

Opinion piece



**Cite this article:** Fazekas P. 2021

Hallucinations as intensified forms of mind-wandering. *Phil. Trans. R. Soc. B* **376**: 20190700.

<http://dx.doi.org/10.1098/rstb.2019.0700>

Accepted: 1 September 2020

One contribution of 16 to a theme issue ‘Offline perception: voluntary and spontaneous perceptual experiences without matching external stimulation’.

**Subject Areas:**

cognition, neuroscience

**Keywords:**

hallucinations, mental imagery, imagery vividness, mind-wandering

**Author for correspondence:**

Peter Fazekas

e-mail: [fazekas.peter@gmail.com](mailto:fazekas.peter@gmail.com)

# Hallucinations as intensified forms of mind-wandering

Peter Fazekas<sup>1,2</sup>

<sup>1</sup>Centre for Philosophical Psychology, Universiteit Antwerpen, Antwerpen, Belgium

<sup>2</sup>Cognitive Neuroscience Research Unit, Centre of Functionally Integrative Neuroscience, Aarhus Universitet, Aarhus, Denmark

PF, 0000-0002-2922-1865

This paper argues for a novel way of thinking about hallucinations as intensified forms of mind-wandering. Starting from the observation that hallucinations are associated with hyperactive sensory areas underlying the content of hallucinatory experiences and a confusion with regard to the reality of the source of these experiences, the paper first reviews the different factors that might contribute to the impairment of reality monitoring. The paper then focuses on the sensory characteristics determining the vividness of an experience, reviews their relationship to the sensory hyperactivity observed in hallucinations, and investigates under what circumstances they can drive reality judgements. Finally, based on these considerations, the paper presents its main proposal according to which hallucinations are intensified forms of mind-wandering that are amplified along their sensory characteristics, and sketches a possible model of what factors might determine if an internally and involuntarily generated perceptual representation is experienced as a hallucination or as an instance of mind-wandering.

This article is part of the theme issue ‘Offline perception: voluntary and spontaneous perceptual experiences without matching external stimulation’.

## 1. Introduction

Hallucinations and dreams have a lot of similarities, which inspired many to theorise about their possible relationship [1–3]. For example, it has been argued that hallucinations might best be understood as rapid eye movement (REM) dream intrusions [4–6], or in the light of an implementation of Hobson’s activation-input-modulation theory of dreaming [7,8]. Conversely, it has also been suggested that dreams should be viewed as hallucinations [3,9,10]. On the other hand, with the advance of the neuro-cognitive theory of dreaming and the so-called continuity hypothesis which asserts that dream cognition is similar to waking cognition, exploring the relationship between mind-wandering and dreaming has recently got into the forefront of scientific investigations. In this framework, dreaming is interpreted as intensified mind-wandering [11–13]. Interestingly, these two strands of research have been motivated by opposing theoretical frameworks. This might be the reason why the third link between these three phenomena—i.e. the relationship between hallucinations and mind-wandering—has never really been in the centre of interest (for a very recent exception see [14]).

The major goal of the present paper is to explore this so-far neglected relationship. Important similarities will be uncovered and on the basis of them a novel hypothesis will be proposed, according to which hallucinations are intensified forms of mind-wandering. This view fits well with the idea that mind-wandering can be seen as a psychological baseline [15], and points towards a possible general framework that unifies mind-wandering, hallucinations and dreaming in terms of the amplification of the sensory characteristics of episodes of mind-wandering occurring in different general states of consciousness.

The role imagery vividness plays in hallucinations is in the centre of this paper. Hallucinations are associated both with posterior hyperactivity, i.e. spontaneous activity in sensory processing areas (not triggered by the bottom-up processing of a stimulus), and prefrontal hypoactivity, i.e. deactivation of the

medial aspect of the anterior prefrontal cortex (PFC) [16–19]. As the functioning of the medial PFC has been linked to reality monitoring [19], according to a popular interpretation, the spontaneous activity in the primary and associative sensory areas provides the contents of hallucinatory experiences [16], which are then incorrectly identified as not being internally generated owing to the impairment of the reality monitoring system [17,19,20].

The activity in the content-specific posterior regions has also been implicated as a key determinant of the vividness of the accompanying conscious experiences [21,22]. This raises the question of how the vividness of a self-generated experience is related to the judgement of the reality monitoring system. Some suggest that these two features form independent dimensions of the phenomenological space that characterize conscious experiences [23]. Others claim that the vividness of experiences is determined by the judgement of the reality monitoring system [24]. This paper argues for the third option: that vividness influences whether experiences are assessed as being externally triggered or internally generated.

The paper will proceed as follows. First, it will review existing theoretical approaches and empirical findings regarding how posterior hyperactivity and prefrontal hypoactivity is related to hallucinations, the role of the reality monitoring system, and the sensory and cognitive cues that influence reality judgements (§2). Then, the paper will focus on the sensory characteristics that are typically experienced as factors of vividness and their relationship to the sensory hyperactivity observed in hallucinations (§3), and review evidence showing that in certain cases with intact reality monitoring these sensory characteristics are the major determining factors that drive reality judgements (§4). Finally, the paper will propose a hypothetical relationship between hallucinations and mind-wandering, and sketch a possible mechanism of what might underly whether an involuntarily self-generated perceptual representation is experienced as a hallucination or as an instance of mind-wandering (§5).

## 2. Background

Hallucinations are sensory experiences that occur involuntarily in the absence of a matching external stimulation of the relevant sensory receptors, yet are perceived as if they were experiences of external objects or events [25–27]. In contemporary literature, there is an abundance of mechanistic models trying to account for this phenomenon, many of which focus on spontaneous sensory hyperactivity and impaired reality monitoring as the major source of hallucinations [28].

### (a) Spontaneous sensory hyperactivity

In a now classical paper, Allen *et al.* [16] reviewed articles reporting data on the neural substrates of hallucinations using positron emission tomography, magnetic resonance imaging (MRI) and diffusion tensor imaging. A central element of their findings was the over activation of the secondary (and occasionally the primary) sensory cortices, which Allen *et al.* identified as the source of the contents of perceptual experiences occurring in the absence of sensory stimuli. More recently, Zmigrod *et al.* [18] have shown in an activation likelihood estimation meta-analysis of functional neuroimaging data, that sensory processing areas are active during hallucinations. In the case of auditory hallucinations, significant brain activity was

observed in the bilateral somatosensory cortex, bilateral insula, superior temporal gyrus, Broca's area and its right hemisphere homologue and the secondary auditory cortex (Wernicke's area). Visual hallucinations were primarily associated with significant activation in the bilateral secondary and associate visual cortices (extrastriate visual areas around the ventral lingual and fusiform gyri, and in the more dorsal cuneus and precuneus regions).

Accounting for this hyperactivity in the sensory cortices is in the focus of many models of hallucinations. According to the *cortical irritation model*, hallucinations result from the intrinsic overactivity of the brain areas that contain specific image memories or representations [29,30]. The *cortical hyperexcitability model* claims that, in the case of Parkinson's disease, a lack of sensory input owing to the deafferentation of certain sensory structures leads to chronic hyperexcitability, which results in an increase in spontaneous activity [31,32]. The *dream imagery intrusion model* [4–6] and the *activation, input, modulation model* [8] assert that intrusive REM dream imagery causes the hyperactivity of the sensory areas. According to the *unbalanced top-down activation model*, tonically hyperactive top-down attentional excitations activate images [33]. Finally, according to the *dysfunction of the attentional control networks model*, unattenuated top-down activation originating from the default mode network might also be the reason of the hyperactivity of the primary and associative sensory areas [34,35].

### (b) Impaired reality monitoring

Whatever mechanism might lead to the internal generation of certain perceptual representations, in the hallucinating brain these are interpreted as being triggered by an external source.

The *source monitoring framework* proposes that memories do not contain propositional labels or tags that directly specify their source [36], but instead, the origin of memories is inferred, for example, from characteristic features of mental representations and their formation [37,38]. If the source monitoring judgement is concerned with the internal or external origin of the memory (that is, whether an event was imagined or really did occur), the attribution process is referred to as *reality monitoring* [39]. Reality monitoring is defined as a mnemonic ability, but the cognitive operations involved in monitoring the origin of retrieved information might overlap with those that monitor the origin of real-time information, i.e. are responsible for the reality testing of current perceptual experience [37,40]. Indeed, *source monitoring deficit models* of hallucinations [41] argue that it is an impairment in reality monitoring and a bias towards external sources in reality judgements that is behind rendering internally generated representations appear as if they were triggered by external sources.

Neural level findings supporting this view identify the medial aspect of the anterior PFC as a key component of brain networks that are engaged when distinguishing internally and externally generated information. It exhibits differential activity during the retrieval of such internal versus external information, and its disruption is associated with misattributions of reality, confusing internally generated information with events taking place in the outside world [19,42–45]. The idea that hypoactivity in this region and thus reality monitoring dysfunction is what underlies hallucinations have been reinforced by many findings [19]. For instance, individuals with schizophrenia are impaired on seen versus imagined

memory tasks that require increased anterior PFC activity in healthy volunteers [46,47]. Relatedly, the anterior PFC region that is activated in healthy volunteers during reality monitoring performance is among the areas that are dysfunctional in patients with schizophrenia [44,48,49].

### (c) Cues driving the judgement of the reality monitoring system

The original source monitoring framework [37–39] proposes that the types of attributes that are particularly important for reality monitoring include information about the sensory characteristics of the stimulus presentation, the context of the stimulus, the semantic content and the cognitive processing engaged. Sensory characteristics of the stimulus include all sensory information from sense modality, through qualities like vividness and detailedness, to attributes like whether a piece of auditory information was told by a male or female voice, etc. Contextual information includes similar sensory characteristics about the details of the spatial and temporal surrounding of the stimulus. Semantic information includes embeddedness in supporting memories, knowledge, beliefs and affect. Finally, information about cognitive processing includes the type of operations (e.g. voluntarily initiated and controlled imagery) and the signatures of cognitive efforts made.

This idea that cognitive operations involved in the generation of sensory representations leave a trace in real time about the origin of these representations that reality monitoring can be sensitive to implies that self generation may produce a variety of cues that can then straightforwardly inform and influence the reality judgement processes. According to the *corollary discharge dysfunction model* [50], a candidate of such a cue is the efference copy or corollary discharge mechanism that predicts the sensory consequence of motor commands. The model extends this framework to cognitive operations in general, and claims that it is a comparison of a top-down efference copy with the bottom-up sensory input that provides information about the self-generated nature of a motor action or cognitive operation. Interestingly, the corollary discharge dysfunction model predicts that hallucinations might arise even without an impairment of the general reality monitoring system, when either the generation of corollary discharges, or the comparator process is dysfunctional [51]. Findings of self-recognition deficits in patients with schizophrenia relating to motor action [52] and corollary discharge dysfunction in schizophrenia [53–56] support this model.

However, as direct evidence for a specific comparator model relating to inner speech or auditory imagery is lacking and theoretical considerations have been raised to dispute that cognitive operations in general have the same physiological consequences as motor action [57,58], the viability of the corollary discharge dysfunction model of hallucinations is yet to be determined.

Shifting the focus from cues about cognitive operations to certain sensory characteristics of self-generated representations, the rest of the paper will explore whether features of sensory characteristics like vividness and detailedness are able to drive reality judgements similarly to how dysfunctions of the efference copy mechanism might even in the case of an intact reality monitoring system.

## 3. Imagery vividness

The claim that the contents of hallucinatory experiences are subserved by spontaneous neural activity in sensory and associative areas [16,59] is indicated by findings associating auditory hallucinations in healthy individuals during periods of silence with the random activity of speech-sensitive auditory processing areas [60], and is also well supported by findings from many other strands of research [61,62]. These regions process incoming information and form content-specific neural representations in the case of stimulus-triggered perception [63–65], are also responsible for maintaining information in working memory after stimulus offset [66–71], and serve as the representational underpinnings of mental imagery [72–81]. The activity of these sensory and associative areas has also been found to underly the perceptual contents of dreaming [62,82] and mind-wandering [15,22].

To see what features of this posterior activity the phenomenological notions of vividness and detailedness map onto, this section provides a brief overview of a recent analysis of imagery vividness and its neural underpinnings [22] (for an earlier attempt see [38]).

### (a) Factors of vividness

Vividness has been routinely used to compare and contrast different varieties of conscious experiences like those occurring in dreams and wakefulness [83–89], or during mental imagery and stimulus-triggered perception [80,81,90]. However, the notion of vividness has been claimed to be notoriously problematic as it is only intuitively defined with the use of other concepts like clarity, detail, brightness, intensity, etc., which themselves are then left unexplained [91–95]. Even whether what standard empirical tools, like the vividness of visual imagery questionnaire (VVIQ) [96], measure is a single feature of experience or rather a construct with more than one components is debated [94].

To settle these issues it has recently been argued that because tools like the VVIQ ask subjects to measure their imagery against ‘normal vision’ (an imagery experience is maximally vivid on VVIQ if it is ‘as vivid as normal vision’ [96]), the level of imagery vividness (as measured by e.g. VVIQ) carries information about how *degraded* or *reduced in quality* an experience is [22].

The *quality* of an experience in this context refers to the way the content-elements entering consciousness appear in a conscious experience. According to a recently proposed theoretical model, consciousness can be reduced in quality along many different dimensions [97–99]. Importantly from our present perspective, two of these major dimensions are *subjective intensity* (determined by how much the content-element in question stands out from the perceived background; with more intense content-elements having more strength [100] or liveliness [94]), and *subjective specificity* (determined by how distinguishable a content-element is from other content-elements; with less specific experience of a content-element being more generic or vague). These major dimensions themselves are determined by many modality-specific factors or sub-dimensions that can be modulated either individually or in any combination. For example, the subjective intensity in the visual modality increases with increasing contrast, saturation or brightness. Similarly, subjective specificity in the visual modality increases with increasing precision (e.g. a

colour appearing as having a more specific shade), or by being sharper and less blurry, or by being rich in detail (cf. how a well-exposed photo contains more detail than an over- or under-exposed one).

### (b) The neural correlates of vividness

The factors of subjective intensity in the visual case—contrast, saturation and brightness—are neurally encoded in a similar fashion, by the strength of the response functions of populations of neurons that represent corresponding features like orientation [101], hue [102] and object surfaces [103], respectively. Consequently, variance in subjective intensity along these sub-dimensions manifests itself as modulations in the blood-oxygen-level-dependent signal and in high-frequency electroencephalogram activity [104–106] over content-specific regions.

The neural underpinnings of the different factors of subjective specificity in the visual case are a bit more diverse. While the precision component has been associated with the sharpness (inverse variance) of the response function of the neural population that encodes the feature in question [107,108], apparent blur and detailedness have been associated with neural sensitivity to higher spatial frequencies [109–112] and, especially in the case of stimulus-independent percepts, the level of recruitment of early visual representations [113–115]. Accordingly, increased subjective specificity might be indicated by a broader involvement of early visual processing in the neural correlate of self-generated experiences.

This analysis makes it possible to scrutinize recent findings and identify the distinct neural signatures of subjective intensity and subjective specificity. For instance, it was found that variations in the moment-to-moment experienced vividness of visual imagery correlated with the intensity of the neural activity (strength of simultaneously recorded functional MRI signal) in the primary and associative visual areas [80,90]. As we have seen, these correlations suggest that in these mental imagery tasks experienced vividness changes along the subjective intensity dimension. It has also been reported that VVIQ measures of vividness correlate with the overlap between the activity in visual areas during mental imagery and perception [76,80,116], and also with the strength of top-down recruitment of early visual areas [81,117]. These findings suggest that the experienced vividness of mental imagery changes along the subjective specificity (blurriness/detailedness) dimension as well.

Similar reports of an overlap in the activity of auditory association areas during perception and imagery and of correlations between the vividness of auditory imagery and the level of activity in auditory areas [118–120] and the representational specificity of heard sounds [121], indicate that the links proposed between experienced vividness and brain activity can be generalized to at least the auditory case as well.

## 4. Imagery vividness and hallucinations

The important lesson from the previous section is that standard measures of imagery vividness carry information about those sensory characteristics—vividness and detailedness in the original source monitoring framework [37–39]—that are the modality- and content-independent sensory features of conscious experiences (i.e. characterize all experiences regardless of their modality and particular content [100]) that the reality monitoring system is sensitive to.

When an experience is maximally vivid according to these measures, then it does not appear to be degraded or reduced in quality at all. It seems to present its content as ‘normal perception’ would that is, as if it was triggered by external stimuli. Highly elevated activity in spontaneously hyperactive sensory areas could thus produce perceptual experiences that, despite being internally generated, are as intense and as specific as the experiences that would arise from sensory engagement with corresponding external stimuli—and thus could provide strong signals for the reality monitoring system, driving it to judge them as being brought about by external sources. This section explores the prospects of this hypothesis, focusing on cases where the reality monitoring system is presumably fully functional.

### (a) Hallucinations with intact reality monitoring

Hallucinations occur in individuals with no clinical diagnosis as well. For instance, around 6–7% of the general population report auditory verbal hallucinations [122]. So-called *continuum models of psychosis* [123–125] interpret this as a sign that hallucinatory experiences are distributed throughout the general population existing on a continuum ranging from very mild expressions to severe symptoms [58].

While an early study reported that participants who were prone to experience hallucinations were more likely than others to misidentify self-generated words as real auditory experiences [126], two more recent studies found no evidence of an impairment in source or reality monitoring in hallucination-prone individuals [58,127]. These findings indicate a possible double dissociation between reality monitoring dysfunction and hallucinations in the non-clinical population: on the one hand, in hallucination-prone individuals with intact reality monitoring hallucinations can nevertheless occur, while, on the other hand, some healthy individuals with reality monitoring problems (e.g. bilateral paracingulate sulcus absence [128]) do not experience hallucinations [19,122].

Although this issue is not settled yet (see [129] for a recent report that in the case of visual hallucinations hallucination-prone individuals do show similar reality monitoring bias than the clinical population), current evidence does not provide unambiguous support for the claim that hallucinations experienced by non-clinical individuals are underlain by reality monitoring impairment and associated anterior PFC dysfunction.

### (b) Elevated imagery vividness in hallucination-prone individuals

While an impairment of reality monitoring is not always found, the spontaneous elevated activity in the sensory cortex that is a characteristic sign of clinical hallucinations is present in hallucination-prone individuals. For instance, common hallucination-related activity in auditory processing areas (e.g. superior temporal gyrus) is reported in non-clinical groups as well [59,60,130]. Similarly, a correlation has been found in non-clinical individuals between auditory hallucination proneness scores and superior temporal gyrus activity when imagining voices that are subsequently misidentified as being heard [131].

As we have seen, this increased activity in content-specific sensory areas corresponds to an increased vividness of the accompanying conscious experience [22], and thus ties these



findings to the classical idea [132] that the vividness of hallucinatory experiences has a major role in the false attribution of the experienced properties to external sources. More precisely, the findings that hyperactive sensory areas alone—without reality monitoring impairments—can lead to hallucinations implicate that the sensory characteristics corresponding to the elevated sensory activity are able to drive reality judgements. The elevated sensory activity most probably achieves this effect via ‘misinforming’ the reality monitoring system by providing signals that are as strong as usually can only be triggered by actually present external stimuli.

This line of thought has recently motivated a *multi-factor account of hallucinations* according to which there are differences in the mechanisms that underly clinical and non-clinical hallucinations. In the case of non-clinical individuals, hallucinations might arise when certain characteristics (e.g. intensity/vividness) of internally generated sensory activity are unusual in such a way that an otherwise intact reality monitoring system fails to recognize them as being self-generated. In the case of clinical individuals, an additional reality monitoring impairment (underlain by anterior medial PFC dysfunction) might also contribute to the severity of the symptoms [19,58,122,129].

The role attributed by this model to vividness is in line with the hypothesis that hallucinators have an imagery system that produces vivid and detailed images [133], with the associations that have been found between hallucinations and increased imagery vividness in the case of schizophrenia [134–138], Parkinson’s disease [139] and Alzheimer’s disease [140], and with the findings that vivid imagery can lead to false remembering [141–143].

### (c) Hallucinations and the general vividness and frequency of voluntary mental imagery

Despite the popularity of the hypothesis that there is a link between vivid imagery and hallucinations, it is in tension with the findings of a number of studies that report no signs of increased imagery vividness in clinical or non-clinical hallucinators, and argue that vivid imagery *per se* does not account for hallucinatory experiences [144]. For instance, the tendency of healthy individuals to have hallucinatory experiences was found to be explained better by non-specific response bias than by increased imagery vividness, suggesting that mental imagery has an only indirect role in non-clinical hallucinations [145]. Another study focusing on schizophrenia patients found increased vividness of mental imagery, however, the correlation between the hallucination and imagery scales themselves was very low, indicating that vivid imagery is independent of hallucinations and might be a trait of schizophrenia [136]. This conclusion has been reinforced by another study showing that although mental imagery vividness in a schizophrenia patient, their first-degree relatives and also in high-schizotypy controls was higher than in low-schizotypy controls, it was independent of the predisposition towards hallucinations [146]. A further study comparing non-clinical participants with higher and lower predisposition to visual hallucinations found no difference in VVIQ-measured [96] imagery vividness [129]. However, participants with high predisposition reported using visual imagery more frequently in their everyday life, which led the authors to conclude that it is the greater use of visual imagery that might lead to the greater misattribution of internal images to external events

[129]. Similarly, the existence of a link between imagery vividness and hallucination proneness can also be questioned on the basis of the observation that people with hyperphantasia do not routinely hallucinate [147].

To start reconciling these findings with the hypothesis that unusually vivid experiences can be responsible for driving mistaken reality judgements, consider the role that hypersensitive sensory areas play during mental imagery and hallucinations. Hypersensitive sensory areas can be activated more easily and respond with stronger signals. Thus they can be recruited more easily and generate more intense activity patterns when triggered by voluntary mental imagery, and also when triggered spontaneously. When triggered voluntarily, the rate of occurrence and strength of the responses correspond to the general frequency and vividness of mental imagery (respectively). When especially strong responses are triggered spontaneously, the rate of occurrence and strength of these responses correspond to the frequency and vividness of hallucinatory experiences.

Next, note that while the vividness and/or the frequency of mental imagery will be high for everyone with the hypersensitive sensory cortex, not all of these individuals will necessarily report a high frequency of hallucinations as the occurrence of spontaneous (involuntary) triggering events might very well be independent of both voluntary triggering and the sensitivity of the sensory areas triggered. According to a recent finding, for instance, people with aphantasia, who have problems with *voluntary* mental imagery, report the same amount of *involuntary* mind-wandering and dream experiences as the age-matched control group [148]. The result is a picture which is in line with the findings: the increased vividness/frequency of imagery is determined by sensory cortex sensitivity, whereas hallucination proneness is independent, as it is determined by the further factor of the frequency of suitable *spontaneous* triggering. Moreover, it is also in line with the original hypothesis: when spontaneously triggered, hypersensitive sensory areas respond more often with unusually intense activation, leading to hallucinatory experiences.

Although according to this model hallucinations are linked to increased sensory activity, and increased sensory activity is associated with increased sensitivity of these areas, increased sensitivity alone does not account for the frequency of spontaneous activations. That is, what the findings introduced in this section seem to be incompatible with is not the link between vivid imagery and hallucinations, but with the claim that the sensitivity of the sensory areas is the only factor that determines the frequency of hallucinatory experiences.

## 5. Hallucinations and mind-wandering

Now that we have seen the importance of spontaneous triggering events in the background of hallucinatory experiences, we are in a position to fully acknowledge the inappropriateness of using *voluntary* mental imagery as a basis of comparison when studying hallucinations. Current studies use standard imagery questionnaires to evaluate if the occurrence of hallucinations correlates with the frequency or general vividness of voluntary mental imagery experiences. Hallucinations, however, are characteristically *involuntary*: they occur spontaneously in the sense that they are triggered by top-down or lateral cues that are not under deliberate cognitive control [149,150]. So a more appropriate basis for comparison would

be a phenomenon that shares this crucial feature of spontaneity with hallucinations.

### (a) Hallucinations as intensified mind-wandering: a proposal

There is a form of involuntary mental imagery (self-generated, or ‘phantom’ [149] perceptual experience) that is so common that it occupies almost half of our wakeful mental life [151], and whose main defining feature is its spontaneity [152]. It is *mind-wandering*. When the mind wanders, it is engaged in a series of trains of thought and often imagery as well [153,154] that appear and unfold in a spontaneous and relatively unconstrained manner with widely varying content-elements lacking any obvious connection to each other, to stimuli in the environment or to ongoing task requirements [152,155,156].

Mind-wandering correlates with default mode network activity [157,158] and is often thought of as a psychological baseline, a kind of functioning that the mind returns to whenever it is otherwise unoccupied [15]. The content of the involuntary imagery occurring during mind-wandering is subserved by the same content-specific sensory areas that play a similar role in voluntary mental imagery and in hallucinations as well [11,159,160], and the intensity of the activity of these areas has been associated with the vividness of the accompanying conscious experience in the case of mind-wandering too [22]. Higher imagery vividness leads to higher meta-awareness (consciously registering the content of and the fact that one is having an episode of mind-wandering) [161], and thus individuals with more vivid mind-wandering experiences better remember that their mind often wanders, which results in an increased number of self-reported mind-wandering episodes [15,22]. That is, individuals with more sensitive sensory areas (which, as we have seen it, gives rise to more vivid self-generated experiences) report more frequent mind-wandering.

Here, we propose that the similarities in the spontaneous, transient and relatively unconstrained nature of hallucinations and episodes of mind-wandering are owing to similarities in the underlying mechanism, namely that similar content-specific areas get triggered by similar spontaneous triggering events (resting state default mode network activity, involuntary lateral and/or top-down cues) in both cases. When these sensory areas are hypersensitive then the spontaneous triggering events that normally give rise to episodes of mind-wandering can occasionally elicit so intense responses that their strong signals trick the (otherwise well-functioning) reality monitoring system, which then fails to register them as being internally generated leading to conscious experiences that are seemingly externally triggered, i.e. seem to reflect real events and objects. That is, (at least non-clinical) hallucinations are intensified forms of mind-wandering—experiences that are produced by the same mechanisms that underly mind-wandering but are intensified by the hyperactivity of content-specific sensory regions.

### (b) Empirical and theoretical support

According to the proposal above, in the case of overly sensitive sensory areas cues originated from resting state networks elicit elevated activity. The intensity and range of this activity provides those sensory characteristics that the reality monitoring system is

particularly sensitive to. When the strength of these responses are below a threshold then the reality monitoring system correctly categorizes them as being internally generated and the corresponding spontaneous imagery is experienced as an instance of mind-wandering. However, when the strength of the responses are above the threshold, then even intact reality monitoring categorizes them mistakenly as being externally triggered and the corresponding imagery is experienced as a hallucination. In the case of sensitive sensory areas these responses are generally stronger and occasionally above the threshold. As stronger below-threshold responses correlate with higher reported mind-wandering frequency, while above-threshold responses correspond to hallucinations, this model predicts that hallucinations correlate with the frequency of mind-wandering.

A recent study has provided data that allow for testing this prediction. The study focused on the relationship between mind-wandering and visual hallucinations in Parkinson’s disease, and also explored the underlying brain network coupling [14]. The findings show that individuals with Parkinson’s disease who also experience visual hallucinations report significantly more episodes of mind-wandering than Parkinson’s disease patients without hallucinations, thus they provide empirical evidence of a positive correlation between mind-wandering and hallucinations. Moreover, this increased frequency of mind-wandering is associated with a greater coupling between the primary visual cortex and the dorsal default network, which suggests that the elevated sensory activity underlying both hallucinations and mind-wandering is driven by default mode network activity. As the frequency of mind-wandering in Parkinson’s disease with hallucination was not significantly different than in the control group, whereas controls reported a significantly higher number of mind-wandering episodes than Parkinson’s disease patients without hallucinations, the association between the frequency of mind-wandering and the coupling between primary visual cortex and the dorsal default network in the hallucinating phenotype of Parkinson’s disease is preserved, rather than increased, relative to controls. Because, owing to impaired sensory and attentional abilities the baseline level of sensory activity is lower in Parkinson’s disease [14], the preserved level of default mode network activity can elicit more intense responses in sensory areas, giving rise to more vivid experiences than in controls. Therefore, preserved involuntary mental imagery becomes unusually vivid, which, in accordance with the proposal of this paper, leads to those hallucinations that are characteristic of this phenotype of Parkinson’s disease. (See also the cortical hyperexcitability model [31,32].)

The proposal of this paper is also in line with previous findings reporting an association between visual hallucinations in Parkinson’s disease and increased activity and connectivity in the default mode network both in the resting state [162,163] and during hallucinatory experiences [164]. Moreover, the coupling between the default mode network and the visual cortex has been associated with the vividness (both intensity and detail) of experiences during episodes of mind-wandering in healthy individuals as well [165]. Patients with schizophrenia also report more episodes of mind-wandering and the frequency of mind-wandering has been found to correlate with the severity of the positive symptoms, including hallucinations [166].

Over and above these empirical findings, certain theoretical considerations also resonate with the proposal of this paper quite well. For instance, the so-called *resting state hypothesis* claims that audio-verbal hallucinations are underlain by

strong interactions between the default mode network and the auditory cortex, resulting in increased resting state activity in the auditory cortex triggered by an elevated resting state activity in the default mode network [167]. The *dysfunction of the attentional control networks* model of hallucinations also agrees that the sensory hyperactivity characteristic of hallucinations is triggered by default mode network activations, and claims that the underlying dynamical shifts in the interactions within and between the default mode network, the dorsal attention network and the ventral attention network [34,35,168,169] are similar to the network dynamics that characterize mind-wandering [152,170].

## 6. Conclusion

This paper has presented and argued for a multi-factor account of hallucinations. This account starts from the observation that hallucinations are associated with hyperactive sensory areas underlying the content of hallucinatory experiences and a confusion with regard to the reality of the source of these experiences. This confusion is often expressed as an impairment of reality monitoring, however, as the first part of the paper has reviewed, reality monitoring can be impaired in a lot of different ways. Reality monitoring impairment can be a general problem with the central reality monitoring system associated with medial anterior PFC, but reality judgements can also be mistaken even if the reality monitoring system is intact—when the cues that this system is sensitive to carry information that ‘tricks’ the system that otherwise functions normally.

Corollary discharge dysfunction has been briefly considered as an instance of the failure of normal functioning that might produce one kind of such misleading cues (about the cognitive source of internally generated representations). Then, the second major part of the paper considered the sensory characteristics that are typically experienced as factors of vividness (intensity, specificity) as another possible type of misleading cues, with a special focus on their relationship with the sensory hyperactivity observed in hallucinations. As far as the hyperactivity of the sensory areas is a universal characteristic of hallucinations, mistaken reality judgements

driven by misleading sensory cues can be an important factor of the phenomenon of hallucinatory experiences.

Finally, the third part of the paper proposed a hypothetical relationship between hallucinations and mind-wandering, reinterpreting hallucinations as intensified forms of mind-wandering that are amplified along their sensory characteristics. A possible underlying mechanism has also been suggested sketching how the reactivity of the sensory areas, their interaction with the default mode network and a threshold-like limit in the sensitivity of the reality monitoring system can lead from episodes of mind-wandering to hallucinations.

Of course, such a simple model can possibly account for what is perceived as real only in the absence of the other factors. Interactions between sensory and cognitive cues might play important roles that require future investigations. Exploring these interactions might help clarify issues, like, for instance, why maladaptive daydreaming, a condition with highly vivid experiences that can absorb the individual eliciting compulsive daydreaming and leading to impaired functioning [171–173], is nevertheless experienced as being internally generated. It might be the case that cognitive cues regarding the certain degree of voluntary control in the initiation and/or maintenance of episodes of mind-wandering [174] that is present in maladaptive daydreaming have a crucial part in signalling internal generation to the reality monitoring system despite the highly amplified sensory characteristics. That is, amplified sensory characteristics might drive the reality monitoring system towards mistaken judgements only when cognitive cues do not interfere with them. As largely independent factors, misleading cognitive cues, misleading sensory cues and the dysfunction of the central reality monitoring system might underly different varieties of hallucinations to different degrees and in different combinations.

**Data accessibility.** This article has no additional data.

**Competing interests.** We declare we have no competing interests.

**Funding.** This study was supported by the FWO postdoctoral fellowship 12B3918N.

**Acknowledgements.** The author wishes to thank Georgina Nemeth, Bence Nanay and an anonymous referee for their helpful comments on earlier versions of this paper.

## References

- Hobson JA. 1997 Dreaming as delirium: a mental status analysis of our nightly madness. *Semin. Neurol.* **17**, 121–128. (doi:10.1055/s-2008-1040921)
- Hobson JA, Pace-Schott EF. 2002 The cognitive neuroscience of sleep: neuronal systems, consciousness and learning. *Nat. Rev. Neurosci.* **3**, 679–693. (doi:10.1038/nrn915)
- Waters F, Blom JD, Dang-Vu TT, Cheyney AJ, Alderson-Day B, Woodruff P, Collerton D. 2016 What is the link between hallucinations, dreams, and hypnagogic-hypnopompic experiences? *Schizophr. Bull.* **42**, 1098–1109. (doi:10.1093/schbul/sbw076)
- Arnulf I, Bonnet AM, Damier P, Bejjani BP, Seilhean D, Derenne JP, Agid Y. 2000 Hallucinations, REM sleep, and Parkinson's disease. *Neurology* **55**, 281. (doi:10.1212/WNL.55.2.281)
- Manni R, Terzaghi M, Ratti P-L, Repetto A, Zangaglia R, Pacchetti C. 2011 Hallucinations and REM sleep behaviour disorder in Parkinson's disease: dream imagery intrusions and other hypotheses. *Conscious. Cogn.* **20**, 1021–1026. (doi:10.1016/j.concog.2010.10.009)
- Arnulf I. 2013 Dream imagery, rapid eye movement sleep behavior disorder, and hallucinations. *Sleep Biol. Rhythms* **11**, 15–20. (doi:10.1111/j.1479-8425.2012.00546.x)
- Hobson A, Pace-Schott EF, Stickgold R. 2000 Dreaming and the brain: toward a cognitive neuroscience of conscious states. *Behav. Brain Sci.* **23**, 793–842. (doi:10.1017/S0140525X00003976)
- Diederich NJ, Goetz CG, Stebbins GT. 2005 Repeated visual hallucinations in Parkinson's disease as disturbed external/internal perceptions: focused review and a new integrative model. *Mov. Disord.* **20**, 130–140. (doi:10.1002/mds.20308)
- Windt JM. 2010 The immersive spatiotemporal hallucination model of dreaming. *Phenomenol. Cogn. Sci.* **9**, 295–316. (doi:10.1007/s11097-010-9163-1)
- Macpherson F. 2013 The philosophy and psychology of hallucination: an introduction. In *Hallucination: philosophy and psychology* (eds F Macpherson, D Platchias), pp. 1–38. Cambridge, MA: MIT Press.
- 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. Hum. Neurosci.* **7**, 412. (doi:10.3389/fnhum.2013.00412)
- Domhoff GW, Fox KCR. 2015 Dreaming and the default network: a review, synthesis, and



- counterintuitive research proposal. *Conscious. Cogn.* **33**, 342–353. (doi:10.1016/j.concog.2015.01.019)
13. Domhoff GW. 2018 *The emergence of dreaming: mind-wandering, embodied simulation, and the default network*. New York: NY: Oxford University Press.
  14. Walpola IC, Muller AJ, Hall JM, Andrews-Hanna JR, Irish M, Lewis SJG, Shine JM, O'Callaghan C. 2020 Mind-wandering in Parkinson's disease hallucinations reflects primary visual and default network coupling. *Cortex* **125**, 233–245. (doi:10.1016/j.cortex.2019.12.023)
  15. Mason MF, Norton MI, Van Horn JD, Wegner DM, Grafton ST, Macrae CN. 2007 Wandering minds: the default network and stimulus-independent thought. *Science* **315**, 393–395. (doi:10.1126/science.1131295)
  16. Allen P, Larøi F, McGuire PK, Aleman A. 2008 The hallucinating brain: a review of structural and functional neuroimaging studies of hallucinations. *Neurosci. Biobehav. Rev.* **32**, 175–191. (doi:10.1016/j.neubiorev.2007.07.012)
  17. Waters F *et al.* 2012 Auditory hallucinations in schizophrenia and nonschizophrenia populations: a review and integrated model of cognitive mechanisms. *Schizophr. Bull.* **38**, 683–693. (doi:10.1093/schbul/sbs045)
  18. Zmigrod L, Garrison JR, Carr J, Simons JS. 2016 The neural mechanisms of hallucinations: a quantitative meta-analysis of neuroimaging studies. *Neurosci. Biobehav. Rev.* **69**, 113–123. (doi:10.1016/j.neubiorev.2016.05.037)
  19. Simons JS, Garrison JR, Johnson MK. 2017 Brain mechanisms of reality monitoring. *Trends Cogn. Sci.* **21**, 462–473. (doi:10.1016/j.tics.2017.03.012)
  20. Brébion G, Ohlsen RI, Bressan RA, David AS. 2012 Source memory errors in schizophrenia, hallucinations and negative symptoms: a synthesis of research findings. *Psychol. Med.* **42**, 2543–2554. (doi:10.1017/S003329171200075X)
  21. Fazekas P, Nemeth G, Overgaard M. 2019 White dreams are made of colours: what studying contentless dreams can teach about the neural basis of dreaming and conscious experiences. *Sleep Med. Rev.* **43**, 84–91. (doi:10.1016/j.smrv.2018.10.005)
  22. Fazekas P, Nemeth G, Overgaard M. 2020 Perceptual representations and the vividness of stimulus-triggered and stimulus-independent experiences. *Perspect. Psychol. Sci.* **15**, 1200–1213.
  23. Ffytche DH. 2013 The hallucinating brain: neurobiological insights into the nature of hallucinations. In *Hallucination: philosophy and psychology* (eds F Macpherson, D Platchias), pp. 45–64. Cambridge, MA: MIT Press.
  24. Lau H. 2019 Consciousness, metacognition, & perceptual reality monitoring. *PsyArXiv*. (doi:10.31234/osf.io/ckbyf)
  25. Epstein J, Isenberg N, Stern E, Silbersweig D. 2002 Toward the neuroanatomical understanding of psychiatric illness: the role of functional imaging. In *Defining psychopathology in the 21st century: DSM-V and beyond* (eds JE Helzer, JJ Hudziak), pp. 57–69. Washington, DC: American Psychiatric Press.
  26. Dodgson G, Gordon S. 2009 Avoiding false negatives: are some auditory hallucinations an evolved design flaw? *Behav. Cogn. Psychother.* **37**, 325–334. (doi:10.1017/S1352465809005244)
  27. Wilkinson S. 2014 Accounting for the phenomenology and varieties of auditory verbal hallucination within a predictive processing framework. *Conscious. Cogn.* **30**, 142–155. (doi:10.1016/j.concog.2014.09.002)
  28. O'Brien J *et al.* 2020 Visual hallucinations in neurological and ophthalmological disease: pathophysiology and management. *J. Neurol. Neurosurg. Psychiatry* **91**, 512–519. (doi:10.1136/jnnp-2019-322702)
  29. Levine DN, Finklestein S. 1982 Delayed psychosis after right temporoparietal stroke or trauma. *Neurology* **32**, 267. (doi:10.1212/WNL.32.3.267)
  30. Noda S, Mizoguchi M, Yamamoto A. 1993 Thalamic experiential hallucinosis. *J. Neurol. Neurosurg. Psychiatry* **56**, 1224–1226. (doi:10.1136/jnnp.56.11.1224)
  31. Burke W. 2002 The neural basis of Charles Bonnet hallucinations: a hypothesis. *J. Neurol. Neurosurg. Psychiatry* **73**, 535–541. (doi:10.1136/jnnp.73.5.535)
  32. Painter DR, Dwyer MF, Kamke MR, Mattingley JB. 2018 Stimulus-driven cortical hyperexcitability in individuals with Charles Bonnet hallucinations. *Curr. Biol.* **28**, 3475–3480. (doi:10.1016/j.cub.2018.08.058)
  33. Grossberg S. 2000 How hallucinations may arise from brain mechanisms of learning, attention, and volition. *J. Int. Neuropsychol. Soc.* **6**, 583–592.
  34. Shine JM, Halliday GM, Naismith SL, Lewis SJG. 2011 Visual misperceptions and hallucinations in Parkinson's disease: dysfunction of attentional control networks? *Mov. Disord.* **26**, 2154–2159. (doi:10.1002/mds.23896)
  35. Shine JM, O'Callaghan C, Halliday GM, Lewis SJG. 2014 Tricks of the mind: visual hallucinations as disorders of attention. *Prog. Neurobiol.* **116**, 58–65. (doi:10.1016/j.pneurobio.2014.01.004)
  36. Kundendorf RG. 1986 Hypnotic hallucinations as 'unmonitored' images: an empirical study. *Imagin. Cogn. Pers.* **5**, 255–270. (doi:10.2190/KNE8-QT3L-RDB0-6KWK)
  37. Johnson MK, Hashtroudi S, Lindsay DS. 1993 Source monitoring. *Psychol. Bull.* **114**, 3–28. (doi:10.1037/0033-2909.114.1.3)
  38. Mitchell KJ, Johnson MK. 2009 Source monitoring 15 years later: what have we learned from fMRI about the neural mechanisms of source memory? *Psychol. Bull.* **135**, 638–677. (doi:10.1037/a0015849)
  39. Johnson MK, Raye CL. 1981 Reality monitoring. *Psychol. Rev.* **88**, 67–85. (doi:10.1037/0033-295X.88.1.67)
  40. Garrison JR, Bond R, Gibbard E, Johnson MK, Simons JS. 2017 Monitoring what is real: the effects of modality and action on accuracy and type of reality monitoring error. *Cortex* **87**, 108–117. (doi:10.1016/j.cortex.2016.06.018)
  41. Barnes J, Boubert L, Harris J, Lee A, David AS. 2003 Reality monitoring and visual hallucinations in Parkinson's disease. *Neuropsychologia* **41**, 565–574. (doi:10.1016/S0028-3932(02)00182-3)
  42. Dobbins IG, Wagner AD. 2005 Domain-general and domain-sensitive prefrontal mechanisms for recollecting events and detecting novelty. *Cereb. Cortex* **15**, 1768–1778. (doi:10.1093/cercor/bhi054)
  43. Kensinger EA, Schacter DL. 2005 Neural processes underlying memory attribution on a reality-monitoring task. *Cereb. Cortex* **16**, 1126–1133. (doi:10.1093/cercor/bhj054)
  44. Simons JS, Davis SW, Gilbert SJ, Frith CD, Burgess PW. 2006 Discriminating imagined from perceived information engages brain areas implicated in schizophrenia. *Neuroimage* **32**, 696–703. (doi:10.1016/j.neuroimage.2006.04.209)
  45. Metzak PD, Lavigne KM, Woodward TS. 2015 Functional brain networks involved in reality monitoring. *Neuropsychologia* **75**, 50–60. (doi:10.1016/j.neuropsychologia.2015.05.014)
  46. Vinogradov S, Luks TL, Schulman BJ, Simpson GV. 2008 Deficit in a neural correlate of reality monitoring in schizophrenia patients. *Cereb. Cortex* **18**, 2532–2539. (doi:10.1093/cercor/bhn028)
  47. Stephane M, Kuskowski M, McClannahan K, Surerus C, Nelson K. 2010 Evaluation of speech misattribution bias in schizophrenia. *Psychol. Med.* **40**, 741–748. (doi:10.1017/S003329170999081X)
  48. Whalley HC, Simonotto E, Flett S, Marshall I, Ebmeier KP, Owens DGC, Goddard NH, Johnstone EC, Lawrie SM. 2004 fMRI correlates of state and trait effects in subjects at genetically enhanced risk of schizophrenia. *Brain* **127**, 478–490. (doi:10.1093/brain/awh070)
  49. MacDonald AW, Carter CS, Kerns JG, Ursu S, Barch DM, Holmes AJ, Stenger VA, Cohen JD. 2005 Specificity of prefrontal dysfunction and context processing deficits to schizophrenia in never-medicated patients with first-episode psychosis. *Am. J. Psychiatry* **162**, 475–484. (doi:10.1176/appi.ajp.162.3.475)
  50. Feinberg I. 1978 Efference copy and corollary discharge: implications for thinking and its disorders. *Schizophr. Bull.* **4**, 636–640. (doi:10.1093/schbul/4.4.636)
  51. Seal M, Aleman A, McGuire P. 2004 Compelling imagery, unanticipated speech and deceptive memory: neurocognitive models of auditory verbal hallucinations in schizophrenia. *Cognit. Neuropsychiatry* **9**, 43–72. (doi:10.1080/13546800344000156)
  52. Blakemore S.-J., Wolpert DM, Frith CD. 2002 Abnormalities in the awareness of action. *Trends Cogn. Sci.* **6**, 237–242. (doi:10.1016/S1364-6613(02)01907-1)
  53. Ford JM, Mathalon DH, Heinks T, Kalba S, Faustman WO, Roth WT. 2001 Neurophysiological evidence of corollary discharge dysfunction in schizophrenia. *Am. J. Psychiatry* **158**, 2069–2071. (doi:10.1176/appi.ajp.158.12.2069)
  54. Ford JM, Mathalon DH. 2004 Electrophysiological evidence of corollary discharge dysfunction in schizophrenia during talking and thinking. *J. Psychiatr. Res.* **38**, 37–46. (doi:10.1016/S0022-3956(03)00095-5)



55. Ford JM, Mathalon DH, Roach BJ, Keedy SK, Reilly JL, Gershon ES, Sweeney JA. 2013 Neurophysiological evidence of corollary discharge function during vocalization in psychotic patients and their nonpsychotic first-degree relatives. *Schizophr. Bull.* **39**, 1272–1280. (doi:10.1093/schbul/sbs129)
56. Ford JM, Mathalon DH. 2019 Efference copy, corollary discharge, predictive coding, and psychosis. *Biol. Psychiatry Cogn. Neurosci. Neuroimaging* **4**, 764–767. (doi:10.1016/j.bpsc.2019.07.005)
57. Gallagher S. 2004 Neurocognitive models of schizophrenia: a neurophenomenological critique. *Psychopathology* **37**, 8–19. (doi:10.1159/000077014)
58. Garrison JR, Moseley P, Alderson-Day B, Smailes D, Fernyhough C, Simons JS. 2017 Testing continuum models of psychosis: no reduction in source monitoring ability in healthy individuals prone to auditory hallucinations. *Cortex* **91**, 197–207. (doi:10.1016/j.cortex.2016.11.011)
59. Diederer KJM, Daalman K, de Weijer AD, Neggers SF. W., van Gastel W, Blom JD, Kahn RS, Sommer IEC. 2011 Auditory hallucinations elicit similar brain activation in psychotic and nonpsychotic individuals. *Schizophr. Bull.* **38**, 1074–1082. (doi:10.1093/schbul/sbr033)
60. Hunter MD, Eickhoff SB, Miller TWR, Farrow TFD, Wilkinson ID, Woodruff PWR. 2006 Neural activity in speech-sensitive auditory cortex during silence. *Proc. Natl Acad. Sci. USA* **103**, 189. (doi:10.1073/pnas.0506268103)
61. 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)
62. Sidari 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**, 171–178. (doi:10.1038/nn.4545)
63. 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)
64. Lamme VA. F. 2006 Towards a true neural stance on consciousness. *Trends Cogn. Sci.* **10**, 494–501. (doi:10.1016/j.tics.2006.09.001)
65. Dehaene S, Changeux J. 2011 Experimental and theoretical approaches to conscious processing. *Neuron* **70**, 200–227. (doi:10.1016/j.neuron.2011.03.018)
66. 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)
67. 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)
68. D'Esposito M, Postle B. 2015 The cognitive neuroscience of working memory. *Annu. Rev. Psychol.* **66**, 115–142. (doi:10.1146/annurev-psych-010814-015031)
69. 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)
70. Christophel TB, Klink PC, Spitzer B, Roelfsema PR, Haynes J-D. 2017 The distributed nature of working memory. *Trends Cogn. Sci.* **21**, 111–124. (doi:10.1016/j.tics.2016.12.007)
71. Fazekas P, Nemeth G. 2018 Dream experiences and the neural correlates of perceptual consciousness and cognitive access. *Phil. Trans. R. Soc. B* **373**, 20170356. (doi:10.1098/rstb.2017.0356)
72. Ishai A, Ungerleider LG, Haxby JV. 2000 Distributed neural systems for the generation of visual images. *Neuron* **28**, 979–990. (doi:10.1016/S0896-6273(00)00168-9)
73. O'Craven KM, Kanwisher N. 2000 Mental imagery of faces and places activates corresponding stimulus-specific brain regions. *J. Cogn. Neurosci.* **12**, 1013–1023. (doi:10.1162/08989290051137549)
74. Stokes M, Thompson R, Cusack R, Duncan J. 2009 Top-down activation of shape-specific population codes in visual cortex during mental imagery. *J. Neurosci.* **29**, 1565–1572. (doi:10.1523/JNEUROSCI.4657-08.2009)
75. Reddy L, Tsuchiya N, Serre T. 2010 Reading the mind's eye: decoding category information during mental imagery. *Neuroimage* **50**, 818–825. (doi:10.1016/j.neuroimage.2009.11.084)
76. Albers AM, Kok P, Toni I, Dijkerman HC, de Lange FP. 2013 Shared representations for working memory and mental imagery in early visual cortex. *Curr. Biol.* **23**, 1427–1431. (doi:10.1016/j.cub.2013.05.065)
77. Tong F. 2013 Imagery and visual working memory: one and the same? *Trends Cogn. Sci.* **17**, 489–490. (doi:10.1016/j.tics.2013.08.005)
78. Johnson M, Johnson M. 2014 Decoding individual natural scene representations during perception and imagery. *Front. Hum. Neurosci.* **8**, 1–14. (doi:10.3389/fnhum.2014.00059)
79. Naselaris T, Olman CA, Stansbury DE, Ugurbil K, Gallant JL. 2015 A voxel-wise encoding model for early visual areas decodes mental images of remembered scenes. *Neuroimage* **105**, 215–228. (doi:10.1016/j.neuroimage.2014.10.018)
80. Dijkstra N, Bosch SE, van Gerven MA. J. 2017 Vividness of visual imagery depends on the neural overlap with perception in visual areas. *J. Neurosci.* **37**, 1367–1373. (doi:10.1523/JNEUROSCI.3022-16.2016)
81. Dijkstra N, Bosch SE, van Gerven MAJ. 2019 Shared neural mechanisms of visual perception and imagery. *Trends Cogn. Sci.* **23**, 423–434. (doi:10.1016/j.tics.2019.02.004)
82. Horikawa T, Tamaki M, Miyawaki Y, Kamitani Y. 2013 Neural decoding of visual imagery during sleep. *Science* **340**, 639–642. (doi:10.1126/science.1234330)
83. Rechtschaffen A, Buchignani C. 1983 Visual dimensions and correlates of dream images. *Sleep Res.* **12**, 189.
84. Antrobus J, Hartwig P, Rosa D, Reinsel R, Fein G. 1987 Brightness and clarity of REM and NREM imagery: photo response scale. *Sleep Res.* **16**, 240.
85. Rechtschaffen A, Buchignani C. 1992 The visual appearance of dreams. In *The neuropsychology of sleep and dreaming* (eds J Antrobus, M Bertini), pp. 143–156. Hillsdale, NJ: Lawrence Erlbaum.
86. Kerr N. 1993 Mental imagery, dreams and perception. In *Dreaming as cognition* (eds C Cavallero, D Foulkes), pp. 18–37. London, UK: Harvester Wheatsheaf.
87. Antrobus J, Kondo T, Reinsel R, Fein G. 1995 Dreaming in the late morning: summation of REM and diurnal cortical activation. *Conscious. Cogn.* **4**, 275–299. (doi:10.1006/ccog.1995.1039)
88. Fosse R. 2000 REM mentation in narcoleptics and normals: an empirical test of two neurocognitive theories. *Conscious. Cogn.* **9**, 488–509. (doi:10.1006/ccog.2000.0466)
89. Antrobus JS, Wamsley EJ. 2009 REM/NREM differences in dream content. In *The neuroscience of sleep* (eds R Stickgold, MP Walker), pp. 310–315. London, UK: Academic Press.
90. Fulford J, Milton F, Salas D, Smith A, Simler A, Winlove C, Zeman A. 2017 The neural correlates of visual imagery vividness – an fMRI study and literature review. *Cortex* **105**, 26–40. (doi:10.1016/j.cortex.2017.09.014)
91. Cornoldi C, De Beni R, Giuberti F, Marucci F, Massironi M, Mazzone G. 1991 The study of vividness of images. In *Mental images in human cognition* (eds RH Logie, M Denis), pp. 305–312. Amsterdam, The Netherlands: Elsevier.
92. Denis M. 1995 Vividness of visual imagery and the evaluation of its effects on performance. *J. Ment. Imag.* **19**, 136–138.
93. McKelvie SJ. 1995 The VVIQ as a psychometric test of individual differences in visual imagery vividness: a critical quantitative review and plea for direction. *J. Ment. Imag.* **19**, 1–106.
94. McKelvie SJ. 1995 The VVIQ and beyond: vividness and its measurement. *J. Ment. Imag.* **19**, 197–252.
95. Kind A. 2017 Imaginative vividness. *J. Am. Philos. Assoc.* **3**, 32–50. (doi:10.1017/apa.2017.10)
96. Marks DF. 1973 Visual imagery differences in the recall of pictures. *Br. J. Psychol.* **64**, 17–24. (doi:10.1111/j.2044-8295.1973.tb01322.x)
97. Fazekas P, Overgaard M. 2016 Multidimensional models of degrees and levels of consciousness. *Trends Cogn. Sci.* **20**, 715–716. (doi:10.1016/j.tics.2016.06.011)
98. Fazekas P, Overgaard M. 2018 A multi-factor account of degrees of awareness. *Cogn. Sci.* **42**, 1833–1859. (doi:10.1111/cogs.12478)
99. Fazekas P, Overgaard M. 2018 Multiple factors and multiple mechanisms determine the quality of conscious experiences: a reply to Anzulewicz and Wierchoń. *Cogn. Sci.* **42**, 2101–2103. (doi:10.1111/cogs.12647)

100. Morales J. In preparation. Mental strength: a theory of experience intensity.
101. Carrasco M, Ling S, Read S. 2004 Attention alters appearance. *Nat. Neurosci.* **7**, 308–313. (doi:10.1038/nm1194)
102. Fuller S, Carrasco M. 2006 Exogenous attention and color perception: performance and appearance of saturation and hue. *Vis. Res.* **46**, 4032–4047. (doi:10.1016/j.visres.2006.07.014)
103. Rossi AF, Paradiso MA. 1999 Neural correlates of perceived brightness in the retina, lateral geniculate nucleus, and striate cortex. *J. Neurosci.* **19**, 6145–6156. (doi:10.1523/JNEUROSCI.19-14-06145.1999)
104. Masuda N, Doiron B. 2007 Gamma oscillations of spiking neural populations enhance signal discrimination. *PLoS Comput. Biol.* **3**, 2348–2355. (doi:10.1371/journal.pcbi.0030236)
105. Le Van Quyen M, Staba R, Bragin A, Dickson C, Valderrama M, Fried I, Engel J. 2010 Large-scale microelectrode recordings of high-frequency gamma oscillations in human cortex during sleep. *J. Neurosci.* **30**, 7770. (doi:10.1523/JNEUROSCI.5049-09.2010)
106. Panzeri S, Macke JH, Gross J, Kayser C. 2015 Neural population coding: combining insights from microscopic and mass signals. *Trends Cogn. Sci.* **19**, 162–172. (doi:10.1016/j.tics.2015.01.002)
107. Martinez-Trujillo JC, Treue S. 2004 Feature-based attention increases the selectivity of population responses in primate visual cortex. *Curr. Biol.* **14**, 744–751. (doi:10.1016/j.cub.2004.04.028)
108. Maunsell JH. R, Treue S. 2006 Feature-based attention in visual cortex. *Trends Neurosci.* **29**, 317–322. (doi:10.1016/j.tins.2006.04.001)
109. Webster MA, Georgeson MA, Webster SM. 2002 Neural adjustments to image blur. *Nat. Neurosci.* **5**, 839–840. (doi:10.1038/nm906)
110. Gobell J, Carrasco M. 2005 Attention alters the appearance of spatial frequency and gap size. *Psychol. Sci.* **16**, 644–651. (doi:10.1111/j.1467-9280.2005.01588.x)
111. Abrams J, Barbot A, Carrasco M. 2010 Voluntary attention increases perceived spatial frequency. *Attent. Percept. Psychophys.* **72**, 1510–1521. (doi:10.3758/APP.72.6.1510)
112. Webster MA, Marcos S. 2017 Neural adaptation to blur. In *Handbook of visual optics*, vol. II (ed. P Artal), pp. 307–323. London, UK: CRC Press.
113. Kauffmann L, Ramanoël S, Peyrin C. 2014 The neural bases of spatial frequency processing during scene perception. *Front. Integr. Neurosci.* **8**, 1–14. (doi:10.3389/fnint.2014.00037)
114. Musel B, Kauffmann L, Ramanoël S, Giavarini C, Guyader N, Chauvin A, Peyrin C. 2014 Coarse-to-fine categorization of visual scenes in scene-selective cortex. *J. Cogn. Neurosci.* **26**, 2287–2297. (doi:10.1162/jocn\_a\_00643)
115. Lu Y *et al.* 2018 Revealing detail along the visual hierarchy: neural clustering preserves acuity from V1 to V4. *Neuron* **98**, 417–428. (doi:10.1016/j.neuron.2018.03.009)
116. Cui X, Jeter CB, Yang D, Montague PR, Eagleman DM. 2007 Vividness of mental imagery: individual variability can be measured objectively. *Vis. Res.* **47**, 474–478. (doi:10.1016/j.visres.2006.11.013)
117. Dijkstra N, Zeidman P, Ondobaka S, van Gerven MAJ, Friston K. 2017 Distinct top-down and bottom-up brain connectivity during visual perception and imagery. *Sci. Rep.* **7**, 1–9. (doi:10.1038/s41598-017-05888-8)
118. Daselaar SM, Prat Y, Huijbers W, Pennartz CMA. 2010 Modality-specific and modality-independent components of the human imagery system. *Neuroimage* **52**, 677–685. (doi:10.1016/j.neuroimage.2010.04.239)
119. Herholz SC, Halpern AR, Zatorre RJ. 2012 Neuronal correlates of perception, imagery, and memory for familiar tunes. *J. Cogn. Neurosci.* **24**, 1382–1397. (doi:10.1162/jocn\_a\_00216)
120. Halpern AR. 2015 Differences in auditory imagery self-report predict neural and behavioral outcomes. *Psychomusicology: Music Mind Brain* **25**, 37–47. (doi:10.1037/pmu0000081)
121. Lima CF *et al.* 2015 Feel the noise: relating individual differences in auditory imagery to the structure and function of sensorimotor systems. *Cereb. Cortex* **25**, 4638–4650. (doi:10.1093/cercor/bhv134)
122. Garrison JR, Fernyhough C, McCarthy-Jones S, Simons JS, Sommer IEC. 2019 Paracingulate sulcus morphology and hallucinations in clinical and nonclinical groups. *Schizophr. Bull.* **45**, 733–741. (doi:10.1093/schbul/sby157)
123. DeRosse P, Karlsgodt KH. 2015 Examining the psychosis continuum. *Curr. Behav. Neurosci. Rep.* **2**, 80–89. (doi:10.1007/s40473-015-0040-7)
124. van Os J, Reininghaus U. 2016 Psychosis as a transdiagnostic and extended phenotype in the general population. *World Psychiatry* **15**, 118–124. (doi:10.1002/wps.20310)
125. Baumeister D, Sedgwick O, Howes O, Peters E. 2017 Auditory verbal hallucinations and continuum models of psychosis: a systematic review of the healthy voice-hearer literature. *Clin. Psychol. Rev.* **51**, 125–141. (doi:10.1016/j.cpr.2016.10.010)
126. Larøi F, Van der Linden M, Marczewski P. 2004 The effects of emotional salience, cognitive effort and meta-cognitive beliefs on a reality monitoring task in hallucination-prone subjects. *Br. J. Clin. Psychol.* **43**, 221–233. (doi:10.1348/0144665031752970)
127. McKague M, McAnally KI, Skovron M, Bendall S, Jackson HJ. 2012 Source monitoring and proneness to auditory-verbal hallucinations: a signal detection analysis. *Cognit. Neuropsychiatry* **17**, 544–562. (doi:10.1080/13546805.2012.676311)
128. Buda M, Fornio A, Bergström ZM, Simons JS. 2011 A specific brain structural basis for individual differences in reality monitoring. *J. Neurosci.* **31**, 14308. (doi:10.1523/JNEUROSCI.3595-11.2011)
129. Aynsworth C, Nemat N, Collerton D, Smailes D, Dudley R. 2017 Reality monitoring performance and the role of visual imagery in visual hallucinations. *Behav. Res. Ther.* **97**, 115–122. (doi:10.1016/j.brat.2017.07.012)
130. Allen P *et al.* 2012 Neuroimaging auditory hallucinations in schizophrenia: from neuroanatomy to neurochemistry and beyond. *Schizophr. Bull.* **38**, 695–703. (doi:10.1093/schbul/sbs066)
131. Sugimori E, Mitchell KJ, Raye CL, Greene EJ, Johnson MK. 2014 Brain mechanisms underlying reality monitoring for heard and imagined words. *Psychol. Sci.* **25**, 403–413. (doi:10.1177/0956797613505776)
132. Galton F. 1883 *Inquiries into human faculty and its development*. London, UK: MacMillan.
133. Barrett TR. 1993 Verbal hallucinations in normals—II: self-reported imagery vividness. *Pers. Individ. Differ.* **15**, 61–67. (doi:10.1016/0191-8869(93)90041-Z)
134. Aleman A, Nieuwenstein MR, Bökler KBE, de Haan EHF. 2000 Mental imagery and perception in hallucination-prone individuals. *J. Nerv. Ment. Dis.* **188**, 830–836. (doi:10.1097/00005053-200012000-00007)
135. Aleman A, Bökler KBE, Hijman R, de Haan EHF, Kahn RS. 2003 Cognitive basis of hallucinations in schizophrenia: role of top-down information processing. *Schizophr. Res.* **64**, 175–185. (doi:10.1016/S0920-9964(03)00060-4)
136. Sack AT, van de Ven VG, Etschenberg S, Schatz D, Linden DEJ. 2005 Enhanced vividness of mental imagery as a trait marker of schizophrenia? *Schizophr. Bull.* **31**, 97–104. (doi:10.1093/schbul/sbi011)
137. Brébion G, Ohlsen RI, Pilowsky LS, David AS. 2008 Visual hallucinations in schizophrenia: confusion between imagination and perception. *Neuropsychology* **22**, 383–389. (doi:10.1037/0894-4105.22.3.383)
138. Mondino M, Dondé C, Lavallé L, Haesebaert F, Brunelin J. 2019 Reality-monitoring deficits and visual hallucinations in schizophrenia. *Eur. Psychiatry* **62**, 10–14. (doi:10.1016/j.eurpsy.2019.08.010)
139. Shine JM, Keogh R, O'Callaghan C, Muller AJ, Lewis SJG, Pearson J. 2015 Imagine that: elevated sensory strength of mental imagery in individuals with Parkinson's disease and visual hallucinations. *Proc. R. Soc. B* **282**, 20142047. (doi:10.1098/rspb.2014.2047)
140. El Haj M, Badcock JC, Jardri R, Larøi F, Roche J, Sommer IE, Gallouj K. 2019 A look into hallucinations: the relationship between visual imagery and hallucinations in Alzheimer's disease. *Cognit. Neuropsychiatry* **24**, 275–283. (doi:10.1080/13546805.2019.1632180)
141. Gonsalves B, Reber PJ, Gitelman DR, Parrish TB, Mesulam MM, Paller KA. 2004 Neural evidence that vivid imagining can lead to false remembering. *Psychol. Sci.* **15**, 655–660. (doi:10.1111/j.0956-7976.2004.00736.x)
142. Stephan-Otto C, Siddi S, Senior C, Cuevas-Esteban J, Cambra-Martí MR, Ochoa S, Brébion G. 2017 Remembering verbally-presented items as pictures: brain activity underlying visual mental images in schizophrenia patients with visual

- hallucinations. *Cortex* **94**, 113–122. (doi:10.1016/j.cortex.2017.06.009)
143. Stephan-Otto C, Siddi S, Senior C, Muñoz-Samons D, Ochoa S, Sánchez-Laforga AM, Brébion G. 2017 Visual imagery and false memory for pictures: a functional magnetic resonance imaging study in healthy participants. *PLoS ONE* **12**, e0169551. (doi:10.1371/journal.pone.0169551)
144. Aleman A, Bocker KBE, Haan EHF. 1999 Disposition towards hallucination and subjective versus objective vividness of imagery in normal subjects. *Pers. Individ. Differ.* **27**, 707–714. (doi:10.1016/S0191-8869(98)00270-0).
145. van de Ven V, Merckelbach H. 2003 The role of schizotypy, mental imagery, and fantasy proneness in hallucinatory reports of undergraduate students. *Pers. Individ. Differ.* **35**, 889–896. (doi:10.1016/S0191-8869(02)00304-5)
146. Oertel V, Rotarska-Jagiela A, van de Ven V, Haenschel C, Grube M, Stangier U, Maurer K, Linden DEJ. 2009 Mental imagery vividness as a trait marker across the schizophrenia spectrum. *Psychiatry Res.* **167**, 1–11. (doi:10.1016/j.psychres.2007.12.008)
147. Zeman A *et al.* 2020 Phantasia—the psychological significance of lifelong visual imagery vividness extremes. *Cortex* **130**, 426–440. (doi:10.1016/j.cortex.2020.04.003)
148. Dawes AJ, Keogh R, Andrillon T, Pearson J. 2020 A cognitive profile of multi-sensory imagery, memory and dreaming in aphantasia. *Sci. Rep.* **10**, 10022. (doi:10.1038/s41598-020-65705-7)
149. Pearson J, Westbrook F. 2015 Phantom perception: voluntary and involuntary nonretinal vision. *Trends Cogn. Sci.* **19**, 278–284. (doi:10.1016/j.tics.2015.03.004)
150. Nanay B. 2016 Hallucination as mental imagery. *J. Conscious. Stud.* **23**, 65–81.
151. Killingsworth MA, Gilbert DT. 2010 A wandering mind is an unhappy mind. *Science* **330**, 932–932. (doi:10.1126/science.1192439)
152. Christoff K, Irving ZC, Fox KCR, Spreng RN, Andrews-Hanna JR. 2016 Mind-wandering as spontaneous thought: a dynamic framework. *Nat. Rev. Neurosci.* **17**, 718–731. (doi:10.1038/nrn.2016.113)
153. Perogamvros L, Baird B, Seibold M, Riedner B, Boly M, Tononi G. 2017 The phenomenal contents and neural correlates of spontaneous thoughts across wakefulness, NREM sleep, and REM sleep. *J. Cogn. Neurosci.* **29**, 1766–1777. (doi:10.1162/jocn\_a\_01155)
154. Wang H-T, Poerio G, Murphy C, Bzdok D, Jefferies E, Smallwood J. 2018 Dimensions of experience: exploring the heterogeneity of the wandering mind. *Psychol. Sci.* **29**, 56–71. (doi:10.1177/0956797617728727)
155. Smallwood J, Schooler JW. 2006 The restless mind. *Psychol. Bull.* **132**, 946–958. (doi:10.1037/0033-2909.132.6.946)
156. Smallwood J, Schooler JW. 2015 The science of mind wandering: empirically navigating the stream of consciousness. *Annu. Rev. Psychol.* **66**, 487–518. (doi:10.1146/annurev-psych-010814-015331)
157. Raichle ME, MacLeod AM, Snyder AZ, Powers WJ, Gusnard DA, Shulman GL. 2001 A default mode of brain function. *Proc. Natl Acad. Sci. USA* **98**, 676. (doi:10.1073/pnas.98.2.676)
158. Raichle ME. 2015 The Brain's default mode network. *Annu. Rev. Neurosci.* **38**, 433–447. (doi:10.1146/annurev-neuro-071013-014030)
159. Fox KCR, Spreng RN, Ellamil M, Andrews-Hanna JR, Christoff K. 2015 The wandering brain: meta-analysis of functional neuroimaging studies of mind-wandering and related spontaneous thought processes. *Neuroimage* **111**, 611–621. (doi:10.1016/j.neuroimage.2015.02.039)
160. Andrews-Hanna J, Irving ZC, Fox KC. R., Spreng RN, Christoff K. 2018 The neuroscience of spontaneous thought: an evolving, interdisciplinary field. In *The Oxford handbook of spontaneous thought* (eds KCR Fox, K Christoff), pp. 143–164. New York, NY: Oxford University Press.
161. Schooler JW. 2002 Re-representing consciousness: dissociations between experience and meta-consciousness. *Trends Cogn. Sci.* **6**, 339–344. (doi:10.1016/S1364-6613(02)01949-6)
162. Yao N *et al.* 2014 The default mode network is disrupted in Parkinson's disease with visual hallucinations. *Hum. Brain Mapp.* **35**, 5658–5666. (doi:10.1002/hbm.22577)
163. Franciotti R *et al.* 2015 Default mode network links to visual hallucinations: a comparison between Parkinson's disease and multiple system atrophy. *Mov. Disord.* **30**, 1237–1247. (doi:10.1002/mds.26285)
164. Shine JM, Muller AJ, O'Callaghan C, Hornberger M, Halliday GM, Lewis SJG. 2015 Abnormal connectivity between the default mode and the visual system underlies the manifestation of visual hallucinations in Parkinson's disease: a task-based fMRI study. *NPJ Parkinson's Dis.* **1**, 15003. (doi:10.1038/npjparkd.2015.3)
165. Turnbull A, Wang H-T, Schooler JW, Jefferies E, Margulies DS, Smallwood J. 2019 The ebb and flow of attention: between-subject variation in intrinsic connectivity and cognition associated with the dynamics of ongoing experience. *Neuroimage* **185**, 286–299. (doi:10.1016/j.neuroimage.2018.09.069)
166. Shin D-J, Lee TY, Jung WH, Kim SN, Jang JH, Kwon JS. 2015 Away from home: the brain of the wandering mind as a model for schizophrenia. *Schizophr. Res.* **165**, 83–89. (doi:10.1016/j.schres.2015.03.021)
167. Northoff G, Qin P. 2011 How can the brain's resting state activity generate hallucinations? A 'resting state hypothesis' of auditory verbal hallucinations. *Schizophr. Res.* **127**, 202–214. (doi:10.1016/j.schres.2010.11.009)
168. Muller AJ, Shine JM, Halliday GM, Lewis SJ. G. 2014 Visual hallucinations in Parkinson's disease: theoretical models. *Mov. Disord.* **29**, 1591–1598. (doi:10.1002/mds.26004)
169. Hall JM, O'Callaghan C, Shine JM, Muller AJ, Phillips JR, Walton CC, Lewis SJ. G, Moustafa AA. 2016 Dysfunction in attentional processing in patients with Parkinson's disease and visual hallucinations. *J. Neural Transm.* **123**, 503–507. (doi:10.1007/s00702-016-1528-3)
170. Collerton D, Taylor J-P, Tsuda I, Fujii H, Nara S, Aihara K, Katori Y. 2016 How can we see things that are not there? Current insights into complex visual hallucinations. *J. Conscious. Stud.* **23**, 195–227.
171. Somer E. 2002 Maladaptive daydreaming: a qualitative inquiry. *J. Contemp. Psychother.* **32**, 197–212. (doi:10.1023/A:1020597026919)
172. Somer E, Lehfelf J, Bigelsen J, Jopp DS. 2016 Development and validation of the Maladaptive Daydreaming Scale (MDS). *Conscious. Cogn.* **39**, 77–91. (doi:10.1016/j.concog.2015.12.001)
173. Somer E, Soffer-Dudek N, Ross CA, Halpern N. 2017 Maladaptive daydreaming: proposed diagnostic criteria and their assessment with a structured clinical interview. *Psychol. Conscious.: Theory Res. Pract.* **4**, 176–189. (doi:10.1037/cns0000114)
174. Seli P, Risko EF, Smilek D, Schacter DL. 2016 Mind-wandering with and without intention. *Trends Cogn. Sci.* **20**, 605–617. (doi:10.1016/j.tics.2016.05.010)