribute to consciousness directly or only indirectly (either as enabling factors or by implementing unconscious functions).

The claustrum, a thin sheet of cells underneath the insular cortex and above the putamen that is connected bidirectionally with most cortical regions[63], was hypothesized to play a critical part in information integration leading to consciousness[64]. A single case report of a patient implanted with electrodes for remedial surgery to treat epileptic seizures describes how electrical stimulation of the white matter tract underneath the claustrum caused the patient to stare blankly ahead, seemingly unconscious, until the stimulation had stopped, without subsequent recall and without causing any seizure activity[65]. However, another case report describes a patient who was conscious despite the fact that the claustrum and neighbouring limbic structures, including the insular cortex, had been destroyed bilaterally as a consequence of herpes simplex encephalitis[66].

The role of the thalamus in consciousness remains controversial. Small bilateral lesions in the intralaminar nuclei of the thalamus can lead to coma[67,68], and chronic thalamic electrical stimulation may promote recovery in some patients with disorders of consciousness[69]. Although the so-called core neurons in primary thalamic nuclei have focused connectivity, several higher-order thalamic nuclei are rich in widely projecting matrix cells[70], which may facilitate interactions among distant cortical areas[71]. Thus, some thalamic cells may represent critical enabling factors for consciousness. Nonetheless, extensive lesions of the thalamus in rodents have found little effect on EEG recordings, behavioural measures or FOS expression in the cortex during wakefulness[72]. In addition, structural imaging of 143 patients with severe brain injury and disorders of consciousness revealed no correlation between the level of arousal and thalamic atrophy, but did find a correlation between decreased motor function and/or the ability to communicate and thalamic atrophy[57]. The activity of thalamic relay nuclei correlates poorly with the level of consciousness in patients who are in a vegetative state[73]. With regard to the role of the thalamus in content-specific NCC, electrophysiological recordings from macaque monkeys show that activity in neurons in the lateral geniculate nucleus of the thalamus does not correlate with the percept reported by the animal[74,75], whereas activity in neurons in the pulvinar (a higher-order thalamic nucleus) does correlate[75,76]. The contribution of the higher-order thalamus to consciousness has been challenged by other findings, however. In humans, unawareness induced by anaesthesia correlates more closely with the impairment in cortical function than with thalamic activity[77,78]. When participants experience vivid sleep-onset hallucinations, the thalamus is already deactivated[79].

Cerebral cortex: primary versus higher areas. Whether the primary visual cortex (V1) contributes to visual consciousness directly or whether it has only an indirect role — much like the retina's role in visual consciousness — is the subject of ongoing debate[80,81]. Several visual stimuli that are known to affect the activity of V1 neurons do not elicit a corresponding visual experience[82,83]. Similarly, it is possible to decode the orientation of a masked stimulus from the haemodynamic activity of the participants' V1 while the stimulus is presented, although the participants cannot correctly guess the orientation[84]. Single-neuron recordings in monkeys, carried out during paradigms such as binocular rivalry, suggest that activity in most V1 neurons is linked to the identity of the physical stimulus rather than the percept. This contrasts with the activity of neurons higher up in the visual hierarchy, which correlates with the percept rather than the stimulus[24,85,86]. Although several fMRI studies have found that activity in human V1 can be correlated with perception during

Abulic
A state associated with impairment in decision making and loss of self-initiated actions.

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binocular rivalry[87,88] or working memory tasks[89], this is not true for motion-induced blindness[90]. Lesions of V1 lead to the striking phenomenon of unconscious vision or blindsight, whereby the affected participants report not seeing an item but still perform above chance on forced-choice tasks[91]. Their subjective blindness could be a result of the insufficient feedforward activation of higher visual areas, or to lack of feedback to V1, in which case V1 would be necessary for conscious vision. Patients with damaged extrastriate visual areas and intact V1 can have quadrantanopia, however[92] (that is, blindness for one-quarter of the visual field), suggesting that an intact V1 alone is insufficient for conscious vision. Conversely, in patients with blindsight and a damaged V1, but intact extrastriate areas, phosphene-like percepts can be induced by stimulating the parietal cortex using TMS[93]. Taken together, the evidence discussed here supports the hypothesis that V1 is not part of the content-specific NCC for the attributes that have been tested so far. It is not known whether this conclusion applies to other visual attributes, such as precise spatial topography, luminosity and detailed contours[94–96]. Whether other primary areas, such as the primary somatosensory and auditory cortex, are or are not part of the content-specific NCC remains to be determined (for examples, see REFS 97–99).

A case has been made for distinguishing between a ventral cortical stream for perception and a dorsal stream for action[100]. According to this view, activity in the ventral stream is involved in conscious, stable visual object identification and scene formation[101,102], whereas the dorsal stream deals with the unconscious, moment-to-moment visual control of skilled actions[103]. However, the dichotomy between ventral and dorsal streams in terms of the identity versus the location of visual stimuli has been called into question[104], suggesting that both dorsal and ventral streams are important for visual experience[105,106].

Human neuroimaging studies that contrast brain activity during wakefulness with brain activity during the vegetative state[107], general anaesthesia[108] or deep sleep[109,110] indicate that there is reduced activity in the fronto-parietal cortices during loss of consciousness. The area with the strongest correlation between decreases in brain activity and loss of consciousness is the posteromedial cortex[8,111]. These findings suggest that the fronto-parietal cortices are potential candidates for the full NCC. However, other findings contradict this interpretation, including the observation that blood flow is increased in the fronto-parietal areas during generalized seizures when patients are unconscious[112]. Unlike findings from report-based paradigms, no-report paradigms suggest that frontal activation is more important for task preparation and execution than for conscious perception per se[36] (but see REF. 113).

A predominant involvement of the posterior cortex in content-specific consciousness is seen when matching expectations and performance during stimulus presentation[114], when contrasting awareness with task relevance[34] and in many other recent EEG, magnetoencephalography and electrocorticography experiments[29,34,115–117]. As mentioned earlier, experiments using a no-task, within-state paradigm during both NREM and REM sleep also highlight a posterior cortical hot zone[44]. In fact, during REM sleep, when participants are dreaming vividly, activity in the prefrontal cortex is often low compared with the awake state[118]. In addition, multivariate pattern analysis of fMRI data can decode the identity of visual stimuli from activity in the medial and lateral occipital regions, but not from the prefrontal cortex and intraparietal sulcus, despite the fact that these areas show task-related activation. The decoding strength in the extrastriate visual cortex correlates with the precision of visual working memory for the stimuli, whereas decoding of the frontal cortex and intraparietal sulcus activity correlates with the type and difficulty of the task and the attentional context[119].

More than a century of reports describing electrical brain stimulation carried out during neurosurgery suggest that it is difficult to directly elicit experiences from the stimulation of frontal sites, whereas it is easier to trigger specific experiences by stimulating the posterior cortex[120], such as the perception of faces[121] or the feeling of wanting to move a limb[122]. Most importantly, the commonly held view that the content of consciousness is linked to fronto-parietal activation ignores the ample evidence obtained from lesion studies that consciousness does not require an intact prefrontal cortex. For example, complete bilateral frontal lobectomy[123] and large bilateral prefrontal resections do not impair consciousness[124]. Likewise, prefrontal lobotomy and leucotomy, in which the prefrontal cortex is cut off from its thalamic inputs, do not impair consciousness[125]. In addition, participants who underwent widespread bilateral resection of their prefrontal areas (termed topectomy, a procedure that is sometimes combined with lobotomy) do not show gross behavioural changes and certainly maintain consciousness[126]. More recently, a young woman with extensive bilateral prefrontal damage of unclear aetiology was found to have grossly deficient scores in tests of frontal lobe function, but intact perceptual abilities, and was obviously conscious[127] (see also REF. 128). Taken together, the data suggest that the frontal portions of the broad fronto-parietal network once commonly thought to be necessary for consciousness may play a more prominent part in the control and execution of cognitive tasks than in supporting experience per se (FIG. 2).

Cortical neural responses to visual stimuli, such as natural scenes and faces, can occur quickly (within 120–140 ms) and presumably are mediated by a feedforward volley through the V1 into the extrastriate cortex and inferior temporal cortex[129–131]. It has been suggested that a stimulus-evoked feedforward sweep only gives rise to a conscious percept when it is joined by a re-entrant sweep from the higher-level cortex coming back to the visual cortex[132,133]. In monkeys trained to signal whether or not they saw a salient figure embedded in a background image, the early feedforward response of V1 neurons was the same[134]. By contrast, a later feedback component was suppressed only when the monkey did not see the figure, and this later component (but not the early one) was eliminated by light anaesthesia. Directional connectivity studies in humans[135] and in mice[136] suggest that top-down connectivity is stronger when somato sensory stimuli are perceived than when they are not.

Phosphene
A visual experience, in particular featuring flashes of light, that occurs in response to direct mechanical, electrical or magnetic stimulation of the visual cortex.

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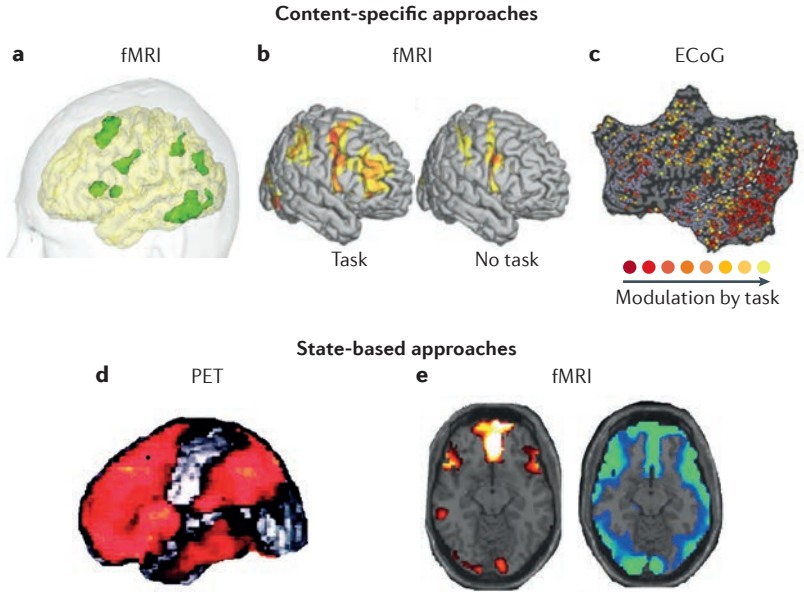

Figure 2 | **Identifying content-specific and full neural correlates of consciousness.** **a** | Findings from a functional MRI (fMRI) experiment searching for content-specific neural correlates of consciousness (NCC). This experiment contrasts activity during the presentation of visual words with activity when the same words are masked and therefore invisible. This task involves an explicit report via button-press. Such studies have suggested that activity in the fronto-parietal networks and extrastriate occipital regions[142] constitute the NCC. **b** | Similar fronto-parietal activity patterns are found during a binocular rivalry paradigm in which volunteers actively report their percept (task). However, when the volunteers experience rivalry without explicitly reporting perceptual alternations (no task), neural activity in the frontal areas is absent, whereas activation of the occipital and parietal regions remains[36]. **c** | Findings from an electrocorticogram (ECoG) study using a visual one-back paradigm in patients with epilepsy implanted with subdural electrode arrays. Participants press a button when two consecutive pictures are identical (the target trial). The colours indicate how strongly neural activity is modulated by the motor task (red represents low modulation and yellow represents high modulation) across all significantly responsive electrodes (either during non-target or target trials). Occipito-temporal visual cortical regions (shown to the right of the dashed white line) respond rapidly to the seen stimulus irrespective of the type of task (red), whereas the frontal regions are modulated by the type of task (yellow)[117]. **d** | Findings from a positron emission tomography (PET) study using a between-states paradigm that contrasts brain activity during deep non-rapid eye movement (NREM) sleep with wakefulness indicate that there are decreased levels of absolute cerebral blood flow in both parietal and frontal cortices during sleep. This suggested that activity in a fronto-parietal network may be essential for consciousness[110]. **e** | Voxel-by-voxel maps of the mean percentage change in fMRI signals during absence seizures: fronto-parietal activity increases during the loss of consciousness (warm colours) and decreases during the subsequent recovery of consciousness (cool colours)[112]. Part **a** is adapted with permission from REF. 142, Elsevier. Part **b** is adapted from REF. 36, republished with permission of Society for Neuroscience, from Binocular rivalry: frontal activity relates to introspection and action but not to perception, Frässle, S., Sommer, J., Jansen, A., Naber, M. & Einhäuser, W., **34**, 5, 2014; permission conveyed through Copyright Clearance Center, Inc. Part **c** is adapted with permission from REF. 117, Elsevier. Part **d** is adapted from REF. 110, republished with permission of Society for Neuroscience, from Activity of midbrain reticular formation and neocortex during the progression of human non-rapid eye movement sleep, Kajimura, N., Uchiyama, M., Takayama, Y., Uchida, S., Uema, T., Kato, M., Sekimoto, M., Watanabe, T., Nakajima, T., Horikoshi, S., Ogawa, K., Nishikawa, M., Hiroki, M., Kudo, Y., Matsuda, H., Okawa, M. & Takahashi, K., **19**, 22, 1999; permission conveyed through Copyright Clearance Center, Inc. Part **e** is adapted from REF. 112, republished with permission of Society for Neuroscience, from Dynamic time course of typical childhood absence seizures: EEG, behavior, and functional magnetic resonance imaging, Bai, X., Vestal, M., Berman, R., Negishi, M., Spann, M., Vega, C., Desalvo, M., Novotny, E. J., Constable, R. T. & Blumenfeld, H., **30**, 17, 2010; permission conveyed through Copyright Clearance Center, Inc.

Neuroimaging studies of patients with brain injuries[137] and sedated patients[77,138] that used dynamic causal modelling and other indices of connectivity also support the hypothesis that preserved feedback connectivity is necessary for consciousness[96,132,139–142].

Cell-type specificity. An outstanding question is whether distinct cortical cell types — such as the spindle neurons (also known as von Economo neurons) found in layer 5 of the frontal lobe in species with large, convoluted brains[143]— are associated with specific aspects of consciousness. Auto-radiographic studies in cats have shown that the infragranular layers of the visual cortex have reduced activity during slow wave sleep[144]. Large, thick-tufted pyramidal neurons found in a variety of species in layer 5B seem well equipped to integrate local and long-distance inputs[145] through their distal apical tufts that receive large amounts of feedback from higher-level cortical regions. However, these thick-tufted cells project primarily to subcortical structures, and their limited connectivity within the cortex suggests that they may be better suited to implement cortico–subcortico–cortical loops that mediate unconscious functions and behaviours[146]. By contrast, thin-tufted pyramidal cells that exclusively form cortico–cortical connections in layer 5A are more heavily interconnected, as are some cells in layer 6 (REF. 146).

Supragranular pyramidal cells are even more densely connected than layer 5A neurons[146–148]. Both the feedforward projections from, and feedback projections to, these neurons originate in the superficial layers of the cortex and exhibit greater topographic precision than those originating in infragranular layers[149]. The apical dendrites of the supragranular pyramidal cells — as well as the different types of inhibitory interneurons located in superficial layers[150] — are also a target of feedback connections and of diffusely projecting thalamic matrix cells[146]. In addition, although infra- and supragranular layers are interconnected, they can become uncoupled by attentional processes[151,152] and sleep[153]. Functionally, excitatory neurons in the supragranular cortex are more specific in their discharge properties than infragranular neurons[154]. Neuronal avalanches — spatiotemporal patterns of spontaneous activity that reflect a critical state of network excitability favourable to information integration — only occur in supragranular layers[155]. Across many species, neural activity in the supragranular layers often correlates with conscious sensation. For example, a negative slow potential that is likely to originate from supragranular layers[156] occurs between the onset of a stimulus and the behavioural response only when a near-threshold stimulus is perceived[157,158]. Under propofol anaesthesia, this late negative shift in slow cortical potentials disappears, whereas the early positive component is preserved[159]. As another example, in the somatosensory cortex of awake monkeys there is a correlation between conscious detection and the magnitude and latency of the N1 event-related potential (ERP) generated by excitatory input onto supragranular layers (which is most likely to be feedback connections originating in higher cortical areas)[99]. Notably, the N1 is abolished during general anaesthesia[160] and slow wave sleep[161].

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Neurophysiology

Gamma synchrony. When the systematic search for the NCC began, the discovery of synchronized neuronal discharges in the visual cortex in response to two jointly moving visual stimuli in the low gamma range (30–70 Hz) in anaesthetized and awake cats generated much excitement[162]. This led to the proposal that consciousness requires the synchronization of populations of neurons via rhythmic discharges in the gamma range[21], which might account for the binding of multiple stimulus features within a single experience[163] (FIG. 3a–c). Stimulus-specific gamma range synchronization in the visual cortex of cats is facilitated by attention[164] and by the stimulation of the reticular formation[165,166]. Gamma synchrony reflects perceptual dominance during binocular rivalry, although firing rates may not change[167]. Human EEG and magnetoencephalography studies also suggest that long-distance gamma synchrony may correlate with visual consciousness[168,169].

However, many of these studies did not carefully dissociate conscious visibility from selective attention (see Supplementary information S1 (box)). When this is done, high-range gamma synchronization correlates with attention, independent of whether the stimulus was seen by the participant, whereas mid-range gamma synchronization correlates with the visibility of the stimulus[170] (see also REF. 33). Gamma synchrony can persist or even increase during early NREM sleep, during anaesthesia[171,172] or during seizures[173], and can be present during exposure to stimuli that induce unconscious emotional responses[174], suggesting that gamma synchrony can occur in the absence of consciousness. An electrocorticography study of the human visual cortex showed that narrowband gamma oscillations are reliably elicited by some spatial patterns, such as luminance gratings, but not by others, such as noise patterns and natural images that observers readily see and recognize[175]. This has led to the conclusion that gamma band oscillations are not necessary for seeing[176].

P3b. Another well-studied electrophysiological candidate marker of consciousness is a late (>300 ms after the onset of stimulus), positive, fronto-parietal ERP evoked by visual or auditory stimuli, called the P3b, which was first described 50 years ago[177]. Studies using content-specific, task-based paradigms using masking, attentional blink and manipulations of stimulus strength show that the P3b component of the ERP is a robust correlate of the subjective report of stimulus detection[17,141,178] (FIG. 3d–f). Thus the P3b, measured using an auditory oddball paradigm, has been proposed as a signature of consciousness, revealing a non-linear amplification (also referred to as ignition) of cortical activity through a distributed network involving fronto-parietal areas[142].

However, this interpretation has been contradicted by several experimental results. For example, task-irrelevant stimuli do not trigger a P3b[179] even when participants are clearly conscious of them[34], whereas stimuli that are not consciously detected can trigger a P3b[180]. The P3b does not signal conscious perception when participants already have a target stimulus held in working memory[114]. Findings from between-state paradigms have also cast doubt on the use of P3b ignition as a reliable signature of consciousness because the P3b elicited by global violations of auditory regularities (for example, sequences of same–different tones) does not discriminate between vegetative (presumably unconscious) and minimally conscious patients[181]. In fact, the P3b is absent in the majority of patients with brain damage who remain clearly conscious[182–184] and has minimal sensitivity (0.14) in identifying minimally conscious patients[185]. Multivariate decoding of the P3b signal can improve the sensitivity of global violations in response to auditory regularities[186]. When this kind of analysis is applied, however, it is possible to find a P3b-like component in 40% of coma patients who are unresponsive, sedated and hypothermic[187]. Hence, the P3b is not a marker of consciousness.

The visual awareness negativity, an ERP deflection that starts as early as 100 ms after the onset of a stimulus, peaks around 200–250 ms and is localized to the posterior cortex (occipital, temporal and posterior parietal lobes)[34,188], may correlate better with conscious perception. Even earlier parietal and temporal evoked responses (at around 70 ms after the onset of a stimulus) correlate with the perception of phosphenes elicited by parietal and occipital TMS, respectively[106].

Activated EEG. The low-voltage fast activity observed with EEG recordings performed during attentive wakefulness, also known as activated or desynchronized EEG[49], was one of the first candidate electrophysiological indices of consciousness and is still one of the most sensitive and useful markers available. Over the past 30 years, intracellular recordings from cortical and thalamic neurons in cats have revealed the mechanisms that underlie the transition from the low-voltage fast activity that characterizes wakefulness to the high-voltage, slow activity that characterizes deep slow wave sleep and some forms of anaesthesia[189]. When thalamic neurons are hyperpolarized, they switch from a tonic to a bursting firing mode, resulting in the synchronization of the EEG in the spindle (12–14 Hz) and theta (5–8 Hz) ranges[189]. Larger oscillations in the delta range (<4 Hz; also known as slow waves) appear in the EEG when cortical neurons start alternating synchronously between depolarized up-states and hyperpolarized down-states every second or so[190] (FIG. 3g). This sequence of physiological events accompanies a loss of consciousness in physiological, pharmacological and pathological conditions[40]. In general, slow waves can be initiated by a decrease in the firing rate of subcortical activating systems[49,191], by excessive inhibition exerted by the globus pallidus on the thalamus[192] or by a critical level of cortical deafferentation[193] common in comatose patients[194].

Detecting repeated high-amplitude slow waves (>75 μV) in the EEG is an effective way to assess the loss of consciousness in humans and is better than most available alternatives[172,195]. For example, slow waves are prominent during deep sleep early in the night when participants, if awakened, often claim they had not experienced anything at all[54]. Similarly, a sudden increase in slow wave power coincides with the behavioural loss of consciousness on

Auditory oddball paradigm
A sequence of auditory stimuli in which the last stimulus differs from the preceding stimuli; for example, a sequence of low-frequency tones followed by a single high-frequency tone

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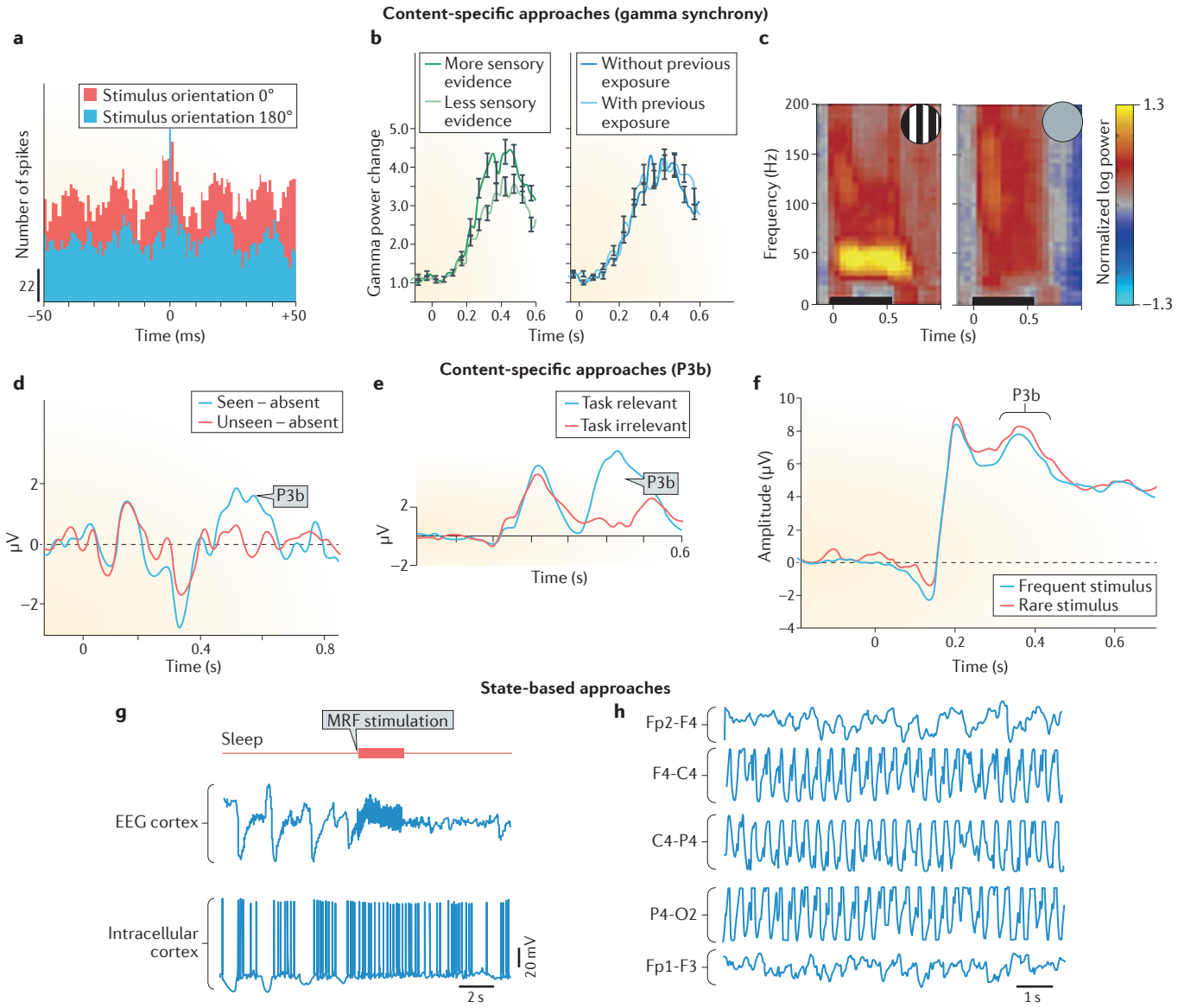

Figure 3 | **Candidate neurophysiological markers of consciousness. a** | Gamma synchrony has been proposed as a correlate of consciousness. Cross-correlograms computed from the firing responses of two units recorded 7 mm apart in the visual cortex of cats reveal a strong gamma band synchronization when a moving bar stimulates the receptive fields of the two neurons[162]. **b** | In the high-level visual cortex in humans, however, the intracranial gamma band responses increase when a degraded stimulus is made visible by increasing sensory evidence, but not when visibility is increased to the same extent by previous exposure[33]. **c** | Visible grating stimuli elicit robust gamma oscillations (30–80 Hz) in the human visual cortex, whereas equally visible noise patterns or natural images do not, indicating that gamma activity is not necessary for visual conciousness[175]. **d** | The late positive component of the event-related potential (known as the P3b) was also proposed as a signature of consciousness. In attentional blink paradigms, the P3b is a robust correlate of stimulus visibility[178]. **e** | When visual awareness and task relevance are manipulated independently, however, the P3b is absent for task-irrelevant stimuli regardless of whether the participants are aware of them or not[34]. **f** | Stimuli that are not consciously detected can trigger a significant P3b component[180]. **g** | A shift in electroencephalography (EEG) activity from low to high frequencies has been proposed as a correlate of consciousness. Electrical stimulation of the midbrain reticular formation (MRF) elicits EEG activation and behavioural awakening in a cat; EEG slow waves are replaced by low-voltage fast activity and by a steady membrane potential in the cortex[247,248]. **h** | High-amplitude bilateral slow waves have been observed in awake, conscious patients in some cases of non-convulsive status epilepticus[199]. Part **a** is from REF. 162, Nature Publishing Group. Part **b** is adapted from REF. 33, Society for Neuroscience, from Local category-specific gamma band responses in the visual cortex do not reflect conscious perception, Aru, J., Axmacher, N., Do Lam, A. T., Fell, J., Elger, C. E., Singer, W. & Melloni, L., **32**, 43, 2012; permission conveyed through Copyright Clearance Center, Inc. Part **c** is adapted from REF. 175, Hermes, D., Miller, K. J., Wandell, B. A. & Winawer, J. Stimulus dependence of gamma oscillations in human visual cortex. *Cereb. Cortex*, 2015, **25**, 9, 2951–2959, by permission of Oxford University Press. Part **d** is from REF. 178, Nature Publishing Group. Part **e** is adapted from REF. 34. Part **f** is adapted with permission from REF. 180, Elsevier. Part **g** is adapted with permission from REF. 248, American Association for the Advancement of Science. Part **h** is adapted with permission from REF. 199, John Wiley &Sons.

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the induction of general anaesthesia using propofol[172,196]. In a clinical context, the changing pattern of waves from delta, through theta, to alpha range frequency dominance corresponds to the progression from the vegetative state or coma, to the minimally conscious state, to behavioural consciousness[197].

Nevertheless, distinguishing conscious from unconscious participants on the basis of global EEG patterns is not always reliable. For example, a widespread alpha rhythm has been recorded in patients in a severe post-anoxic coma[198]. Conversely, EEG can show persistent large slow waves in conscious participants during some rare instances of status epilepticus[199,200] (FIG. 3h). In such patients, slow wave activity may only involve some cortical areas. Intracranial electrical recordings show that, during sleep, some cortical areas may become activated while the scalp EEG is still dominated by slow waves[201]. Source modelling reveals that such locally activated EEG over a parieto-occipital region just before awakening from slow wave sleep occurs when participants report visual dreams, even though other cortical areas display low-frequency activity at this time[44]. Thus, it may be that local, rather than global, EEG activation is important for consciousness, especially over the posterior cortical hot zone.

Neural differentiation and integration

EEG remains a fundamental clinical tool for discriminating between conscious and unconscious patients[202]. Such an evaluation typically includes qualitative features of the spontaneous EEG (such as activation) and how EEG activity responds to perturbations (such as when patients open their eyes) that capture the degree of spatiotemporal differentiation of electrical activity in the brain[203]. This classic notion of the activated EEG, often overlooked in the search for the NCC, suggests that a large repertoire of neural activity patterns is important for consciousness[1]; recent studies support this view. For example, in rats, the number of unique fMRI blood-oxygen-level-dependent (BOLD) patterns increases with the recovery of consciousness from desflurane anaesthesia[204]. The repertoire of fMRI functional connectivity configurations is greater during wakefulness than during propofol anaesthesia in monkeys[205]. Likewise, electrocortical dynamics become more stable on loss of consciousness, regardless of anaesthetic-specific effects on activity[206]. In humans, measuring the entropy of brain activity is used to assess the depth of anaesthesia[207] and provides a useful prognostic index of recovery of consciousness in patients in a vegetative state[208,209].

It is generally recognized that consciousness also requires an integrated neural substrate[1]. In support of this notion, fMRI studies of whole-brain functional connectivity show that integration decreases and modularity increases when consciousness is lost during sleep[77,210], anaesthesia[212] and coma[211]. On a finer timescale, similar conclusions have been obtained by measuring functional connectivity using EEG, especially in the alpha and theta ranges[213–215]. However, indices of cortical integration (such as EEG coherence and Granger causality) can be increased in conditions in which consciousness is lost, such as during propofol anaesthesia[216] or generalized seizures[217], whereas EEG measures of differentiation (such as the bispectral index and spectral entropy) are only useful at the group level as a result of wide variations across participants[181,218].

The perturbational complexity index (PCI), obtained using a combination of TMS and EEG recordings[219], assesses the level of consciousness based on the notion that being conscious requires both the differentiation and integration of cortical activity[1,38]. Unlike measures of the differentiation of spontaneous activity, PCI evaluates the deterministic responses of the cortex to perturbations and is therefore largely insensitive to random processes or to locally generated patterns that are not genuinely integrated. Unlike measures of integration that rely on widespread neural synchronization, the PCI is low when neural activations are spatially extended but undifferentiated, as is often the case during anaesthesia and generalized seizures. TMS can also be used to assess consciousness when participants are disconnected from sensory inputs and motor outputs, such as during REM sleep and ketamine anaesthesia[220], because it bypasses sensory pathways. PCI is the first metric that successfully assesses the level of consciousness under different conditions (for example, during different sleep stages, during exposure to anaesthetics and in disorders of consciousness) and at the level of single participants (FIG. 4).

Conclusions

Much progress has occurred since the initiation of the modern quest for the NCC in 1990 (REF. 21). Conceptual work by both philosophers and scientists[221] has clarified the importance of investigating the neural correlates of both specific conscious contents and of consciousness as a whole[43]. The aim is to avoid confounds resulting from task performance and changes in behavioural state, so that the NCC proper can be isolated from the neural events leading up to and following on from becoming conscious[30–32,222] (FIG. 1). Based on the definition of the NCC, it is also critical to consider not only correlative evidence from neuroimaging studies and unit recordings, but also causal evidence from brain stimulation and lesion studies.

The anatomical basis of the full NCC and content-specific NCC do not comprise the wide fronto-parietal network emphasized in past studies, but are primarily localized to a more restricted temporo-parietal-occipital hot zone with additional contributions from some anterior regions (FIG. 2). At least some cortical areas in this posterior privileged zone, especially high-level sensory areas, are close to qualifying as content-specific NCC. For example, in humans, the activation of face areas correlates closely and systematically with seeing faces, whereas stimulation of the same region (on the right side) can induce or alter the perception of faces[121] and, crucially, lesions in the face areas render participants unable to recognize familiar faces (face-blindness).

Experiencing specific contents associated with activity in the posterior hot zone does not require the amplification of fronto-parietal network activity[142]. Instead, the frontal cortex is involved in allocating attention and in task execution, monitoring and reporting. This illustrates the danger of relying too heavily on correlative evidence from neuroimaging studies to identify the NCC: as a result

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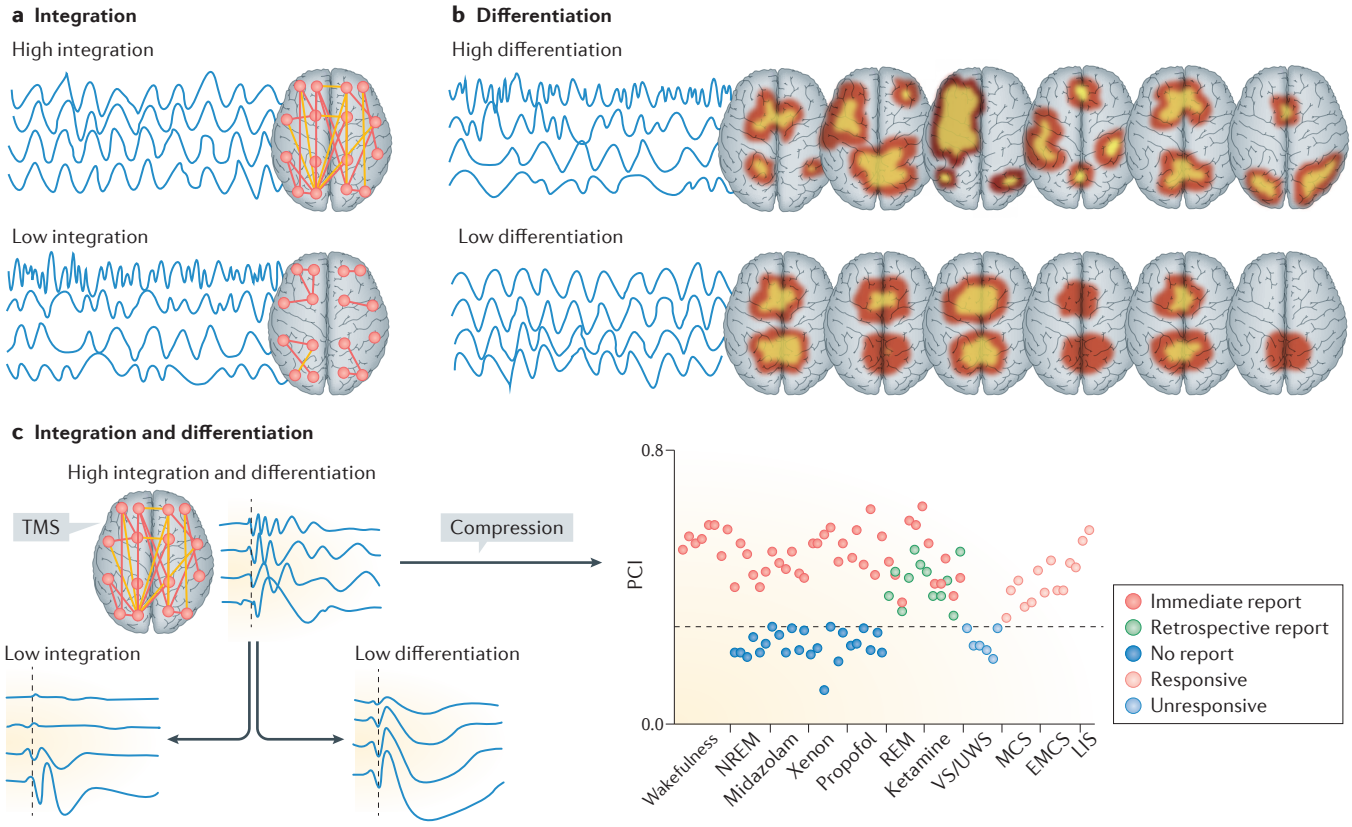

Figure 4 | **Neural differentiation and integration as neural correlates of consciousness. a** | Schematic diagram showing two idealized time series of spontaneous brain activity (electroencephalography (EEG)) and the corresponding maps of cortical connectivity. Integration, as measured by indices of functional connectivity, tends to be high when time series are highly correlated (top panel) and low when time series are not highly correlated (bottom panel). **b** | The same two time series are shown together with the corresponding maps of spatiotemporal variability in brain activity. Differentiation, here reflected in the difference between subsequent maps of cortical activity, tends to be high (top panel) when the time series are not highly correlated (maximum for random time series) and low when they are highly correlated (bottom panel). **c** | The perturbational complexity index (PCI) simultaneously quantifies integration and differentiation. Calculating the PCI involves perturbing the brain with transcranial magnetic stimulation (TMS), recording the results of EEG to detect the pattern of causal cortical interactions engaged by the TMS perturbation (integration) and compressing this pattern to calculate its spatiotemporal variability with algorithmic complexity measures (differentiation). Responses that are both integrated and differentiated are less compressible, resulting in high PCI values. By contrast, local (low integration) or stereotypical (low differentiation) EEG responses to TMS can be effectively compressed, yielding low PCI values. As shown in the right-hand panel (real data), jointly measuring integration and differentiation with PCI establishes a common measurement scale that is valid across many different conditions along the unconsciousness–consciousness spectrum and is reliable at the level of single paticipants[219,220], including: those who are awake and able to report immediately that they are conscious (red circles); participants in rapid eye movement (REM) sleep and under ketamine anaesthesia who are unresponsive, but able to report retrospectively that they were conscious (green circles); participants in non-REM (NREM) sleep or general anaesthesia (midazolam, propofol and xenon) who provide no conscious report on awakening (dark blue circles); vegetative state/unresponsive wakefulness syndrome (VS/UWS) patients (light blue circles); and responsive brain-injured patients who are in a minimally conscious state (MCS), emerged from the MCS (EMCS) or in a locked-in syndrome (LIS) (light red circles). Part **c** is adapted with permission from REF. 219, American Association for the Advancement of Science.

of the intricate connectivity of the brain, activity in the prefrontal cortex, basal ganglia and even cerebellum can co-vary systematically with conscious perception without being responsible for it.

It is possible that some prefrontal regions mediate content-specific NCC for particular types of experiences, such as feelings of reflection and introspection, valuation and affect, whereas other prefrontal regions contribute nothing directly to conscious experience, not unlike the basal ganglia and cerebellum. It remains unclear whether neurons within primary sensory areas contribute specific contents to consciousness — for example, whether neurons in V1 are responsible for perceived location in visual space.

The brainstem reticular formation, paramedian thalamus and perhaps parts of the postero-medial cortex are likely to provide the background conditions for full consciousness. By serving as activating influences and cortical hubs, they enable effective interactions among the cortical areas that directly contribute conscious content.

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Box 1 | **Hard cases and hard calls**

It is possible to search for either full or content-specific neural correlates of consciousness (derived from experiments in healthy control subjects) in some patients who are behaviourally unresponsive as a result of brain lesions. For example, one patient in a vegetative state, when told to imagine playing tennis or to mentally navigate around her house, displayed increased haemodynamic activity in specific cortical regions in a manner similar to healthy control subjects[11]. However, many patients who are behaviourally conscious fail this test[225]. Furthermore, it is not clear what should be concluded in patients whose brain is severely damaged and inactive, except for one or a few isolated cortical areas that show signs of metabolic and electrophysiological activation[226]. Can these isolated islands of functioning tissue support a limited kind of consciousness? What does it feel like to have such an island of brain activity, if, indeed, the patient in question feels anything at all?

Similar problems arise when studying parasomnias. Sleepwalkers often display complex behaviours, but it is difficult to wake them and they often do not recall any dreams[227]. Neuroimaging during sleepwalking episodes reveals mixed signs of local sleep and wake states[228,229]. It is also difficult to assess consciousness during epileptic seizures (for example, partial seizures with automatisms and many instances of generalized seizures, such as absences) that are not always associated with a complete loss of consciousness[230]. Likewise, patients who are anaesthetized with dissociative anaesthetics such as ketamine, which have complex effects on the brain, are fully unresponsive, but, on awakening, report long, vivid dreams from the period of anaesthesia[220]. The assessment of consciousness in patients who remain immobile — for example, owing to extreme Parkinson-like states[231] or catatonia[232] — is even more problematic.

Similarly the extent of conscious experience in newborn infants, who have immature brains and restricted connectivity among cortical structures, is unclear[233]. A wave resembling the P300 has been reported in 6–16-month-old infants, although it is weaker, more variable and later than in adults[234]. However, as mentioned in the main text, a P3b-like wave can be present in patients who are comatose and hypothermic[187], but is not present in some conscious adults[181]. Thus resorting to the P3b wave to infer the presence of consciousness in infants, and its absence in newborn and pre-term infants, is problematic.

Assessing consciousness is even more problematic in non-human animals. It is likely that mammals that share many behavioural traits with humans, and have brains organized in a similar manner to human brains, are conscious. Indeed, in laboratory settings used to study the neural correlates of consciousness in humans, macaque monkeys act very similarly to humans, including signalling when they do not see a stimulus under blindsight conditions[18]. However, in species that are more distant from humans in evolutionary and neural terms, the question of whether they are conscious becomes more difficult. Birds, fish, cephalopods and insects are capable of sophisticated, learned, non-stereotyped behaviours that are typically associated with consciousness in humans[235–237]. Although their nervous systems may be smaller and organized differently, they are still very complex. For example, bee brains contain close to 1 million nerve cells packed up to ten times more densely than in the neocortex and assembled in non-linear feedback circuits[238]. Yet humans can perform complex behaviours in a seemingly non-conscious manner, such as detecting the meaning of words, doing simple arithmetic or typing rapidly[239–244], and highly complex structures in the human brain, such as the cerebellum, do not contribute to consciousness[47–48]. Thus, when a bee chooses a branch point marked by red in a maze[245], we do not know if it does so consciously[246], as humans might do under such circumstances, or by following an unconscious programme.

In short, no single brain area seems to be necessary for being conscious, but a few areas, especially in the posterior cortical hot zone, are good candidates for both full and content-specific NCC.

Regarding the neurophysiological markers of consciousness, hopes that gamma activity or synchrony, or the ERP P3b, could be signatures of consciousness have proved illusory (FIG. 3). An activated EEG, one of the oldest electrophysiological indices of consciousness, is a better marker of consciousness than these measures, as long as it is taken into account that it is the local rather than global EEG activation that is important, especially within the posterior cortical hot zone.

Novel indices of consciousness assess the differentiation of neural activity (the availability of a large repertoire of different firing patterns) and its integration (whether a neural system behaves as a single entity). The most promising of these is the simultaneous causal assessment of differentiation and integration — for example, the complexity of EEG responses following a TMS pulse[219,220] (FIG. 4).

The sheer number of causal interactions in the brain[1], together with the fleeting nature of many experiences[223], pose challenges to even sophisticated experimental approaches to the NCC. The NCC by themselves can provide little information about consciousness in patients with severe brain damage, infants, fetuses, non-human species or intelligent machines (BOX 1). Further progress in this field will require, in addition to empirical work, testable theories that address in a principled manner what consciousness is and what is required of its physical substrate[224].

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Acknowledgements
The authors thank laboratory members and colleagues for their various contributions to the work presented here. This work was supported by the Templeton World Charity Foundation, the McDonnell Foundation and the Distinguished Chair in Consciousness Science (University of Wisconsin) to G.T. and by the EU project 686764 'Luminous' to M.M. C.K. thanks the Allen Institute for Brain Science founders, P. G. Allen and J. Allen, for their vision, encouragement and support.

Competing interests statement
The authors declare no competing interests.

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ERRATUM

Neural correlates of consciousness: progress and problems

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Nature Reviews Neuroscience **17**, 307–321 (2016)

The traces in panel **e** of Figure 3 were incorrectly colour coded. The colour coding has been corrected in the online version of the article.

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