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Understanding visual hallucinations: A new synthesis

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ABSTRACT

Despite decades of research, we do not definitively know how people sometimes see things that are not there. Eight models of complex visual hallucinations have been published since 2000, including Deafferentation, Reality Monitoring, Perception and Attention Deficit, Activation, Input, and Modulation, Hodological, Attentional Networks, Active Inference, and Thalamocortical Dysrhythmia Default Mode Network Decoupling. Each was derived from different understandings of brain organisation. To reduce this variability, representatives from each research group agreed an integrated Visual Hallucination Framework that is consistent with current theories of veridical and hallucinatory vision. The Framework delineates cognitive systems relevant to hallucinations. It allows a systematic, consistent, investigation of relationships between the phenomenology of visual hallucinations and changes in underpinning cognitive structures. The episodic nature of hallucinations highlights separate factors associated with the onset, persistence, and end of specific hallucinations suggesting a complex relationship between state and trait markers of hallucination risk. In addition to a harmonised interpretation of existing evidence, the Framework highlights new avenues of research, and potentially, new approaches to treating distressing hallucinations.

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Each morning, I saw a Japanese lady from my ward window. She used to come out from the ward further up and sit around the corner. She had a cream and black jacket and white sneakers, and sleek black hair in a Japanese style. She never said anything but I was worried to death about her. The wind was howling and the rain fell and she was soaking wet.

Description of a recurrent hallucination from a person with a diagnosis of dementia with Lewy bodies.

1. The challenges of understanding visual hallucinations

"To see is to believe" is a commonplace expression. Seeing things that are not truly there challenges our basic intuition of a world outside ourselves that we can take as real because it is accurately perceived. The link between visual perception and reality breaks down in numerous ways creating, as we illustrate in Fig. 1, a wide range of non-veridical visual experiences. Foremost amongst these experiences are visual hallucinations, which themselves come in many forms ranging from simple fleeting shapes or lights via intermediate forms to the realistic images in exquisite detail illustrated in the quote.

In this paper, we address purely visual (i.e. unimodal) complex (sometimes referred to as "formed") hallucinations of meaningful percepts, such as people, faces, animals, or objects (ffytche, 2005; Manford and Andermann, 1998; Santhouse, Howard, and ffytche, 2000). Though these phenomena are found in many clinical and non-clinical settings throughout life, our attention is on those experienced in adulthood (Collerton, Perry, and McKeith, 2005). This focus on unimodal visual hallucinations is not to dismiss the relevance of multimodal hallucinations or simpler perceptual phenomena; rather it is an effort to tackle, in a parsimonious fashion, a phenomenon of significant clinical and theoretical importance in its most usual form that, once understood, will allow further studies of its variants.

Thus, we will focus on those recurrent hallucinations that most often (though not always as we discuss later) are associated with clinical neurological or perceptual disorders, and that have the striking character illustrated in the quote. They are most commonly of a human figure or face, though animals, objects and meaningful patterns are also frequent. They generally move. They are perceptually convincing. They occur episodically at particular times and in particular places (Collerton et al., 2005).

1.1. The importance of complex visual hallucinations

Interest in complex visual hallucination comes from a number of sources. They are fascinating phenomena in their own right. Consequently, they have attracted attention for centuries with repeated attempts to understand them within the explanatory frameworks of the time (Berrios and Markova, 2015).

Currently, pure visual hallucinations are recognised as characteristic of neurological diseases including Lewy body disorders (Parkinson's disease (Barnes and David, 2001; Goetz, Ouyang, Negron, and Stebbins, 2010; Goetz, Stebbins, and Ouyang, 2011), Parkinson's disease dementia, and dementia with Lewy bodies (Mosimann et al., 2006)), and rarer disorders such as peduncular hallucinosis (Benke, 2006) and narcolepsy (Leu-Semenescu et al., 2011), as well as the Charles Bonnet syndrome of eye disease (ffytche, 2005). Multimodal hallucinations with a visual component are found in advanced Lewy body disorders (Goetz, Stebbins, and Ouyang, 2011; Montagnese et al., 2021), in delirium (Webster and Holroyd, 2000), and in psychotic disorders (Dudley et al., 2018; Waters et al., 2014) such as schizophrenia (Chouinard et al., 2019;

Fig. 1. Complex visual hallucinations and other experiences. Seeing things that are not there encompasses a wide range of fuzzy, hard to precisely define phenomena (Blom, 2015) including from left to right: presence, passage, fortification, and simple (or unformed) hallucinations, illusions, voluntary images, multimodal hallucinations with a visual component, flashbacks, and dreams (see Glossary for definitions). In the centre are the formed hallucinations of faces, figures, and animals that we address.

David et al., 2011; Gauntlett-Gilbert and Kuipers, 2005; Toh, McCarthy-Jones, Copolov, and Rossell, 2019; Van Ommen et al., 2016).

They are often accompanied by impairments in perception; other perceptual phenomena such as illusions; and other forms of hallucinations such as presence and passage, especially in neurodegenerative disorders as they progress (Goetz et al., 2010, 2011). They tend to be associated with high rates of distress, possibly reflecting secondary delusions (Gauntlett-Gilbert and Kuipers, 2005; Scott, Schein, Feuer, and Folstein, 2001), with carer stress (Renouf, ffytche, Pinto, Murray, and Lawrence, 2018), and in some disorders, faster disease progression (Ibarretxe-Bilbao et al., 2010) and greater institutionalization (Aarsland, Larsen, Tandberg, and Laake, 2000). Treatments are of limited effectiveness (Collerton and Taylor, 2013; O'Brien et al., 2020).

2. Understanding how hallucinations occur

2.1. Problems with current approaches

Given their impact, there have been numerous attempts to account for how well-formed recurrent visual hallucinations occur; some based in a specific clinical disorder, others with an aim to be more generally applicable. However, progress has been limited.

We have identified eight distinct models that have been developed since 2000. All are anchored in a mechanistic approach to the core phenomenon of complex visual hallucinations, though some have wider aims than just accounting for these specific experiences. For example, they may also seek to explain commonly co-occurring visual experiences such as simple or passage hallucinations (Fénelon et al., 2000; Urwyler et al., 2014) or multimodal hallucinations (Fernyhough, 2019). Similarly, they might address associated symptoms such as fluctuations (O'Brien et al., 2005), disturbances of the sleep-wake cycle (Fénelon et al., 2000; Goetz et al., 2010; Mosimann et al., 2006), delusions (Holroyd et al., 2001), and insight (Llebaria et al., 2010) or distress (Dudley et al., 2012).

We illustrate the disparity of these theories' approaches in Table 1 (Further details of each can be found in Fig. 3). Developed by researchers working in different scientific domains, the explanations show no obvious commonality in explicit paradigms or frameworks of brain organisation and range widely in the cognitive systems that they focus on. Thus, their true similarities or differences, or strengths and weaknesses are hard to judge or synthesize into a unified model (Muller, Shine, Halliday, and Lewis, 2014).

As a consequence of these disparities, there has been no *step-by-step* conceptual evolution towards a unified model in the last twenty years, despite a burgeoning volume of clinical and experimental data. This leaves open major questions such as how hallucinations occur only in some people and only at some times, or how phenomenology can vary from person to person and from time to time. Without a common theoretical framework, our ability to derive unique predictions from each model such that research data can be used easily and unambiguously to test single or combined models is highly limited. Furthermore, since models differ in the breadth of cognitive functions that they address, it is hard to assess their degree of overlap; in particular whether a direct effect on a specific function in one model might be mimicked by an indirect effect in another. For example, the acquisition of visual sensory data might be impoverished directly by eye damage or indirectly by dysfunctional spatial attention.

To remedy this systemic weakness in the field we set out to create such a single representation.

2.2. Our proposed visual hallucination framework

Given that the primary distinction between a veridical and a hallucinatory subjective perception lies in its relationship to what is really out there, for the purposes of the framework, we have taken the default position that the subjective quality of both hallucinations and veridical perceptions are the same. There are numerous other possible relationships between veridical and hallucinatory representations (see

Table 1
Models of the genesis of unimodal visual hallucinations since 2000 in chronological order.

Model	Area of application	Explanatory framework	Modulated process	Ascending modulating factors	Descending modulating factors
Deafferentation (Burke, 2002)	Eye disease	Sensory neurophysiology	Neural activity underlying visual perception	Chronic loss of visual input leading to spontaneous activity (hyperexcitability / release) in early visual areas	Not integral to the model
Reality Monitoring (Barnes, Boubert, Harris, Lee, and David, 2003)	Parkinson's disease	Reality monitoring	Attribution of percepts as internal or external	Attended sensory input	Excessively convincing internal imagery to be taken as real
Perception and Attention Deficit (Collerton et al., 2005)	General model	Biased competition perceptual processing	Perceptual activity	Poor visual perception	Impaired attention
AIM (Diederich, Goetz, and Stebbins, 2005)	Lewy body disorders	Activation, Input, Modulation (AIM) model	Visual memories	Poor visual processing	Defective central monitoring
Hodological (ffytche (2008)}	General model	Cortical connectivity	Decreased integration of visual perception network	Altered short range connectivity between visual processing areas	Altered long range connectivity between frontal, parietal and occipital areas
Attentional Networks (Shine, Halliday, Naismith, and Lewis, 2011; Shine, O'Callaghan, Halliday and Lewis, 2014)	Lewy body disorders	Failure across disseminated functional networks	Default mode network has aberrant connectivity with the visual regions, which is not over-ridden by normal functional processes	Ambiguous percepts due to heightened salience modulated by Ventral Attentional Network and poor visual processing	Inability to recruit the dorsal attentional network to override abnormal connectivity between default mode network and visual regions
Active Inference (Friston, 2005; Benrimoh, Parr, Adams, and Friston, 2019; Zarkali et al., 2019)	General model	Bayesian inference	Posterior probability	Lowered precision of the likelihood of sensory data	Influence of an incorrect prior belief
Thalamocortical Dysrhythmia Default Mode Network Decoupling Hypothesis (Onofrj et al., 2019)	Lewy body disorders	Thalamocortical neural synchronisation / Attentional networks	Decoupling of default mode network	Not integral to the model	Frontal attentional network

Models which aimed to explain complex visual hallucinations were identified from MEDLINE and SCOPUS searches supplemented by reference lists, citations, and recommendations from authors. A timescale of 2000–2023 was used to identify current models.

Macpherson, 2013 for an exploration of other potential conceptualisations) and we admit that there is little evidence to confirm this parsimonious assumption, though the proposition is intrinsic to the perceptually convincing characteristics of complex visual hallucinations. Many people describe them as appearing as real as veridical perceptions.

Hence our working hypothesis is that there is no fundamental qualitative difference in perceptual representation between a veridical and a hallucinatory perception of the same image: I.e. a hallucination of a face is underpinned by the same processes as a veridical perception of the same face in the fusiform face area – or in the case of the distorted faces or figures which are not uncommon in hallucinations (Santhouse et al., 2000), the same as veridical perceptions of similarly distorted objects. The limited evidence that does bear upon this hypothesis comes from functional imaging which has indicated that the same brain areas are activated in both hallucinatory and veridical perception (ffytche et al., 1998; Goetz, Vaughan, Goldman, and Stebbins, 2014; Zmigrod, Garrison, Carr, and Simons, 2016). However, since the imaging techniques used in such studies are insensitive to possible differences at the level of neural circuitry, this is a hypothesis that needs testing. A systematic comparison of activation patterns associated with phenomenologically similar hallucinated, looked at, and imagined objects would allow a more direct comparison of the representations of these different types of perceptions. Never the less, this assumption does allow us to investigate how complex hallucinations can happen within the same framework as veridical perceptions, rather than having to develop two separate frameworks.

Given that hallucinations interrupt the flow of veridical visual perception, we have placed the transition processes in visual perception from *what has been seen* to *what is seen* to *what will be seen* at the centre of our Framework. Accounts of these processes have been most developed in generative processing approaches, particularly the Active Inference theory of hallucinations, though similar processes are described in many current theories of visual perception. The key concept here is that what is seen (termed a posterior in computational approaches) is a modification of what was expected to be seen (a prior) before encountering relevant sensory data. In turn each posterior is a prior for the next perception (Benrimoh et al., 2019; Jardri and Denève, 2013; Spratling, 2017). Predictions (sometimes called expectancies) of forthcoming sensory data are derived from what has been seen. These predictions of visual data are compared with the encountered visual input, and mismatches between predicted and actual input (prediction errors) are used to update perception.

In order to integrate the eight explanations of how there can be episodic disconnections between what is 'out there' in the environment and what is 'in there' within the brain, we needed to integrate these state concepts of data predictions, gathering of sensory data, and the comparison of actual and predicted data with the wider cognitive structures found in trait accounts.

3. Developing the framework

3.1. Process

Representatives from all the extant modelling groups in Table 1 together with other active researchers in visual hallucinations worked together to produce an agreed harmonized Visual Hallucinations Framework, to show how existing models could fit within that master construct, and to demonstrate the benefits of a unified approach. Sadly, William (Liam) Burke who developed the Deafferentation model died in 2018, so Dominic ffytche took the lead in integrating this approach into the Framework.

We agreed that we would not try to evaluate the correctness of each other's hallucination theories since evidence is often lacking or ambiguous. Instead, we chose to provide a solid, unified, up-to-date, inclusive, conceptual framework or basic structure for the science of visual hallucinations that could be utilized immediately, and critically refined or extended in the future. Thus, we set out to identify a set of core functional concepts in existing models that would be necessary and sufficient to build an inclusive framework, to translate these into a common language, and to arrange their explicit relationships in accordance with the wider perceptual literature. In developing the Framework, we were mindful of the need to keep it as simple as possible to facilitate communication and testability, while balancing this against the need to capture the details of individual models.

3.2. Specific challenges in developing a harmonized framework

3.2.1. Historical roots of models

Some of the many challenges arose from the independent historical roots of theories. Trait models of hallucinations which address factors that make hallucinatory episodes more likely were generally influenced by the neuropathology of clinical disorders with high levels of hallucinations. They concentrate on cognitive structure (e.g. Collerton et al., 2005) with those structural elements mainly coming from the classic neuropsychological paradigm of functionally specialised modules (e.g. Collerton et al., 2005: Fénelon et al., 2000: O'Brien et al., 2005). In contrast, state models which model factors associated with a specific hallucinatory episode (e.g. Todo, 2020) have been based on information processing models of whole brain dynamics drawn from a different paradigm; that of computational approaches using generative (also called constructive) predictive processing theory (e.g. Jardri and Denève, 2013).

Terminology for basic concepts also differed. For example, biased processing towards a specific perceptual outcome has been expressed in the neuropsychological construct, attention (Shine et al., 2011, 2014); in the biological phenomenon of synaptic gain (Brown and Friston, 2012); or in the computational term, precision (Friston, 2017).

3.2.2. Defining complex visual hallucinations

Furthermore, seeking to explain complex visual hallucinations depends upon them being a coherent, consistent phenomenon which reliably differs from other visual phenomena. This raises the question of how to define and capture these experiences. Complexity arises when trying to define this concept more tightly (Blom, 2015) and distinguish it from other visual experiences. Even apparently simple characteristics can prove difficult to pin down; both conceptually, and in practice. In Table 2 we summarise the common characteristics of definitions of visual hallucinations, illustrate the phenomena which hallucinations are distinguished from by each characteristic, and why distinctions are not absolute. This leaves open the question of how to deal with intermediate cases – for example, perceptions which might be classified as potentially illusionary or potentially hallucinatory. Resolution of this question will depend upon clearer understandings of the mechanisms which underlie these different phenomena. If the links between sensory data and subjective perception can be rigorously specified in hallucinations and in illusions, then it will be easier to classify phenomena.

We therefore agreed on a pragmatic definition: that a complex visual hallucination is seeing a recognisable thing that is not there, and by extension, that our models seek to account for the experiences of people who tell us that they are seeing things, when no-one else does.

3.2.3. The validity of an integrated approach

The models were so varied in the disorders that drove their creation, their basic frameworks - whether neural, computational, or neuropsychological – and the specific functions included in each that there was a question as to whether developing a harmonized framework is a valid undertaking. Given that this is the first iteration of the Framework, we believe that it has plausible content and construct validity which can be tested and refined in future research. Ultimately, however, we believe that it is the predictive validity of a model which is most crucial and most immediately addressable – what can the Framework and its

Table 2
Components of definitions of complex visual hallucinations.

Characteristics of visual hallucinations	Phenomena distinguished from	Challenges
A visual perception of something which is not out there in the visual environment	Veridical or illusionary perception of things which are in the visual environment.	Knowing what is truly out there in the visual environment depends upon what other people may, or may not notice, or subjective evidence from other senses (Aynsworth, Collerton, and Dudley, 2017). Even if the environment is known, the links between what is there and what is seen is not a one-to-one correspondence, as for example change blindness, the inability to perceive significant changes in the visual environment (Jensen, Yao, Street, and Simons, 2011), illustrates.
Which has the subjective quality of a veridical perception	Visual distortions	We do not have direct way of knowing whether someone else's perceptions appear real to them, other than by inference from what they say, or what they do (Aynsworth et al., 2017).
Which is outside of voluntary control	Voluntary visual imagery	A great deal of visual perception is outside of voluntary control (e.g.Prinzmetal, McCool, and Park, 2005), and some complex visual hallucinations are controlled, at least to a degree, directly or indirectly by the people experiencing them (Diederich, Pieri, and Goetz, 2003).
And which occurs when awake	Dreams	There is not a sharp dividing line between sleep and waking, and there are whole classes of complex visual hallucinations – hypnagogic and hypnopompic - which occur on the transitions between waking and sleeping (Azis et al., 2020).

application tell us about the future that we did not otherwise know? In our case, as we later show, what predictions can we derive from a new Visual Hallucination Framework that consideration of individual approaches do not generate?

Two lines of evidence increased our confidence that an overall framework might be feasible: firstly, each model explicitly aimed to account for the same subjective phenomenon - a complex visual hallucination – and secondly many functional concepts recurred in different models, suggesting at least some overlap in approaches.

In the event, we were able to derive a consensus Framework that was acceptable to all contributors via multiple recursive versions based on the interplay between drafts of the framework and the details of individual models, guided by the authors of those approaches and other contributors and based on a shared attempt to translate different concepts from varying paradigms into agreed cognitive functions such as attention, memory, or perception. We show the results in Fig. 2.

4. The Visual Hallucination Framework and understanding existing models

Though it was always believed by researchers that models overlapped to some extent, a crystalizing benefit of the Framework is that it gives far greater clarity than previously possible, not only to major similarities between approaches but also to contrasts. This allows easier comparisons between, or potentially integration of, theories.

4.1. Applying the Framework to each model

Fig. 3 shows the application of the Framework to each individual model. Each was fitted by the relevant research group in the first instance, then sense checked by other authors.

Fig. 3 shows how despite each of the eight models coming from different starting places, they can all be related to the Visual Hallucination Framework. These mappings highlight the benefits of fitting models to the same basic structure. Firstly, we can set boundaries around which cognitive systems are necessary and sufficient to include in causative models. Secondly, the Framework facilitates comparisons between models. Variations between which functions are included in different models give a straightforward means of deriving unique predictions from each. To give just one example, Perception and Attention Deficit (Fig. 3c) would predict that impairments in object but not spatial attention would increase the frequency of complex visual hallucinations, while Active Inference (Fig. 3g) would suggest that impaired modulating but not attentional orienting or object attention would increase frequency. Conversely, associations with sensory data would not distinguish between any of the models.

4.2. Applying each model to the Framework

We can go on to more directly compare models by putting them all on the same Framework. Fig. 4 shows the results. Doing so illustrates quite how wide a range of functions different models have included. There is a common thread in that every theory assigns an important role to the quality of sensory data, with impacts either from direct disturbances in the eye and visual pathways (Deafferentation, AIM, Hodological), or indirect effects via arousal (AIM, Attentional Networks) or via aspects of attention (AIM, Attentional Networks, Thalamocortical Dysrhythmia Default Mode Decoupling) on the acquisition (Active Inference) or representation (Active Inference, Perception and Attention Deficit) of visual information in cortex.

Outside of this overlap in the role of sensory data, causal factors that occur in more than one model are more variable.

For example, descending factors include overly precise and hence influential priors – due to excessive influence from intention/motivation/emotion expectations (as in Attentional Networks, Fig. 3f), or memories of previous hallucinations or other images (AIM, Fig. 3d) - perhaps in combination with a lack of influence of context data, due in turn to a failure to attend to the context (Attentional Networks) or an inability to perceive it due to sensory loss (Deafferentation, Fig. 3a).

Ascending factors include both a direct loss of data from the eye due to local disease (which might paradoxically increase the influence of sensory data through hyper-excitability of primary visual cortex, Deafferentation, Fig. 3a), or from environmental factors such as poor lighting or impoverished visual environments. Data might also be indirectly reduced due to impaired spatial attention (Attentional Networks), object attention (Perception and Attention Deficit, Fig. 3c), or both; caused either directly by impairments of attentional processing, or indirectly by reduced arousal (AIM). The influence of data might be reduced due to a breakdown within the process of relating perceptual priors to evidence (Perception and Attention Deficit).

Loss of connectivity (Hodological, Fig. 3e) or synchronisation (Thalamocortical Dysrhythmia Default Mode Decoupling, Fig. 3h) might mimic the effects of loss of functions by interrupting information flow.

In terms of the influence of sensory input on data comparison, one model (Perception and Attention Deficit) sees a central role for the comparison process per se, but others (Deafferentation, Hodological, Thalamocortical Dysrhythmia Default Mode Decoupling) suggest that the results of that comparison fail to modify what is being seen, while Active Inference implies potential effects at several levels. An inability to build current context from perception is a central feature of two models (Reality Monitoring, Thalamocortical Dysrhythmia Default Mode Decoupling), while limitations in the linkage of context, emotion, intention, and, particularly, memory feature in three (Reality Monitoring, Attentional Networks, AIM).

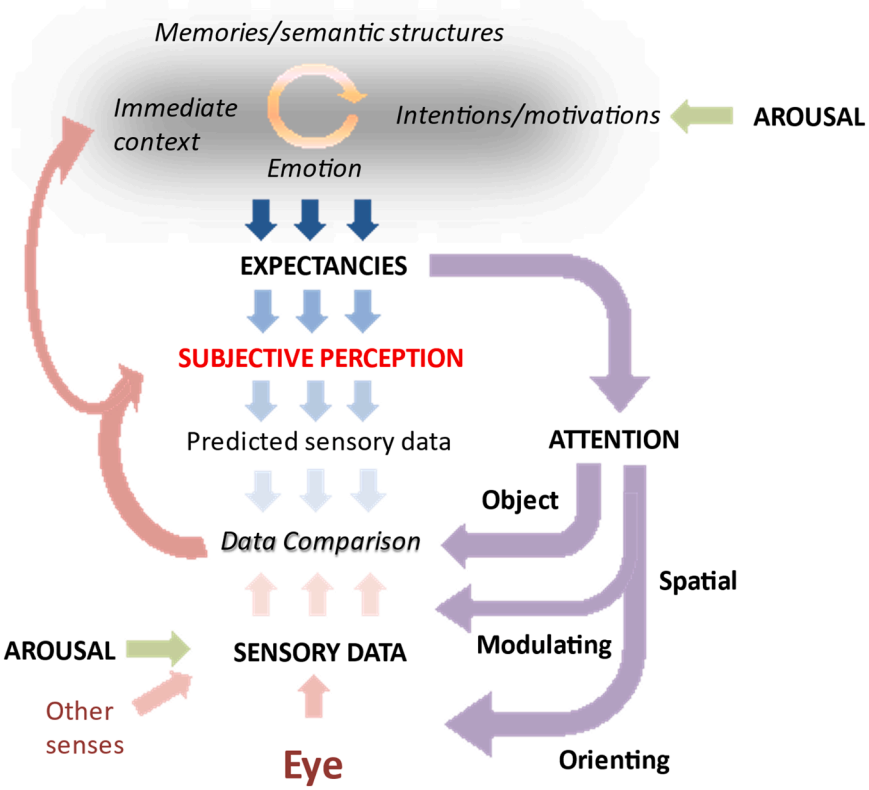

Fig. 2. Harmonised framework for aspects of visual perception relevant to hallucinations. Descending influences are shown in blue, and ascending in red. Horizontal influences are in green. The grey blur over memory, context, emotion, and intention is to indicate that these factors interact to create complex expectancies. Terms are defined in the text and Glossary. Linking arrows show the specfic relationships between concepts, bearing in mind that the influence of these varies dynamically instant by instant. The level of detail for each function was determined by the need to incorporate distinctions in exisiting theories. Thus, perception and attention are much more fractionated than memory or context. Similarly, variables in the Bayesian approach to perception are shown since they are central to theories of hallucinations, but those in other areas such are memory are not. Expectancies act to bias perception towards particular results. They can be generally conceptualised as brain states that reflect prior information about what is possible or probable in the forthcoming sensory environment: i.e. what is expected to be seen in that context (Parr, Rees, and Friston, 2018). In state models, predictions of sensory level data are central. Trait models, in contrast, have tended to emphasise higher order expectances which develop from an interaction between memories, both episodic and semantic, of past similar visual environments (which we can label as memory expectancies), the current visual context (context expectancies), future intentions and motivations (intentional and motivational expectancies), and emotions (emotional expectancies). As with perception, each of these functions has its own subfunctions, some of which are highlighted in specific models as seen in Fig. 3. Emphasising the circular interactions between these functions, the visual context is created from the data arising from current visual (and other sensory) perceptions. In addition to their direct role in modifying predicted data, we suggest that expectancies also indirectly affect sensory data gathering and processing via attention: a central feature of most theories of hallucinations. Attention prioritizes that subset of visual data deemed to be of the highest relevance to the organism's expectancies, thus modulating the quality, and prediction of quality, of ascending visual signals. We have separated attention into two main components, each of which has specific effects on different aspects of sensory data: object attention which primarily acts on the processing of already gathered data, and spatial attention which both influences the acquisition of sensory data and its subsequent processing (Parr and Friston, 2019; Summerfield and Egner, 2009). Spatial attention needs to be further fractionated into two intertwined aspects, attentional orienting and attentional modulation. Attentional orienting directs vision by eye and other movements towards expected places in the visual context where the sensory evidence which is most relevant to testing expected data can be gathered. Attentional modulation uses covert mental saccades to increase or reduce 'gain' to parts of the visual field, without moving the eyes. The place of arousal (a complex concept related to level of consciousness and the sleep-wake cycle and a key factor in clinical models of hallucinations (e.g., Diederich et al., 2005; Manford and Andermann, 1998) is more uncertain in the cognitive literature. It impacts on processing at multiple levels and potentially has a bimodal influence: extremely high or low levels of arousal may be particularly associated with hallucinatory episodes. In visual perception, arousal can bias processing in favour of goal-relevant or perceptually conspicuous stimuli (Mather and Sutherland, 2011). We therefore suggest that its primary effects relevant to hallucinations are at the level of intentional expectancies acting on attention, and on prediction errors. Thus, being very under or over aroused may lead to combined effects on expectancies and on sensory data, potentially accounting for the relationship of visual hallucinations to sleep disorders (Goetz et al., 2010) and sleep transitions (Azis, Ristanovic, and Mittal, 2020) - while maintaining a distinction between hallucinations and dreams - and delirium (Webster and Holroyd, 2000). Information flow through these structures is extremely dynamic with high frequency updating of perceptions and data and rapid variation in the contributions of different components of the Framework over a range of timescales (Hasson, Yang, Vallines, Heeger, and Rubin, 2008; Kiebel, Daunizeau, and Friston, 2008; Perdikis, Huys, and Jirsa, 2011). The synchronised functioning of these components depends on a variety of mechanisms, including thalamocortical interactions (McCormick, McGinley, and Salkoff, 2015; Shine, 2021) with desynchrony producing dysfunctional information flow (Corlett, 2019; Onofrj et al., 2019; Shine et al., 2011, 2014,).

This particular pattern of partial overlaps in models highlights two extreme possibilities. On the one hand, there might be ***no*** consistent single or unifying cause of visual hallucinations even those with the same content. Instead there are many triggering pathways in different disorders or circumstances, all of which lead to hallucinations as a final outcome. Hence, the scientific challenge would be to identify which cause or pathway is active in a specific instance with the clinical implication that treatments would be highly specific. The alternate extreme would be that there is **only one** common final pathway for each type of complex visual hallucination but that the scattering of research field has obscured this cause: regardless of the type of precipitant, once activated, hallucinations have a single anatomical or functional connectome. Hence, a treatment that blocks entry to that pathway would stop hallucinations regardless of precipitant. If this latter possibility is the correct one, then we have to take up the challenge of identifying which existing model or fusion of models best captures that causal path.

We suggest that there may be aspects of truth in both extremes: there may be a common final pathway that leads to the occurrence of a particular type of complex hallucination, a face or animal for example, but there are also factors that account for variations in phenomenology among people and disorders. If correct, this would suggest a shift in research strategies towards investigating not only group commonalities but also individual variations relevant to hallucinations.

The clustering of models around central roles for visual data and its comparison with predicted data in causing a hallucination suggests that this area is the best candidate for a common pathway and that exploration of these functions and of their underlying neurobiology in the early visual cortex and the ventral visual stream will prove fruitful.

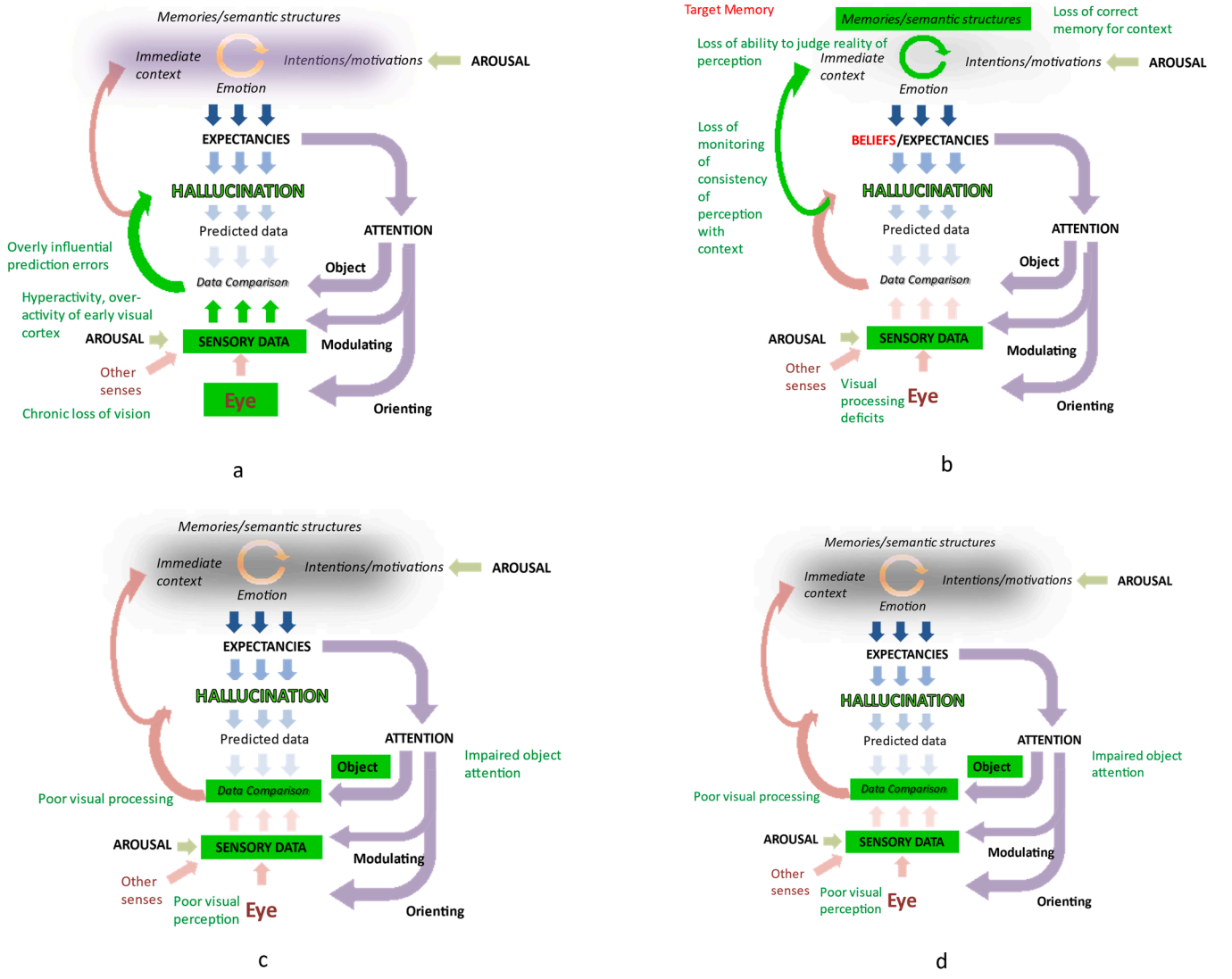

Fig. 3. Details of specific models and their relationship to the Framework. Green shading indicates which aspects of the Framework are mapped to in each model, with the green text indicating the specific effects on functions. 3a Deafferentation. Functional mechanism: false sensory data becomes overly influential, overriding the results of data comparison and expectancies to become incorporated into a hallucinatory perception. The hallucinated perception is replaced by a veridical one when the strength of the false sensory evidence reduces, or expectancies and data comparison override it. Components of framework mapped to: sensory data from the eye and data comparison Neurobiological underpinnings: false sensory data results from hyper-excitability and spontaneous activation of the primary visual cortex caused by chronic loss of visual input. 3b Reality Monitoring Functional mechanism: a combination of visual processing deficits and episodic memory impairment, combined with source-monitoring errors. Components of framework mapped to: sensory data, and memory/semantic structures. The model distinguishes between belief and expectation. Belief is a mental acceptance of something, regardless of supporting empirical evidence: resulting in the mental claim that what is seen is true. Expectation is the act or state of expecting an event as about to happen which could then be processed as true or untrue. Neurobiological underpinnings: frontal (source monitoring), medial temporal (memory), and ventral visual (visual processing) cortices. 3c Perception and Attention Deficit. Functional mechanism: sensory evidences are relatively uninfluential due to failures in visual perception in combination with more imprecise data comparison due to a failure in object attention. An image (the hallucination) which is least inconsistent with expectancies and expected perceptions is "seen". This hallucination then persists since it generates the immediate context which in turn reinforces expectancies which are most consistent with the hallucination. That hallucination is then not modified to something more veridical because of imprecise data comparison. Components of model mapped to: object attention, sensory data, and the data comparison. Neurobiological underpinnings: dorsolateral frontal cortex (object attention), ventral visual stream (visual perception). 3d AIM. Functional mechanism: poor visual processing in combination with defective central monitoring and impaired attentional focus leads to deblocking of visual memories. Components of framework mapped to: sensory data from the eye, arousal, attention, and memory/semantic structures. Neurobiological underpinnings: Parkinson's disease related retinopathy and functional alterations of extra-striate visual pathways (visual processing), brainstem pathology (disordered sleep-wake and dream regulation systems leading to impaired attention and memory control). 3e Hodological. Functional mechanism: transient topological (cortical area based) activity occurs at the time of a visual hallucination within the context of transient or persistent hodological (cortical connectivity based) factors such as hypo-or hyper-connectivity between brain regions. Components of framework mapped to: messaging between functions. Neurobiological underpinnings: cortical connectivity. 3 f Attentional Networks. Functional mechanism: heightened arousal leads to an upregulated Ventral Attention Network (VAN). Concurrent sensory data errors arise from poor sensory evidences as a result of pathological changes not only in the eye but also across visuospatial processing regions of the brain. Concurrently, there is an increased level of activity within the Default Mode Network (DMN) leading to increased self-referential expectancies with impaired cognition impacting upon the recall of accurate memories. During a hallucination, the usual VAN and DMN integration with the Dorsal Attention Network (DAN) which would coordinate exogenous attention is lost and there is increased coupling between the DMN and the visual network allowing the perception of 'false' perceptions to be experienced. At the termination of the hallucination, normal function of the DAN is restored, suppressing activity in the VAN and DMN so that exogenous attention is again functional. Components of framework mapped to: sensory data, arousal, attention, and expectancies. Neurobiological underpinning: eye and visual cortex (sensory data), connectivity of

attentional networks (expectancies, attention, and arousal). 3 g Active Inference. Functional mechanism: an imbalance in the precision of (confidence attached to) priors (expected perceptions) and likelihoods (chance of specific sensory data associated with that perception) allows a false inference to be sustained in the absence of confirmatory sensory data. This occurs either through an over confident prior belief, or by sensory data being discounted if likelihood is overly imprecise such that prediction errors can be ignored. Although precise data may be available, if the brain ascribes a sufficiently low precision (i.e., attends away) from these data, the resulting percepts will be formed as if no precise data were available. Components of framework mapped to: expectancies and sensory data. Spatial attention is particularly relevant in its effect on modulation of gain, rather than orientation. Neurobiological underpinnings: multiple potential processes. In Charles Bonnet syndrome, primary eye disease deprives the brain of precise visual data, leaving prior beliefs the sole arbiters of visual perception. In Lewy body disease, the same mechanism may be at play at higher levels of sensory processing. For instance, impaired cholinergic signalling might reduce the precision assigned to parts of the visual stream, partially decoupling visual perception from sensory input. 3 h Thalamocortical Dysrhythmia Default Mode Network Decoupling hypothesis. Functional mechanism: The frontal attentional network is inhibited, favouring decoupling of the Default Mode Network and leading to random formation of connections that link strong autobiographical correlates to trivial stimuli. Components of framework mapped to: attention, memory, expectancies, and sensory data Neurobiological underpinnings: thalamocortical dysrhythmia.

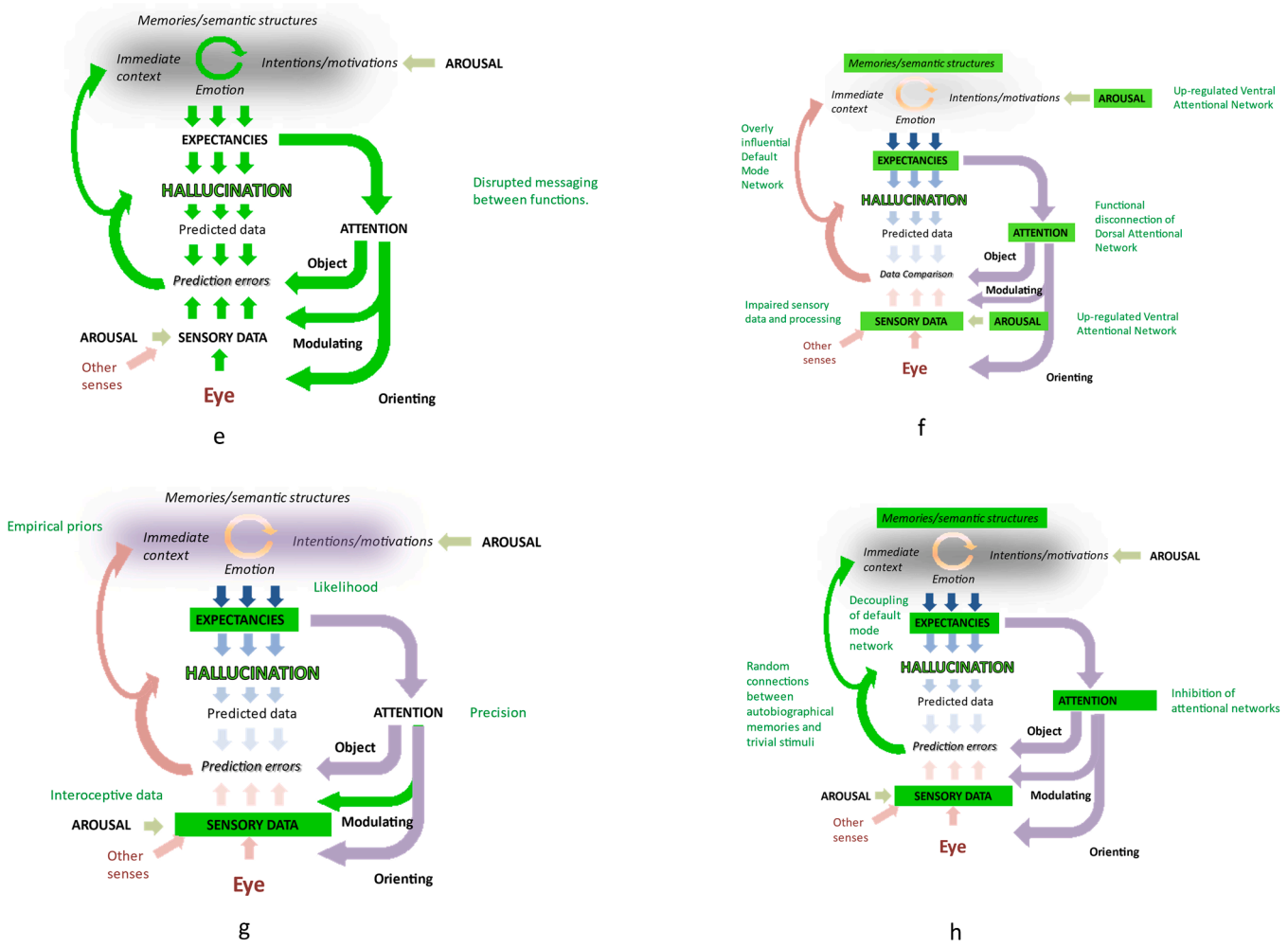

Fig. 3. (*continued*).

Complementing this commonality, the relative scattering of models in terms of higher-level influences might suggest a more varied or individual contribution from those factors. Thus, hallucinations of known people in familiar settings experienced in Parkinson's disease (Urwyler et al., 2014) may be related to context and memory, while the bizarre and distorted hallucinations of eye disease (Santhouse et al., 2000) may suggest a particular contribution from partial or distorted context expectancies influenced by limited sensory data.

Higher level influences may also be relevant to extending the Framework to account for hallucinations with more than just a visual component. In contrast to the visual hallucinations in Lewy body disorders and eye disease (90% plus of which are unimodal, at least in those disorders' earlier stages (Dudley et al., 2019)), those seen in other disorders are much more often accompanied by hallucinations in other sensory domains (Montagnese et al., 2021), sometimes simultaneously. The mechanisms underlying simultaneous multimodal hallucinations are not well understood, but could reflect common expectancies across modalities or cross modality priming (Fernyhough, 2019). Hence, the association in schizophrenia between multimodal hallucinations, psychological trauma and distress (David et al., 2011; Medjkane et al., 2020) may in this disorder indicate a strong role for the memory and emotion expectancies in the Framework.

5. A sharper focus on the dynamics of hallucinatory episodes

If there is a final common pathway, it will only be fully active during the hallucinatory state, even if aspects of it persist outside hallucinatory episodes as trait factors (Goetz et al., 2014; Stebbins et al., 2004).

Testing which traits are associated with hallucinations is relatively straightforward since participants can be assessed at any time, though there are unresolved questions about the relative contributions of acute and chronic impairments and compensations at the mechanistic level

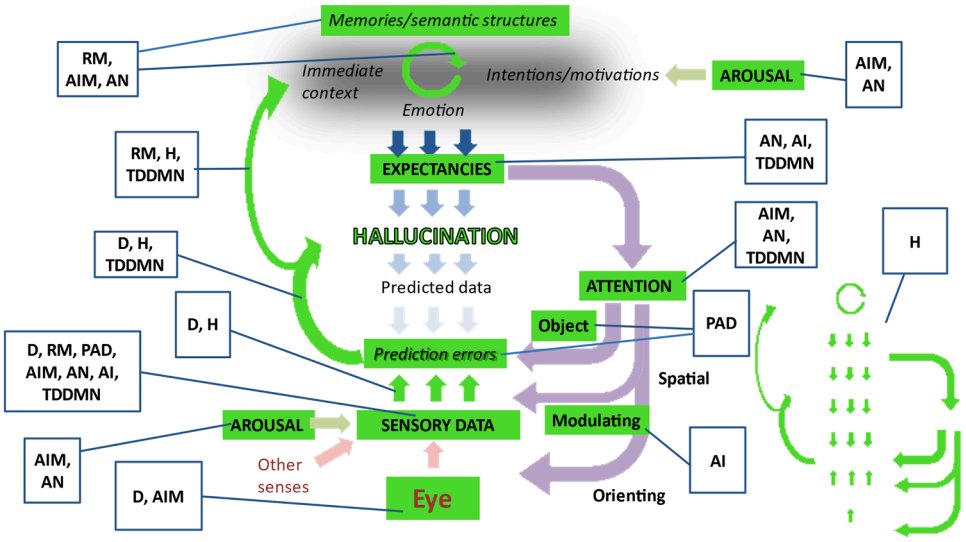

Fig. 4. Existing models and the framework. For clarity the Hodological model is shown separately where it does not overlap with other models since it focusses on connecting tracts rather than functions. AI, Active Inference; AIM, Activation-Input-Modulation; AN, Attentional Networks; D, Deafferentation; H, Hodological; PAD, Perception and Attention Deficit; RM, Reality Monitoring; TDDMN, Thalamocortical Dysrhythmia Default Mode Network Decoupling. Relevant functions for models of hallucinations are highlighted in green and labelled by the models which feature them, accepting that the roles of specific functions may differ in detail.

(Collerton et al., 2005; Bowman, Bruce, Colbourn, and Collerton, 2017), and whether initial and subsequent hallucinations are generated in the same way; given that repeated content may create memory expectances which increase the chance of their reoccurrence.

Assessing the functional changes associated with the dynamics of hallucinatory episodes is much more challenging given the relative unpredictability of specific episodes for most people, and that hallucinations' responsiveness to the environment tends to mean that they disappear in experimental settings (Dujardin et al., 2020). Much of the data in this area therefore comes from participants who have unusually consistent hallucinations and who can therefore be assessed outside and within hallucinatory episodes (ffytche et al., 1998; Goetz et al., 2014), or from hallucination-like phenomena such as pareidolia and other misperceptions, which can be experimentally induced (Shine et al., 2014; Bowman et al., 2017).

5.1. A key role for transition processes

The episodic nature of hallucinations confirms that the traits associated with their occurrence are necessary but insufficient to account for why hallucinatory states occur. Otherwise, hallucinations would be continually present, which in the great majority of cases, they are not. That most perception, even in people who hallucinate and even during hallucinations (Collerton et al., 2005), is veridical suggests that there is some transient disturbance of processing additional to traits which is associated with hallucinatory episodes. That even a "normal" visual system can experience hallucinations in unusual circumstances such as sensory deprivation (Mason and Brady, 2009) or bereavement (Castelnovo et al., 2015) or very occasionally for no apparent cause (Vellante et al., 2012) suggests that periodic instability in visual perception is inescapable. Hence, understanding hallucinatory states is underpinned by understanding how the perceptual system can move from a state of stable veridical perception through a destabilized state to stable hallucinatory perception and then via further destabilization back to stable veridical perception (Corlett et al., 2019; Series, Reichert, and Storkey, 2010).

Veridical transitions within the highly dynamic visual system have two requirements which may also be relevant to a transition from veridical to hallucinatory perception. Firstly, visual perception has to both capture stability and allow change; for example, seeing the same person from different angles as they move around, or seeing something new in an existing visual scene. Secondly, the subjective experience of continuous veridical perception has to be maintained despite gaps in sensory input caused, for example, by eye movement. Together, these factors suggest that there are resilient mechanisms that maintain continuity from one veridical perception to another which must break down in hallucinations (Ross, Morrone, Goldberg, and Burr, 2001). Understanding these mechanisms will be central to understanding how hallucinatory episodes can occur.

5.2. Questions on onset, persistence, and end

Taken together with the proposal that veridical perception is maintained by a continual transformation from what has been seen to what is seen, this emphasis on hallucinatory episodes encourages a move from the general question of how hallucinations occur towards three separate questions which address firstly the onset, secondly the persistence, and thirdly, the end of hallucinatory episodes. Hence, we need to investigate those dynamical changes within the Framework which are associated with sequential phases of hallucinations.

Thus, we seek to understand:

1. Under which conditions can a discontinuity arise within the process of modifying what was veridically seen, such that hallucinatory content takes the place of the next potential veridical perception?
   Referring to Fig. 2 suggests further areas of investigation:
   a. The discontinuity may lie in the transition process per se (the mechanisms that adjudicate between what has been seen and what is to be seen), or in the contributions of what was previously seen, or of visual data to an intact process, or all of these?
   b. Is the process active (another factor knocks out the veridical perception), or is it is passive (another factor fills the void left by the absence of the veridical perception)?
   c. Which factors influence the form of the non-veridical perception? To what extent are these determined by expectations, or by sensory data?
2. How does the hallucinatory perception become excessively influential relative to sensory or other evidence and, or to veridical expectancies for the period that the hallucination is maintained?
3. Under what conditions does the hallucinatory perception become modified to be replaced by a veridical perception? And as with onset:
   a. Is the discontinuity dependent on the transition process per se, or in the contributions of what was previously seen, or of visual data to an intact process, or all of these?
   b. Is the process active (another factor knocks out the hallucinatory perception), or is it passive (another factor fills the void left by the absence of the hallucinatory perception)?

Answering these questions will cast light on variations in the frequency and duration of hallucinatory episodes, and on the relationship between trait and state factors.

Importantly, as illustrated later in the discussion of the role of neurotransmitters, longstanding traits which increase the chance of hallucinatory episodes may be differentially associated with the onset, persistence, and end of specific occurrences, and hence have different relationships with transitions between veridical and hallucinatory states.

5.3. Gaps in current knowledge

Posing these questions highlights gaps in our current knowledge of the phenomenology of visual hallucinations. Critically, very little is known about the subjective experience of transitions into and out of hallucinatory episodes. Are there intermediate perceptual forms between the veridical and hallucinatory which any model would need to account for? If so, is there any relationship that these forms may have to the phenomena such as illusions and pareidolia that are often associated with hallucinations (Fazekas, 2021). In addition, linkages to simultaneous transitions in wider systems such as arousal or alertness need to be investigated. Trait factors would suggest that these are likely to be significant.

Consideration of the Framework suggests temporal, structural, and dynamical constraints that are relevant in understanding and hence modelling hallucinatory episodes.

In terms of timescales, perceptual updating occurs around every 100 ms (Marom, 2010). Hence, the relatively rare onset of hallucinations even in people who are prone to them and the usual duration of hallucinatory episode of seconds to minutes, together suggest that triggers for the transition to or from hallucinations are highly infrequent in comparison to factors that maintain either veridical or hallucinatory perception.

The restricted but distributed structural pathology associated with hallucinatory traits (Erskine et al., 2019) implies that the functioning of partially lesioned systems is critical in understanding their occurrence. In providing an overall set of relationships for the key functions relevant to hallucinations, in conjunction with neuropathological or imaging data, the Framework facilitates modelling of these types of systems without being either over or under inclusive.

The highly complex, nonlinear perceptual system has a capacity for unstable, chaotic dynamics if normal compensatory mechanisms are disturbed. Hence, dynamical modelling has to address how transitions may occur in these types of systems (Tsuda, Yamaguti, and Watanabe, 2016). As an instance, transient fluctuations in distributed neuromodulatory systems may shift functioning past a critical level into a dynamic far-from-equilibrium hallucinatory state (Collerton et al., 2016; Tsukada, Fujii, Aihara, and Tsuda, 2015); and in Parkinson's patients, those with visual hallucinations show differences in dynamic transitions between states compared with patients without visual hallucinations (Zarkali et al., 2021). Computational simulations suggest that lesioned systems can switch from transitions between attractor states (analogous to a transitioning from one perception to another) to remaining in one state (analogous to being stuck in a hallucinatory episode) depending on the overall activity of the system (Fujii, Tsukada, Tsuda, and Aihara, 2015; Nara, Fujii, Tsukada, and Tsuda, 2019, 2022). This formulation indicates that the processes driving states may be more distributed than the traits which make them likely, and places emphasis on the mechanisms by which the brain maintains the synchronised functioning of different parts of the Framework (Onofrj et al., 2019).

6. Relationships with brain function

Though this framework is functional rather than neurobiological, since it incorporates classic neuropsychological functions, relationships to brain systems can be sketched out.

The details of the models in Fig. 3 illustrate how arousal, sensory data, expectancies, and attention have been mapped to specific brain perceptual and cognitive systems by individual research groups. For example, Perception and Attention Deficit highlights the ventral visual stream and Attentional Networks, the eye and early visual cortex. As would be expected from the range of functions included in different models, relevant brain systems also vary to some degree, though there is consistent inclusion of early visual cortex (Deafferentation, Reality Monitoring, Attentional Networks, Active Inference), the dorsal (Deafferentation, Attentional Networks) and ventral (Reality Monitoring, Perception and Attention Deficit, AIM, Attentional Networks, Active Inference) visual streams, together with frontal (Reality Monitoring, Perception and Attention Deficit, Attentional Networks) and medial temporal (Reality Monitoring, AIM, Attentional Networks) cortex and their interconnectors (Hodological) and regulators (Thalamocortical Dysrhythmia Default Mode Decoupling).

In terms of neuromodulatory systems, the key neurotransmitters associated with hallucinations, acetylcholine, dopamine, and serotonin (Russo et al., 2019), have distinct roles within the Framework and may have particular relevance to different phases of hallucinatory episodes.

Acetylcholine has been associated with the precision of sensory and other signals (Marshall et al., 2016; Moran et al., 2013; Warburton, Wesnes, Edwards, and Larrad, 1985) implying that dysfunction may account for a failure to update prior beliefs when sensory evidence is inconsistent with visual percepts, with potentially particular relevance to the persistence of a hallucination. Dopaminergic neurones are known to play a key role in computing and signalling prediction errors (Schultz, Dayan, and Montague, 1997; Watabe-Uchida, Eshel, and Uchida, 2017) and in encoding the salience of environmental stimuli (Diederen and Fletcher, 2021; Schultz, 2016). Thus, abnormal dopamine signals can produce an enhanced significance of external stimuli (Kapur, 2003) and increase the salience of an environmental stimulus by giving a small prediction error much larger weight (Fletcher and Frith, 2009). Hence, dopaminergic function might be especially relevant in the onset and end of hallucinations - consistent with functional changes in anterior insular cortex that predate hallucination onset (de Pierrefeu et al., 2018). Serotonin modulates early sensory processing as well as behavioural responses to visual inputs (Jacob and Nienborg, 2018; Nour and Carhart-Harris, 2017) and agents reducing transmission at serotonergic receptors are known to be effective in suppressing visual hallucinations (Zoldan, Friedberg, Livneh, and Melamed, 1995) while serotonergic agonists can induce them (Stahl, 2018), potentially suggesting a role in transitions to and from veridical vision.

7. Research priorities

One of the most quickly realised benefits of an integrated framework is that it allows existing data to be analysed in a consistent way. In the future, it facilitates a number of different avenues of research with complementary strengths to come together with a common language and structure so that simultaneous predictions can be made at several explanatory levels. For instance, a link between impaired attentional orienting and hallucinations should be seen in poor performance on neuropsychological tests of spatial attention and in reduced activation in relevant attentional networks on functional imaging during spatial attentional tasks, as well as being able to be formally modelled as reduced precision in the Active Inference theory of hallucinations.

More generally, phenomenological research in conjunction with experimental cognitive tasks and functional imaging can link the character of specific hallucinations to changes in particular functions such as arousal, memory, or attention (e.g. Zmigrod et al., 2016 or Ćurčić-Blake et al., 2017 from the perspective of auditory hallucinations). Comparisons of hallucinatory perceptions with voluntary and veridical images with the same content can clarify the relationships between these forms of perception. Studies of the structural pathology of disorders with high levels of hallucinations can produce evidence of chronic constraints on

the visual system (e.g. O'Brien et al., 2005). The effects of these constraints can be modelled in computational approaches to understand the dynamics of hallucinatory perception (e.g., Collerton et al., 2016; Fujii et al., 2015; Jardri and Denève, 2013; Nara et al., 2019, 2022; Tsukada et al., 2015). Pharmacological (e.g. Russo et al., 2019) or environmental (e.g. O'Brien et al., 2020) manipulation of hallucinations in conjunction with functional imaging of hallucinatory episodes (e.g. ffytche et al., 1998; Goetz et al., 2014) can probe transition processes.

8. Clinical implications

This Hallucination Framework has clinical implications for treating hallucinations when they are clinically pertinent or distressing (Collerton and Taylor, 2013). A lack of effective treatments in combination with a reluctance to discuss hallucinations (Menon, 2005) has led to a substantial burden on those who experience them, and on the people around them.

As we indicate in the section on neurotransmitters, different factors may be relevant in the onset, offset, or persistence of hallucinations. The Framework allows a strategic approach to investigating potential treatments; in particular whether combining interventions aimed at different components has synergistic benefits. Improving the amount or quality of visual data through addressing eyesight, attention, or arousal; improving alertness; modifying context expectancies through changing the visual environment; or reducing the contributions of memory expectancies are all thought to have positive effects in at least some cases (Barnes, Connelly, Boubert, and Maravic, 2013; Diederich et al., 2003; Falloon and Talbot, 1981; Singh, Sharan, and Kulhara, 2003), as do some medications (Collerton and Taylor, 2013). However, what is not yet known is whether each provides broadly the same benefit since they all target a common hallucination pathway, or, as seems more probable given their putative mechanisms, whether each affects different components of the pathway with potential additive benefits. In any event, providing acceptable, credible, benign explanations based upon sound science for these phenomena could be effective in reducing stigma and distress (Barnes et al., 2013; Diederich et al., 2003; Falloon and Talbot, 1981; Singh et al., 2003).

9. In summary

In order to reconcile different approaches to explaining visual hallucinations, we have developed a new conceptual Visual Hallucination Framework. Its specificity and inclusiveness have set initial boundaries around relevant cognitive systems and will allow theories to be more easily compared or integrated in the future. The Framework provides a structure for investigating potential common and individual causative pathways, and in putting a new focus on the mechanisms of hallucinatory episodes, suggests potential temporal, structural, and dynamic constraints in simulating these.

We have also perforce produced a pragmatic consensus view of the various components of visual perception and how these relate to each other. This clinically driven development of theory has analogies with how research on aphasia has refined models of language (Vasishth et al., 2019), and studies of amnesia those of memory (Squire and Zola, 1997) and suggests that the interplay between investigation of clinical disorders of vision and normal human perception is equally fruitful.

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References

Aarsland, D., Larsen, J.P., Tandberg, E., Laake, K., 2000. Predictors of nursing home placement in Parkinson's disease: a population-based, prospective study. J. Am. Geriatr. Soc. 48, 938–942.

Aynsworth, C., Collerton, D., Dudley, R., 2017. Measures of visual hallucinations: review and recommendations. Clin. Psychol. Rev. 57, 164–182.

Azis, M., Ristanovic, I., Mittal, V.A., 2020. Hypnagogic and hypnopompic hallucinations: Considerations for clinical high-risk assessment and targets for future research. Schizophr. Res. 222, 514–515.

Barnes, J., David, A.S., 2001. Visual hallucinations in Parkinson's disease, a review and phenomenological survey. J. Neurol., Neurosurg. Psychiatry 70, 727–733.

Barnes, J., Boubert, L., Harris, J., Lee, A., David, A.S., 2003. Reality monitoring and visual hallucinations in Parkinson's disease. Neuropsychologia 41, 565–574.

Barnes, J., Connelly, V., Boubert, L., Maravic, K., 2013. Behavioural coping patterns in Parkinson's patients with visual hallucinations. J. Neuropsychol. 7, 326–334.

Benke, T., 2006. Peduncular hallucinosis. J. Neurol. 253, 1561–1571.

Benrimoh, D., Parr, T., Adams, R.A., Friston, K., 2019. Hallucinations both in and out of context: an active inference account. PLoS One 14, e0212379.

Berrios, G.E., Markova, I.S., 2015. Visual hallucinations: history and context of current research. In: Collerton, D., et al. (Eds.), *The Neuroscience of Visual Hallucinations*. Wiley Blackwell, pp. 1–22.

Blom, J.D., 2015. Defining and measuring hallucinations and their consequences – what is really the difference between a veridical perception and a hallucination? Categories of hallucinatory experiences. In: Collerton, D., et al. (Eds.), *The Neuroscience of Visual Hallucinations*. Wiley Blackwell, pp. 23–45.

Bowman, A.R., Bruce, V., Colbourn, C.J., Collerton, D., 2017. Compensatory shifts in visual perception are associated with hallucinations in Lewy body disorders. Cogn. Res.: Princ. Implic. 2, 1–9.

Brown, H.R., Friston, K.J., 2012. Dynamic causal modelling of precision and synaptic gain in visual perception—an EEG study. Neuroimage 63, 223–231.

Burke, W., 2002. The neural basis of Charles Bonnet hallucinations, a hypothesis. J. Neurol., Neurosurg. Psychiatry 73, 535–541.

Castelnovo, A., Cavallotti, S., Gambini, O., D'Agostino, A., 2015. Post-bereavement hallucinatory experiences: a critical overview of population and clinical studies. J. Affect. Disord. 186, 266–274.

Chouinard, V.A., Shinn, A.K., Valeri, L., Chouinard, P.A., Gardner, M.E., Asan, A.E., Öngür, D., 2019. Visual hallucinations associated with multimodal hallucinations, suicide attempts and morbidity of illness in psychotic disorders. Schizophr. Res. 208, 196–201.

Collerton, D., Taylor, J.P., 2013. Advances in the treatment of visual hallucinations in neurodegenerative diseases. Future Neurol. 8, 433–444.

Collerton, D., Perry, E., McKeith, I., 2005. Why people see things that are not there: a novel perception and attention deficit model for recurrent complex visual hallucinations. Behav. Brain Sci. 28, 737–757.

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.

Corlett, P.R., Horga, G., Fletcher, P.C., Alderson-Day, B., Schmack, K., Powers III, A.R., 2019. Hallucinations and strong priors. Trends Cogn. Sci. 23, 114–127.

Ćurčić-Blake, B., Ford, J.M., Hubl, D., Orlov, N.D., Sommer, I.E., Waters, F., Aleman, A., 2017. Interaction of language, auditory and memory brain networks in auditory verbal hallucinations. Prog. Neurobiol. 148, 1–20.

David, C.N., Greenstein, D., Clasen, L., Gochman, P., Miller, R., Tossell, J.W., Rapoport, J.L., 2011. Childhood onset schizophrenia: high rate of visual hallucinations. J. Am. Acad. Child Adolesc. Psychiatry 50, 681–686.

Diederen, K.M., Fletcher, P.C., 2021. Dopamine, prediction error and beyond. Neuroscientist 27, 30–46.

Diederich, N.J., Pieri, V., Goetz, C.G., 2003. Coping strategies for visual hallucinations in Parkinson's disease. Mov. Disord. 18, 831–832.

Diederich, N.J., Goetz, C.G., Stebbins, G.T., 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.

Dudley, R., Wood, M., Spencer, H., Brabban, A., Mosimann, U.P., Collerton, D., 2012. Identifying specific interpretations and use of safety behaviours in people with distressing visual hallucinations: an exploratory study. Behav. Cogn. Psychother. 40, 367–375.

Dudley, R., Aynsworth, C., Cheetham, R., McCarthy-Jones, S., Collerton, D., 2018. Prevalence and characteristics of multi-modal hallucinations in people with psychosis who experience visual hallucinations. Psychiatry Res. 269, 25–30.

Dudley, R., Aynsworth, C., Mosimann, U., Taylor, J.P., Smailes, D., Collerton, D., Urwyler, P., 2019. A comparison of visual hallucinations across disorders. Psychiatry Res. 272, 86–92.

Dujardin, K., Roman, D., Baille, G., Pins, D., Lefebvre, S., Delmaire, C., Jardri, R., 2020. What can we learn from fMRI capture of visual hallucinations in Parkinson's disease. Brain Imaging Behav. 14, 329–335.

Erskine, D., Taylor, J.P., Thomas, A., Collerton, D., McKeith, I., Khundakar, A., Morris, C., 2019. Pathological changes to the subcortical visual system and its relationship to visual hallucinations in dementia with Lewy bodies. Neurosci. Bull. 35, 295–300.

Falloon, I.R., Talbot, R.E., 1981. Persistent auditory hallucinations: coping mechanisms and implications for management. Psychol. Med. 11, 329–339.

Fazekas, P., 2021. Hallucinations as intensified forms of mind-wandering. Philos. Trans. R. Soc. B 376, 20190700.

Fénelon, G., Mahieux, F., Huon, R., Ziégler, M., 2000. Hallucinations in Parkinson's disease: prevalence, phenomenology and risk factors. Brain 123, 733–745.

Fernyhough, C., 2019. Modality-general and modality-specific processes in hallucinations. Psychol. Med. 49, 2639–2645.

ffytche, D.H., 2005. Visual hallucinations and the Charles Bonnet syndrome. Curr. Psychiatry Rep. 7, 168–179.

ffytche, D.H., 2008. The hodology of hallucinations. Cortex 44, 1067–1083.

ffytche, D.H., Howard, R.J., Brammer, M.J., David, A., Woodruff, P., Williams, S., 1998. The anatomy of conscious vision: an fMRI study of visual hallucinations. Nat. Neurosci. 1, 738–742.

Fletcher, P.C., Frith, C.D., 2009. Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. Nat. Rev. Neurosci. 10, 48–58.

Friston, K.J., 2005. Hallucinations and perceptual inference. Behav. Brain Sci. 28, 764–766.

Friston, K.J., 2017. Precision psychiatry. Biol. Psychiatry.: Cogn. Neurosci. Neuroimaging 2, 640–643.

Fujii, H., Tsukada, H., Tsuda, I., Aihara, K., 2015. Visual hallucinations in dementia with Lewy bodies (I): a hodological view. Advances in Cognitive Neurodynamics (IV). Springer,, Dordrecht, pp. 441–445.

Gauntlett-Gilbert, J., Kuipers, E., 2005. Visual hallucinations in psychiatric conditions: appraisals and their relationship to distress. Br. J. Clin. Psychol. 44, 77–87.

Goetz, C.G., Ouyang, B., Negron, A., Stebbins, G.T., 2010. Hallucinations and sleep disorders in PD: ten-year prospective longitudinal study. Neurology 75, 1773–1779.

Goetz, C.G., Stebbins, G.T., Ouyang, B., 2011. Visual plus nonvisual hallucinations in Parkinson's disease: development and evolution over 10 years. Mov. Disord. 26, 2196–2200.

Goetz, C.G., Vaughan, C.L., Goldman, J.G., Stebbins, G.T., 2014. I finally see what you see: Parkinson's disease visual hallucinations captured with functional neuroimaging. Mov. Disord. 29, 115–117.

Hasson, U., Yang, E., Vallines, I., Heeger, D.J., Rubin, N., 2008. A hierarchy of temporal receptive windows in human cortex. J. Neurosci. 28, 2539–2550.

Holroyd, S., Currie, L., Wooten, G.F., 2001. Prospective study of hallucinations and delusions in Parkinson's disease. J. Neurol., Neurosurg. Psychiatry 70, 734–738.

Ibarretxe-Bilbao, N., Ramirez-Ruiz, B., Junque, C., Marti, M.J., Valldeoriola, F., Bargallo, N., Tolosa, E., 2010. Differential progression of brain atrophy in Parkinson's disease with and without visual hallucinations. J. Neurol., Neurosurg. Psychiatry 81, 650–657.

Jacob, S.N., Nienborg, H., 2018. Monoaminergic neuromodulation of sensory processing. Front. Neural Circuits 12, 51.

Jardri, R., Denève, S., 2013. Computational models of hallucinations. In: Jardri, R., Cachia, A., Thomas, P., Pins, D. (Eds.), The Neuroscience of Hallucinations. Springer, New York, NY, pp. 289–313.

Jensen, M.S., Yao, R., Street, W.N., Simons, D.J., 2011. Change blindness and inattentional blindness. Wiley Interdiscip. Rev.: Cogn. Sci. 2, 529–546.

Kapur, S., 2003. Psychosis as a state of aberrant salience, a framework linking biology, phenomenology, and pharmacology in schizophrenia. Am. J. Psychiatry 160, 13–23.
Kiebel, S.J., Daunizeau, J., Friston, K.J., 2008. A hierarchy of time-scales and the brain. PLoS Comput. Biol. 4, e1000209.
Leu-Semenescu, S., De Cock, V.C., Le Masson, V.D., Debs, R., Lavault, S., Roze, E., Arnulf, I., 2011. Hallucinations in narcolepsy with and without cataplexy: contrasts with Parkinson's disease. Sleep. Med. 12, 497–504.
Llebaria, G., Pagonabarraga, J., Martínez-Corral, M., García-Sánchez, C., Pascual-Sedano, B., Gironell, A., Kulisevsky, J., 2010. Neuropsychological correlates of mild to severe hallucinations in Parkinson's disease. Mov. Disord. 25, 2785–2791.
Macpherson, F., 2013. The philosophy and psychology of hallucination: an introduction. In: Macpherson, F., et al. (Eds.), Hallucination: Philosophy and Psychology. MIT Press, pp. 1–38.
Manford, M., Andermann, F., 1998. Complex visual hallucinations. Clin. Neurobiol. Insights *Brain* 121, 1819–1840.
Marom, S., 2010. Neural timescales or lack thereof. Prog. Neurobiol. 90, 16–28.
Marshall, L., Mathys, C., Ruge, D., De Berker, A.O., Dayan, P., Stephan, K.E., Bestmann, S., 2016. Pharmacological fingerprints of contextual uncertainty. PLoS Biol. 14, e1002575.
Mason, O.J., Brady, F., 2009. The psychotomimetic effects of short-term sensory deprivation. J. Nerv. Ment. Dis. 197, 783–785.
Mather, M., Sutherland, M.R., 2011. Arousal-biased competition in perception and memory. Perspect. Psychol. Sci. 6, 114–133.
McCormick, D.A., McGinley, M.J., Salkoff, D.B., 2015. Brain state dependent activity in the cortex and thalamus. Curr. Opin. Neurobiol. 31, 133–140.
Medjkane, F., Notredame, C.E., Sharkey, L., D'hondt, F., Vaiva, G., Jardri, R., 2020. Association between childhood trauma and multimodal early-onset hallucinations. Br. J. Psychiatry 216, 156–158.
Menon, G.J., 2005. Complex visual hallucinations in the visually impaired: a structured history-taking approach. Arch. Ophthalmol. 123, 349–355.
Montagnese, M., Leptourgos, P., Fernyhough, C., Waters, F., Larøi, F., Jardri, R., Urwyler, P., 2021. A review of multimodal hallucinations: categorization, assessment, theoretical perspectives, and clinical recommendations. Schizophr. Bull. 47, 237–248.
Moran, R.J., Campo, P., Symmonds, M., Stephan, K.E., Dolan, R.J., Friston, K.J., 2013. Free energy, precision and learning: the role of cholinergic neuromodulation. J. Neurosci. 33, 8227–8236.
Mosimann, U.P., Rowan, E.N., Partington, C.E., Collerton, D., Littlewood, E., O'Brien, J. T., McKeith, I.G., 2006. Characteristics of visual hallucinations in Parkinson disease dementia and dementia with Lewy bodies. Am. J. Geriatr. Psychiatry 14, 153–160.
Muller, A.J., Shine, J.M., Halliday, G.M., Lewis, S.J., 2014. Visual hallucinations in Parkinson's disease: theoretical models. Mov. Disord. 29, 1591–1598.
Nara, S., Fujii, H., Tsukada, H., Tsuda, I., 2022. Visual hallucinations in dementia with Lewy bodies originate from necrosis of characteristic neurons and connections in three-module perception model. Sci. Rep. 14172.
Nara, S.A., Fujii, H., Tsukada, H., Tsuda, I., 2019. A three-modules scenario in an interpretation of visual hallucination in dementia with Lewy bodies and preliminary results of computer experiments. 2019 Int. Jt. Conf. Neural Netw. IEEE 1–8.
Nour, M.M., Carhart-Harris, R.L., 2017. Psychedelics and the science of self-experience. Br. J. Psychiatry 210, 177–179.
O'Brien, J., Taylor, J.P., Ballard, C., Barker, R.A., Bradley, C., Burns, A., Weil, R.S., 2020. Visual hallucinations in neurological and ophthalmological disease: pathophysiology and management. J. Neurol., Neurosurg. Psychiatry 91, 512–519.
O'Brien, J.T., Firbank, M.J., Mosimann, U.P., Burn, D.J., McKeith, I.G., 2005. Change in perfusion, hallucinations and fluctuations in consciousness in dementia with Lewy bodies. Psychiatry Res.: Neuroimaging 139, 79–88.
Onofrj, M., Espay, A.J., Bonanni, L., Delli Pizzi, S., Sensi, S.L., 2019. Hallucinations, somatic-functional disorders of PD-DLB as expressions of thalamic dysfunction. Mov. Disord. 34, 1100–1111.
Parr, T., Friston, K.J., 2019. Attention or salience? Curr. Opin. Psychol. 29, 1–5.
Parr, T., Rees, G., Friston, K.J., 2018. Computational neuropsychology and Bayesian inference. Front. Hum. Neurosci. 61.
Perdikis, D., Huys, R., Jirsa, V.K., 2011. Time scale hierarchies in the functional organization of complex behaviors. PLoS Comput. Biol. 7, e1002198.
de Pierrefeu, A., Fovet, T., Hadj-Selem, F., Löfstedt, T., Ciuciu, P., Lefebvre, S., Duchesnay, E., 2018. Prediction of activation patterns preceding hallucinations in patients with schizophrenia using machine learning with structured sparsity. Hum. Brain Mapp. 39, 1777–1788.
Prinzmetal, W., McCool, C., Park, S., 2005. Attention: reaction time and accuracy reveal different mechanisms. J. Exp. Psychol.: Gen. 134, 73–92.
Renouf, S., ffytche, D., Pinto, R., Murray, J., Lawrence, V., 2018. Visual hallucinations in dementia and Parkinson's disease: a qualitative exploration of patient and caregiver experiences. Int. J. Geriatr. Psychiatry 33, 1327–1334.
Ross, J., Morrone, M.C., Goldberg, M.E., Burr, D.C., 2001. Changes in visual perception at the time of saccades. Trends Neurosci. 24, 113–121.
Russo, M., Carrarini, C., Dono, F., Rispoli, M.G., Di Pietro, M., Di Stefano, V., Onofrj, M., 2019. The pharmacology of visual hallucinations in synucleinopathies. Front. Pharmacol. 10, 1379.
Santhouse, A.M., Howard, R.J., ffytche, D.H., 2000. Visual hallucinatory syndromes and the anatomy of the visual brain. Brain 123, 2055–2064.
Schultz, W., 2016. Dopamine reward prediction-error signalling: a two-component response. Nat. Rev. Neurosci. 17, 183–195.
Schultz, W., Dayan, P., Montague, P.R., 1997. A neural substrate of prediction and reward. Science 275, 1593–1599.
Scott, I.U., Schein, O.D., Feuer, W.J., Folstein, M.F., 2001. Visual hallucinations in patients with retinal disease. Am. J. Ophthalmol. 13, 590–598.
Series, P., Reichert, D., Storkey, A.J., 2010. Hallucinations in Charles Bonnet syndrome induced by homeostasis: a deep Boltzmann machine model. Adv. Neural Inf. Process. Syst. 23, 2020–2028.
Shine, J.M., 2021. The thalamus integrates the macrosystems of the brain to facilitate complex, adaptive brain network dynamics. Prog. Neurobiol. 199, 101951.
Shine, J.M., Halliday, G.M., Naismith, S.L., Lewis, S.J., 2011. Visual misperceptions and hallucinations in Parkinson's disease: dysfunction of attentional control networks? Mov. Disord. 26, 2154–2159.
Shine, J.M., O'Callaghan, C., Halliday, G.M., Lewis, S.J., 2014. Tricks of the mind: visual hallucinations as disorders of attention. Prog. Neurobiol. 116, 58–65.
Singh, G., Sharan, P., Kulhara, P., 2003. Role of coping strategies and attitudes in mediating distress due to hallucinations in schizophrenia. Psychiatry Clin. Neurosci. 57, 517–522.
Spratling, M.W., 2017. A review of predictive coding algorithms. Brain Cogn. 112, 92–97.
Squire, L.R., Zola, S.M., 1997. Amnesia, memory and brain systems. Philos. Trans. R. Soc. Lond. Ser. B: Biol. Sci. 352, 1663–1673.
Stahl, S.M., 2018. Beyond the dopamine hypothesis of schizophrenia to three neural networks of psychosis: dopamine, serotonin, and glutamate. CNS Spectr. 23, 187–191.
Stebbins, G.T., Goetz, C.G., Carrillo, M.C., Bangen, K.J., Turner, D.A., Glover, G.H., Gabrieli, J.D.E., 2004. Altered cortical visual processing in PD with hallucinations: an fMRI study. Neurology 63, 1409–1416.
Summerfield, C., Egner, T., 2009. Expectation (and attention) in visual cognition. Trends Cogn. Sci. 13, 403–409.
Todo, M., 2020. Towards the interpretation of complex visual hallucinations in terms of self-reorganization of neural networks. Neurosci. Res. 156, 147–158.
Toh, W.L., McCarthy-Jones, S., Copolov, D., Rossell, S.L., 2019. Have we overlooked the significance of multimodal hallucinations in schizophrenia? Psychiatry Res. 279, 358–360.
Tsuda, I., Yamaguti, Y., Watanabe, H., 2016. Self-organization with constraints—a mathematical model for functional differentiation. Entropy 18, 74.
Tsukada, H., Fujii, H., Aihara, K., Tsuda, I., 2015. Computational model of visual hallucination in dementia with Lewy bodies. Neural Netw. 62, 73–82.
Urwyler, P., Nef, T., Killen, A., Collerton, D., Thomas, A., Burn, D., Mosimann, U.P., 2014. Visual complaints and visual hallucinations in Parkinson's disease. Park. Relat. Disord. 20, 318–322.
Van Ommen, M.M., Van Beilen, M., Cornelissen, F.W., Smid, H.G.O.M., Knegtering, H., Aleman, A., GROUP Investigators, 2016. The prevalence of visual hallucinations in non-affective psychosis, and the role of perception and attention. Psychol. Med. 46, 1735–1747.
Vasishth, S., Nicenboim, B., Engelmann, F., Burchert, F., 2019. Computational models of retrieval processes in sentence processing. Trends Cogn. Sci. 23, 968–982.
Vellante, M., Larøi, F., Cella, M., Raballo, A., Petretto, D.R., Preti, A., 2012. Hallucination-like experiences in the nonclinical population. J. Nerv. Ment. Dis. 200, 310–315.
Warburton, D.M., Wesnes, K., Edwards, J., Larrad, D., 1985. Scopolamine and the sensory conditioning of hallucinations. Neuropsychobiology 14, 198–202.
Watabe-Uchida, M., Eshel, N., Uchida, N., 2017. Neural circuitry of reward prediction error. Annu. Rev. Neurosci. 40, 373.
Waters, F., Collerton, D., Ffytche, D.H., Jardri, R., Pins, D., Dudley, R., Larøi, F., 2014. Visual hallucinations in the psychosis spectrum and comparative information from neurodegenerative disorders and eye disease. Schizophr. Bull. 40 (Suppl_4), S233–S245.
Webster, R., Holroyd, S., 2000. Prevalence of psychotic symptoms in delirium. Psychosomatics 41, 519–522.
Zarkali, A., Adams, R.A., Psarras, S., Leyland, L.A., Rees, G., Weil, R.S., 2019. Increased weighting on prior knowledge in Lewy body-associated visual hallucinations. Brain Commun. 1 (1) fcz007.
Zarkali, A., Luppi, A.I., Stamatakis, E.A., Reeves, S., McColgan, P., Leyland, L.-A., Lees, A. J., Weil, R.S., 2021. Chang. Dyn. Transit. Integr. Segreg. S. Under Vis. hallucinations Parkinson's Dis. https://doi.org/10.1101/2021.06.21.449237.
Zmigrod, L., Garrison, J.R., Carr, J., Simons, J.S., 2016. The neural mechanisms of hallucinations: a quantitative meta-analysis of neuroimaging studies. Neurosci. Biobehav. Rev. 69, 113–123.
Zoldan, J., Friedberg, G., Livneh, M., Melamed, E., 1995. Psychosis in advanced Parkinson's disease: treatment with ondansetron, a 5–HT3 receptor antagonist. Neurology 45, 1305–1308.

Glossary

Attention: Processes that prioritize those (sensory) data sources relevant to task demands over others in order to bias perception

Spatial attention: Processes that direct visual perception towards relevant parts of the visual field

Precision (confidence): The confidence assigned to probability distributions in internal generative models. Highly confident or precise distributions have greater influence on perception, e.g., data assumed to be generated in a precise way cause more belief-updating than those assumed to be generated more stochastically

Cortical excitability: Strength of neuronal response to a given stimulus

Deafferentation: Loss of neural input to sensory system

Expectancy: The chance of particular perceptions or data being present in a specific visual environment

Formed or complex hallucination: A perception of a meaningful image of something which is not there in the visual environment, often a person or animal

Fortification hallucination: Jagged lines in part of the visual field, often associated with migraine
Illusion: An incorrect perception of an object which is in the visual environment
Likelihood probability: Chance of sensory data given some hypothesis or cause of those data.
Multimodal hallucination: A hallucination in two or more sensory modalities at the same time, for example a face which speaks
Object Attention: Processes that prioritize data relevant to aspects of a perceived object
Pareidolia: The propensity to see meaningful images in meaningless shapes. For example, faces in clouds.
Passage hallucination: A brief hallucination of something passing in the peripheral visual field
Posterior probability: Belief about a hypothesis or causes of sensory data after having observed those data. What is believed to have been "seen"
Prediction error: Mismatch between expected and actual sensory data
Predictive processing (generative perception): Framework for visual perception in which what is "seen" is an internal model which is validated against sensory data
Presence hallucination: A feeling of the presence of someone or something, in the absence of seeing it.
Prior probability (sometimes termed an empirical prior or prior belief): Belief about some hypothesis or cause of sensory data before making any observations of those data.
Simple or unformed hallucination: A perception of meaningless dots or shapes which are not there in the visual environment
Synaptic gain: Process of modulating transmission between neurons
Unimodal hallucination: A hallucination in a single sensory modality, for example a purely visual hallucination of a face
Veridical perception: A perception that matches what is there in the visual environment
Voluntary image: A conscious imaging of a perception in the mind's eye