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How Can We See Things That Are Not There?

Current Insights into Complex Visual Hallucinations

Abstract: *It is not at all uncommon to see people, animals, or objects that other people cannot perceive. Data from studies of pathological hallucinations suggests that distributed functional changes within visual and associated systems increases the risk of visual hallucinations, though how this occurs is not yet clear. Candidate theories developed in the context of neurodegenerative disorders, eye disease, and psychosis each emphasize specific aspects of dysfunction within visual systems. Outwith these, there are suggestions that mnemonic and executive systems may play a key role for some people;*

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particularly in those without organic disease. Shifts within dynamic neural networks may explain why some people are at a raised risk of visual hallucinations, and why specific hallucinatory episodes occur. This hypothesis has highlighted limitations in methods for modelling and measuring dynamic brain function. Developments in functional imaging, novel interventional techniques such as transcranial magnetic stimulation, and new methods for analysing complex imaging data such as multi voxel pattern analysis and graph theory, together with advances in theoretical computational models of hallucinations, raise hopes for a better understanding of the brain changes associated with these experiences.

1. Introduction

1.1. Challenges in the definition of visual hallucinations

Hallucinations are one of a number of fuzzy forms of visual experiences (Brewin *et al.*, 2010; Blom, 2014; Ffytche, 2014) including dreams, simple hallucinations, complex hallucinations, misperceptions, illusions, voluntary images, and veridical perceptions (Figure 1, on p. 225).

Having clear definitions of these phenomena is extremely difficult, entwined as they are with shifting historical and philosophical conceptualizations of normal and abnormal vision (Berrios and Marková, 2014), and given that the dimensions along which distinctions are made vary between different groups of experience (Blom, 2014; Collerton, Mosimann and Perry, 2014). Table 1 loosely indicates how we will be using these terms in this paper.

This fuzziness in definition may be intrinsic to generative models of visual perception (Collerton, Mosimann and Archibald, 2012). These models state that, in all cases, the relationship between objects in the environment and subjective perception is loose (Rees, 2014), which then implies that there can never be precise qualitative boundaries and all contiguous phenomena must shade into each other. Hallucinations, then, are the phenomena which are most distant from veridical perceptions. Thus, despite the imprecise boundaries between these phenomena, the core concept of visual hallucinations — seeing something involuntarily which, by all other measures, is not there — has been recognized as existing for many years; though its interpretation has shifted to reflect the cultural and scientific context of the time (Berrios and Marková, 2014).

Visual experience	Key aspects of experience
Dream	An experience during sleep incorporating visual and other sensory elements
Simple hallucination	Unformed dots, blobs, lines, clouds, and similar
Misperception	Idiosyncratic transitory mistake in perception of an object in the environment
Illusion	Persistent alternative perception of an ambiguous object in the environment
Voluntary images	Internal, self-created and controlled images without a corresponding object in the environment
Veridical perception	Good enough perception of a corresponding object in the environment
Complex hallucination	Persisting perception of an object where there is no corresponding object in the environment

Table 1. Key defining characteristics of visual experiences (we use 'object' to mean any meaningful thing — a person, face, word, animal, or implement).

Notwithstanding these challenges in defining and researching hallucinations as distinct from other visual phenomena, it has been possible to classify them reliably enough in practice to allow systematic study to take place. Thus, Figure 2 (on p. 225) illustrates how focused assessment can reliably count these phenomena.

In this paper, we will focus on complex, or formed, hallucinations; partially because we feel these are most closely related to normal visual function, but also because clinically they are a significant challenge, being associated with a range of disorders in which they lead to distress and disability (Scott *et al.*, 2001; Dudley *et al.*, 2012; Gibson *et al.*, 2013). We will review what is currently known about the character of complex hallucinations, who tends to experience them, why they may occur, models for understanding their genesis, and directions for improving that understanding in the future. In doing so, we will concentrate on the current literature, though there is a vast range of classic accounts which are covered in previous reviews.

1.2. The character of complex visual hallucinations

Our approach has been to attempt to explain the regularities of complex hallucinations with the assumption that most hallucinatory experiences are variations on a theme (e.g. Collerton, Perry and McKeith, 2005). However, even within regularities there can be quite

substantial variation. A comparison of the hallucinations of Lewy body disorders (which are associated with a loss of cortical acetylcholine) with those induced by the anticholinergic atropine indicates a basic similarity in content — in both cases, figures and animals are commonly seen — but the timescales and associated phenomenology are quite different. In atropine toxicity, hallucinations are more frequent, more persistent, and are triggered by eye closure (Fisher, 1991); whereas in Lewy body disorders they occur with open eyes and disappear on eye closure. Thus any model must be able to account for both consistency and variation.

Below are two verbatim reports collected from our patients in the course of our clinical work of the types of hallucinations we are aiming to explore; one from a man with dementia with Lewy bodies, the other from a man with macular degeneration. Both are representative of the generality of these experiences (for reviews, see Barnes and David, 2001; Menon *et al.*, 2003; Gauntlett-Gilbert and Kuipers, 2003).

'[I saw a] Japanese lady who was sat outside. It came clear after a while. She was sitting outside and, to me, looking from my ward window which was quite a distance, she used to come out in the morning from the wards further up from me and sit around the corner. She had this green jacket, and black jacket, and she had white sneakers and her hair was all Japanese style with sleek, black hair.

She used to just sit and look at the wall. I was worried to death about her because she was doing this each day. Sometimes the weather was absolutely terrible; the wind was howling and the rain was up. I was at the point of going to talk to these girls, these Japanese girls, to ask them to take her in since she was in a terrible state; soaking wet.

It never entered my head that it was a compost bag, and that is what it turned out to be.' (A person with dementia with Lewy bodies describing his hallucination of a human figure)

'I looked up from where I was sitting across at the south facing window. And in that window, I saw some movement; activity. I could not make it out from where I was sitting but I thought it may be a wasp and went over to interfere with it if it was.

It turned out to be an insect, but a long one: if you could imagine a dragonfly with wings at both ends. It was a little like that, which persuaded me that it was a phantom, a Bonnet product. Where we got the idea from, I do not know.

Anyway, I went up to the window to get a closer look which is where I saw it had wings at each end, and as I did so, it flew towards me and, without thinking, I clapped on it. When I opened my hands, it fell out of them down on to the ground, not freely; quite slowly. And as it did so, it turned somehow towards me. I suppose it must have been a head but I did not see anything clearly like an individual head; not then.

And when it came to rest eventually on the ground, it looked towards me rather, very sadly really. It made me feel quite guilty.' (A person with macular degeneration describing his hallucination of an insect)

2. The General Characteristics of Visual Hallucinations

2.1. Content

Although the content of visual hallucinations is idiosyncratic as the quotes above illustrate, there are some consistencies in the content and character of hallucinations across different people and disorders (Figure 3).

What is seen tends to fall within a limited number of categories. Thus, people and animals tend to be most often seen across disorders, with objects tending to be rarer (Collerton, Perry and McKeith, 2005). Content is influenced by gender and culture; women tend to see children more often, and men, objects, for example (Urwyler *et al.*, 2015). In rare cases, particularly where there is no visual environment, in blindness for example, panoramic hallucinations of entire scenes can occur (*ibid.*), though even so, these are rare; perhaps because total visual loss is exceptional, even in blindness. When visual context is present, hallucinations tend to be of normal size and morphology. Where that context is lacking, as again in eye disease, distortions of size and shape can occur (Waters *et al.*, 2014), suggesting an intimate interaction between visual inputs and generated images in order to maintain the scalar respectability of the hallucination. Although there may be movement within an hallucination, the hallucination itself tends to be relatively static within the visual environment (Collerton, Perry and McKeith, 2005).

Despite around half of hallucinations being of a recognizable person, animal, or object (Boubert and Barnes, 2015; Urwyler *et al.*, 2015), and there often being a repetitive quality to the experience (the repeated hallucination of the Japanese woman at the same time each day, for instance), these are not the stereotyped re-experiencing of specific episodic memories seen in visual flashbacks (Brewin *et al.*, 2010), suggesting that they arise from generic object representations (proto-objects, Collerton, Perry and McKeith, 2005), rather than particular autobiographical memories. People who have hallucinations which are recognized as being of a known person, animal, or object

have a distinctly different cognitive profile with less inhibitory impairment compared to people who see unrecognized images; implying that different disturbances across processing networks lead to different content (Boubert and Barnes, 2015).

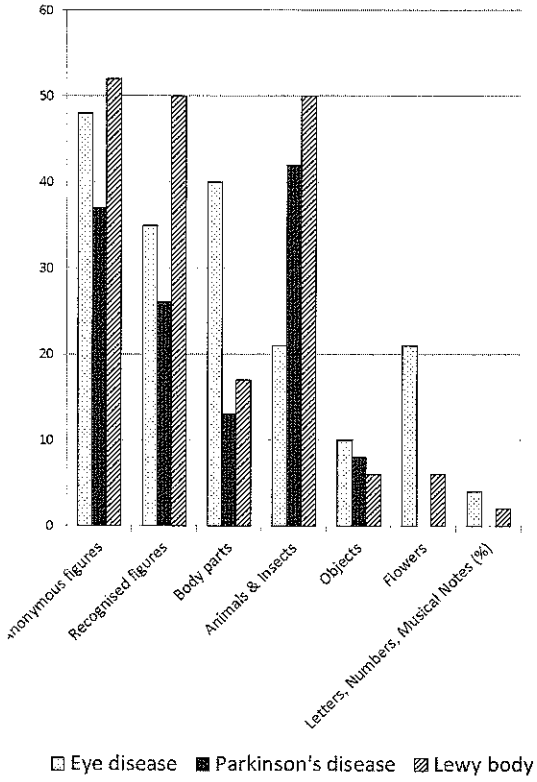

Figure 3. Frequency of the content of visual hallucinations in eye disease, Parkinson's disease, and Lewy body dementia (Parkinson's disease dementia and dementia with Lewy bodies) (Urwyler *et al.*, 2015).

These experiences are perceptually compelling. For most people, like the man with dementia with Lewy bodies, they appear real, and elicit understandable emotional and behavioural responses (Waters *et al.*, 2014). Even when the person experiencing them knows, at least partially, that they are not real, as does the man with eye disease described above, it is hard not to respond.

2.2. Dynamic aspects of visual hallucinations

Hallucinatory episodes have characteristic dynamics. They are rare, occurring only a few times a day at most (see Collerton, Perry and McKeith, 2005, for review). Thus, most perception is not hallucinatory. When hallucinations are continuous, they tend to be associated with grossly disturbed consciousness, as in delirium, drug intoxication, or the exceptionally rare peduncular hallucinosis (Müri, 2014), or very occasionally in eye disease. When they do occur, the timescale is of the order of minutes rather than seconds or hours. This is variable between people but tends to be consistent within each person.

Hallucinations are also discrete, appropriate to context but generally discontinuous with what goes before and after, with relatively abrupt transitions from *not there* to *there* and from *there* to *not there.* They are unstable with regards to changes in visual inputs, e.g. turning lights on or off, looking away, or focusing on one specific aspect of the hallucination. In contrast, they are not unstable with regards to changes in broad top-down information flow. Thus prior emotional state or expectancies do not appear to play a major role in most hallucinations. The exception may be post-bereavement hallucinations, which may reflect lags in expectancies adjusting to the loss of a familiar person (Grimby, 1993; Castelnovo *et al.*, 2015).

If hallucinations do recur, as with the Japanese woman who was seen each morning, they tend to be at the same place and time, though the same time and place does not guarantee recurrence.

2.3. Interactions with the environment

As the quotes earlier illustrate, the existing environment continues to be perceived during an hallucination, at least partially. The hallucination is also highly integrated into shifts in the visual environment. So we can see how the hallucination of the Japanese woman becomes wet when it is raining, and the insect falls as if injured after it is clapped. If an object is present, then it is not hallucinated. For example, if a person is in a room, that same person is not also hallucinated. Thus, hallucinations do not happen alongside reality, though they are incorporated into it. An hallucinatory person may sit on a real chair, but an hallucinatory chair is not seen alongside the real one (perceptual perseveration of an image is different; Santhouse, Howard and Ffytche, 2000). This feature of hallucinations can be demonstrated experimentally in the pareidolia task (see Figure 4), in which hallucinations occur only in the non-meaningful part of scenes. Additionally,

Caputo's illusion (in which looking at your face in a darkened room generates hallucinatory images, 2010) is lessened in people prone to hallucinations (Errington *et al.*, 2015).

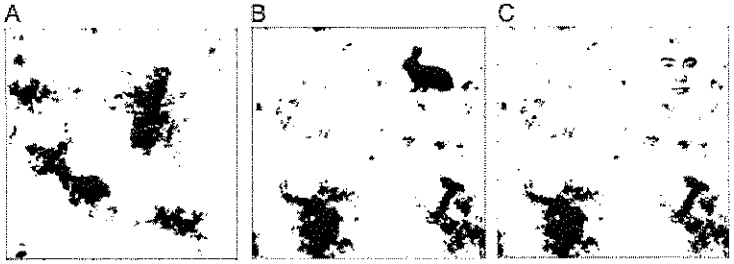

Figure 4. Examples of pareidolia stimuli from Yokoi *et al.* (2014). True perceptions of a rabbit and a face are present in B and C. Pareidolia, perceptions which are not present, are common in people prone to hallucinations, but occur in the blank, meaningless parts of the pattern. They are not incorporated into the real stimuli.

We can therefore see that hallucinations have a characteristic quality in relation to the environment that any successful model will need to account for (Collerton, Perry and McKeith, 2005).

3. Who Tends to Hallucinate?

Moving from what is seen, we also need to account for who tends to hallucinate.

Hallucinatory experiences are not, in themselves, pathological (D'Agostino, Castelnovo and Scarone, 2014). Just about everyone has an hallucination at some time in her or his life, but they are far, far more commonly seen in certain illnesses; specifically eye disease, delirium, psychosis, and Lewy body dementia. If we look across 'normality' and 'disorder' however, it is clear that there is an inverse relationship between frequency of hallucinations and how many people experience them: only a few people have many hallucinations.

Figure 5 illustrates the variations in rates of people with visual hallucinations by disorder. Again, we can see that there is a continuous increase in rates with no evidence of discontinuity.

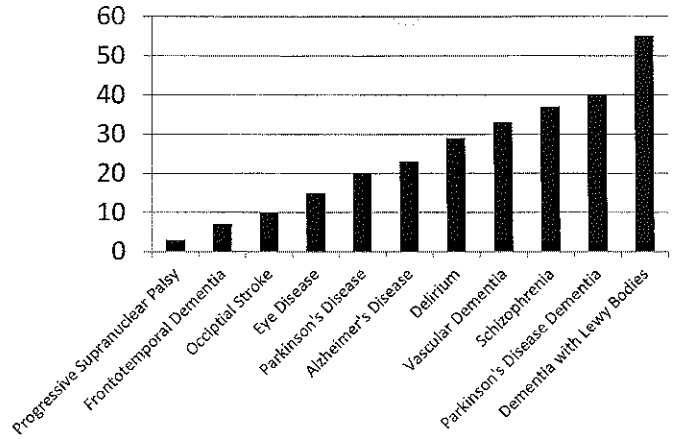

Figure 5. Variations in the prevalance of recurrent complex hallucinations by diagnosis. Data taken from Collerton, Perry and McKeith (2005).

3.1. Associated factors

Looking within and across these disorders, a number of general factors are associated with the risk of visual hallucinations (Collerton, Perry and McKeith, 2005), including sleep disorders — particularly narcolepsy and REM sleep behaviour disorder — poor vision, an impoverished visual environment, cognitive impairment — particularly in visual attention and perception — under- and over-arousal, and a range of drugs — particularly cholinergic antagonists, dopaminergic agonists, and polypharmacy. An exceptionally wide range of medications, both prescribed and illicit, are associated with an increased risk of hallucinations (see Perry, Ashton and Young, 2002, for extensive reviews). However, rationalizing prescribed drugs which have a propensity to cause VH remains the mainstay of treatment (Collerton and Taylor, 2013) and there are no established specific treatments which reduce visual hallucinations *per se*.

3.2. Associations with other visual symptoms

Visual hallucinations are more common in disorders with high levels of other visual abnormalities — see Figure 6 — and are associated with greater impairment in visual function in its widest sense across those disorders. Visual perceptual problems such as agnosia and misperception are common in these disorders and there is a relationship,

at least within eye disease, delirium, and Lewy body disorders, between the severity of visual impairment and the risk of visual hallucinations. However, the risk of visual hallucinations within an individual person tends not to be reliably associated with other visual symptoms at least within Lewy body diseases (Urwyler *et al.*, 2014), and even in those people who do experience hallucinations, the majority of visual errors tend to be errors of omission.

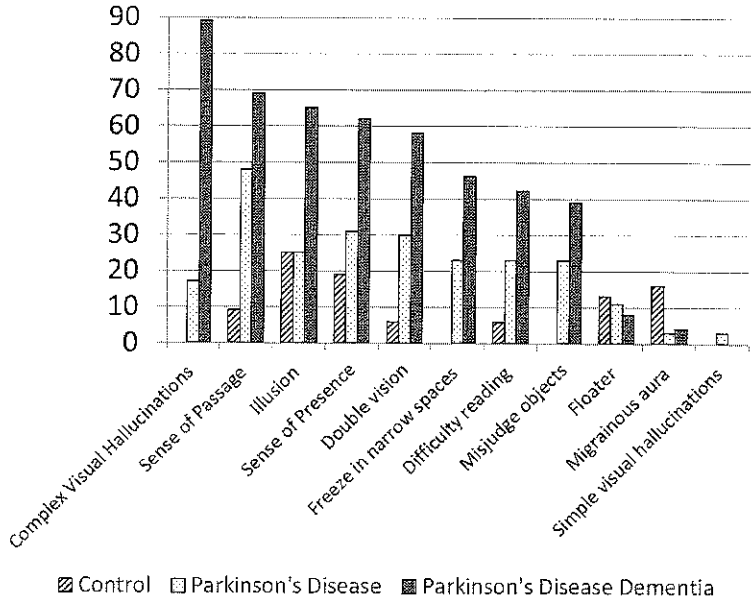

Figure 6. Rates of complex hallucinations and other visual symptoms within Lewy body disorders (Urwyler *et al.*, 2014).

Thus, as Ffytche and Howard (1999) have suggested, hallucinations and other 'positive' visual symptoms depend upon different processes to those underlying 'negative' symptoms such as agnosiae.

To summarize, then, complex visual hallucinations have a distinct form and character, a consistent dynamic quality, and vary systematically and continuously in frequency across disorders and risk factors. We will now explore how we may account for these regularities.

4. How Can Hallucinations Happen?

4.1. The character of veridical visual perception

In contrast to classical, bottom-up, passive models of visual (and other sensory) perception, veridical perception is now understood to be an exceptionally dynamic process which balances multiple influences; both top-down and bottom-up (see, for example, Yuille and Kersten, 2006). What is 'seen' as out there in the visual environment is a good enough, probabilistic, internal model of that world — sparse and functional, resilient and iterative, predictive, dynamic, and able to maintain stability and coherence but also allow rapid change (see, for example, Summerfield and Egner, 2009). We see what we predict is there, not what was there, since by the time visual information could reach the brain and be processed, the world will have changed. The subjective experience of a detailed, coherent, entire visual world is constructed from a relatively small amount of information. Estimating the information and processing capacity of the human brain is difficult, but estimates suggest that there is a million-fold decrease in the information flow capacity of the visual system from retina to early visual cortex such that only around 10,000 bits per second reach layer IV of V1 (Raichle, 2010), with information flow through the secondary pulvinar pathway being more sparse still. Contrast this with the estimated processing power of the human brain, which is perhaps five hundred billion times greater (Markram, 2006), and the preponderance of internal processing over external information is clear.

In order to maintain the model's dynamic coherence and a close relationship between what is seen and what is really out there, it shifts continually to minimize the discrepancy between actual and predicted visual input (Friston, 2005). Thus, even though there is no absolute guarantee that the model truly reflects what is out there, its self-correction is such that in everyday cases the functionality of the model is such that few things that have to be seen are missed, and even fewer things that are not there are seen. Consider that if most people will have one or two hallucinatory experiences in their lifetime, what small a proportion that is of all their perceptions.

Create the right conditions, however, as in change and inattentional blindess experiments, and we can all not see things that are there (see, for example, Simons and Ambinder, 2005). Conversely, in the correct circumstances, everyone has the capacity to 'see things that are not there' (Rees, 2014). For example, the classical illusions in Figure 7

which show the 'hallucinatory' shapes of a triangle and a circle illustrate how even early visual processing is adapted to perceive substance when information is partial (Marr, 1982). Perception, even in its most basic components, is never based on visual input alone, but also on stored information drawn from past experiences. Thus disparity information alone cannot determine depth perception (Marr and Poggio, 1979).

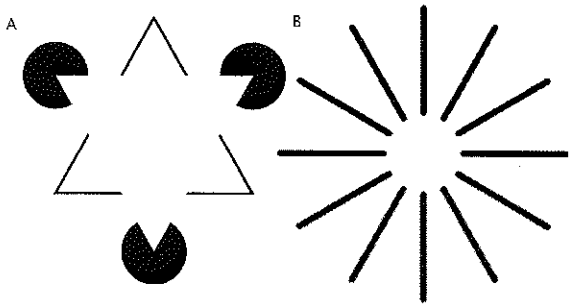

Figure 7. Illusory triangle and circle from Rees (2014).

4.2. Why do we not hallucinate more frequently?

If veridical perception is generative and has an innate capacity to generate hallucinations, the question becomes why we do not hallucinate more frequently? As noted above, even in people who are prone to visual hallucinations, the majority of their perception is veridical.

There are three factors to consider. Firstly, visual input and prior learning constrain hallucinations. As noted earlier, attending to something or someone who is really there takes precedence over seeing the same thing which is not there. Hence, even in people prone to hallucinations, if they are usually attending to something which is there, they lack the attentional capacity to see something that is not there. Consistent with this, poor visual function or a lack of things to see in the environment both increase the chances of hallucinations (Collerton, Perry and McKeith, 2005), though interestingly the complete absence of visual information, through eye closure for example, reduces the probability of hallucinations. Secondly, the visual system appears to be very good at balancing the need not to miss things that are there and not to see things that are not there (Bruce, Green and Georgeson, 2003). In everyday life, it is astoundingly accurate on the basis of, as noted above, relatively little information. You need massive destabilization of visual function through drugs, brain injury, or eye

damage, or prolonged sensory deprivation before visual function degrades to the point of hallucinations. Finally, if, as computational models of the brain suggest, processing is constrained by the need to minimize energy expenditure while maximizing information processing, then if hallucinations are energy costly, there will be an inbuilt bias against them (Friston, 2005). Understanding the mechanisms underlying visual resilience may lead to a greater understanding of when these may break down.

5. Understanding Why People Hallucinate

Looking historically, it is striking that, as evidence has accumulated, so has the framework used to account for hallucinations expanded.

The earliest models (Manford and Andermann, 1998) considered single factor explanations for the propensity towards visual hallucinations, for example cortical irritation, cortical release and hyperactivity, unbalanced top-down activation, or dream intrusion. However, these all struggled to account for regularities in hallucinations across disorders and risk factors. These were then superseded around a decade ago by two more general classes of models; misattribution, and distributed visual impairment.

5.1. Misattribution models

These are developed within research groups looking at younger people with hallucinations associated with psychosis, and draw upon influential models of auditory verbal hallucinations which see these as internal speech which is misattributed to an external source. Thus, it is suggested that visual hallucinations are the erroneous attribution of internally-generated images to external sources (Barnes *et al.*, 2003; Brébion *et al.*, 2008; Barnes, 2014). Two factors may contribute to this error. Mental images may have more perceptual characteristics than are expected by the cognitive system; either because the images are more perception-like than is usual — more vivid, or detailed, or 'life-like' (for example, the lack of a corollary discharge, an internal 'tag' that indicates that perceptions are internally generated; Feinberg, 2011) — or, conversely, that veridical perceptions are more like internally generated images — lacking the compelling nature of most veridical perception. In addition, it may be that, regardless of the differences in the quality of images and perception for these people, they also have a general difficulty in correctly assigning experiences to internal or external sources.

5.2. Distributed impairment models

Developing in parallel to the misattribution model, but within research groups investigating the hallucinations seen in later life, particularly those associated with Lewy body disorders (Collerton, Mosimann and Archibald, 2012), these suggest that combined dysfunctions across at least two different aspects of the distributed visual system increase the frequency of visual hallucinations as summarized in Table 2.

	Perception	**Attention**
Perception and Attention Deficit (Collerton, Perry and McKeith, 2005)	In combination with poor visual perception, The hallucinatory element of a scene is not disconfirmed by discrepant visual input.	continuous activity is under-constrained by an impairment in attentional focus.
Integrative Model (Diederich *et al.*, 2005; 2009; 2014)	Poor visual input and processing in combination with, Visual input cannot be interpreted leading to deblocking of visual memory contents (release phenomena) as visual hallucinations.	defective central visual monitoring produces partial visual deprivation.
Attentional Model (Shine *et al.*, 2011; 2014)	In the presence of ambiguous percepts,	there is an inability to recruit the visually-mediated dorsal attentional network leading to an over-reliance on networks such as the default mode network to interpret the stimuli.

Table 2. Distributed impairment models of complex hallucinations.

5.3. Review of the evidence for multifactor models

As evidence as accrued as to the breadth of changes which are associated with visual hallucinations, so the level of explanation has shifted from single psychological factor and brain area, to psychological function and brain pathway, to psychological capability and brain network.

Our metaphors of brain function have, however, always reflected the dominant technology of the time: from water power and the sewage system, to the telegraph, the computer, and now the internet (Dooremalen and Borsboom, 2010). There is, therefore, a danger that the current network focus of distributed dysfunction is more a reflection of our current metaphor than any reality of underlying brain function. However, there is reason to think that there is more going on than just a fashionable analogy (Sporns, 2013). Functional imaging (Meppelink, 2014), pathology and drug effects (Lewis *et al.*, 2014), and cognitive assessment (Barnes, 2014) all suggest that no single part of the visual system is consistently associated with the risk of visual hallucinations, but that the more the system as a whole is stressed, either by a major single factor or a combination of lesser factors acting either acutely or over a longer timescale, then the greater the risk that hallucinations occur (Carter and Ffytche, 2015).

To illustrate with dementia with Lewy bodies (the disorder with the highest rate of visual hallucinations), dysfunctions are distributed widely across visual and associated processing brain (Gratwicke, Jahanshahi and Foltynie, 2015). Thus, there are multiple pathological processes (Ferrer *et al.*, 2012) which lead to a loss of synaptic plasticity and functional connectivity across networks (Galvin *et al.*, 2011; Kenny *et al.*, 2012); along with a loss of cholinergic, dopaminergic, and other neurotransmitter innervation of the cortex and basal ganglia and a compensatory up-regulation of receptors (Burn and McKeith, 2003).

Comparisons with other disorders with distributed dysfunction but without high rates of visual hallucinations, Alzheimer's disease for example, indicates that the nature of changes across brain networks is likely to be significant. In both disorders there are diffuse, sparse, but also distinct changes across brain areas. Thus, in Lewy body disorders, structural imaging shows atrophy concentrated in the visual areas of the occipital and parietal lobes, while in Alzheimer's disease, temporal atrophy is greater. These structural differences are reflected in function. For example, a comparison of fMRI resting state networks

in Alzheimer's disease and dementia with Lewy bodies demonstrates that both show changes in the balance between short and long range connectivity but that these differ (Peraza, Taylor and Kaiser, 2015). In Alzheimer's disease there is a decrease in small worldedness due to a decrease in short range connections (which is concentrated within the temporal cortex), while in dementia with Lewy bodies there is higher small worldedness due to a decrease in middle and longer range connections across the cortex (especially posterior frontal and parietal).

Widening the focus to look at functional brain changes associated with visual hallucinations across disorders suggests that no single aspect of visual processing is critical — or that all are. Figure 8 (on p. 226) indicates that there is a wide range of brain areas that have been associated with complex visual hallucinations in schizophrenia, stroke and eye disease, Lewy body disorders, and epilepsy. Despite the hallucinatory phenomena being similar, there is no grouping within one particular area; rather changes are found throughout the distributed visual system.

To summarize, then, the current evidence suggests that visual hallucinations are not associated with changes in one or even a small number of specific brain areas, but instead reflect modulations of the functioning of distributed brain networks.

6. The Role of Resilience

Previous models have highlighted the role of dysfunction in the creation of visual hallucinations. Implicit within them is the idea that hallucinations simply emerge when the constraints upon perception are released by a failure elsewhere in the visual system (Burke, 2002). Like a balloon compressed in a hand; move one finger and out pops a section. Thus, hallucinations are seen as essentially passive phenomena, something that happens when something else does not.

In their conceptualization of 'positive' visual symptoms, Ffytche and Howard (1999), however, highlighted the possibility that there is some other process at work to actively create these experiences. While impairment is undoubtedly a factor, there is evidence that it alone cannot account for hallucinations; something else may well be equally important — active, adaptive compensation, or resilience.

Firstly, there is the lack of association between hallucinations and other visual symptoms outlined earlier which suggests that there is an additional factor at work in hallucinations. Secondly, while intact

perceptual and attentional function makes hallucinations very improbable, impaired function does not rule in hallucinations (Figure 9), again supporting the possibility that some other factor is at work. Thirdly, we know that the brain attempts to compensate for injury (Duffau, 2006), such that effects on function are always the result of both injury and compensation. There are now several reports of hyperconnectivity between brain areas associated with hallucinations (Ford *et al.*, 2014; O'Callaghan, Muller and Shine, 2014; Rolland *et al.*, 2014). Finally, visual perception is continually, dynamically, balancing the need to maximize true positives while minimizing false negatives in the context of very partial and rapidly changing information. The mechanisms by which it can maintain perception when information is limited (as it always is) may be key to understanding how hallucinations are generated.

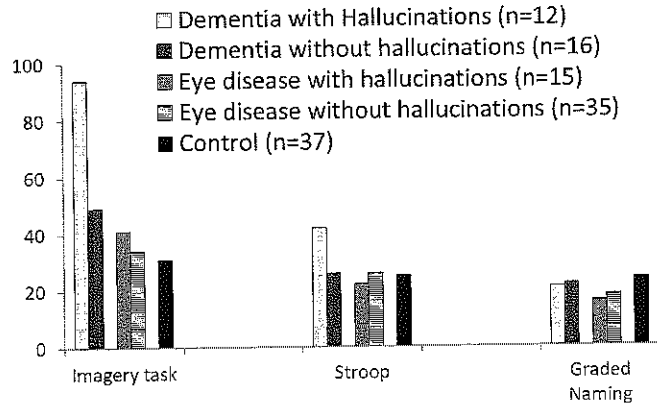

Figure 9. Relationship of hallucinations to impaired performance on perceptual (Imagery and Naming) and attentional (Stroop) tasks. In dementia, combinations of scores on these tasks will accurately predict 80% of individuals without hallucinations and 60% of individuals with, while in eye disease the combination accurately predicts 93% of those without and 18% of those with hallucinations (Makin *et al.*, 2013).

Dolgov and McBeith originally suggested active compensation as a potential factor in 2005. Is there direct evidence, however, that compensation may be relevant? The recent development of the pareidolia paradigm (Uchiyama *et al.*, 2012; 2015) as an analogue of hallucinations has allowed this possibility to be directly tested. In this task, people prone to hallucinations see images that are not there in complex pictures or visual noise. Yokoi *et al.* (2014) have recently shown

that impaired visual perception in dementia with Lewy bodies is accompanied by a compensatory shift in criteria in order to maintain perception of things which are there. Thus, perception dynamically shifts to remain functional in disease states.

We have recently extended this to show that impaired perception in Parkinson's disease with visual hallucinations is accompanied by a compensatory shift in criteria for identifying correct images such that errors in which images are missed are minimized, but at the price of making more errors in which images are seen which are not present (Figure 10). Additionally, if perception is stimulated using visual priming, the criterion shift is reduced, suggesting that impairment and compensation are intimately connected in the mechanisms underlying a propensity to visual hallucinations, and that environmental factors (in this case, visual priming) modulate this relationship. This provides a potential mechanism which can account for the relationship of visual hallucinations with the visual environment in which they are experienced.

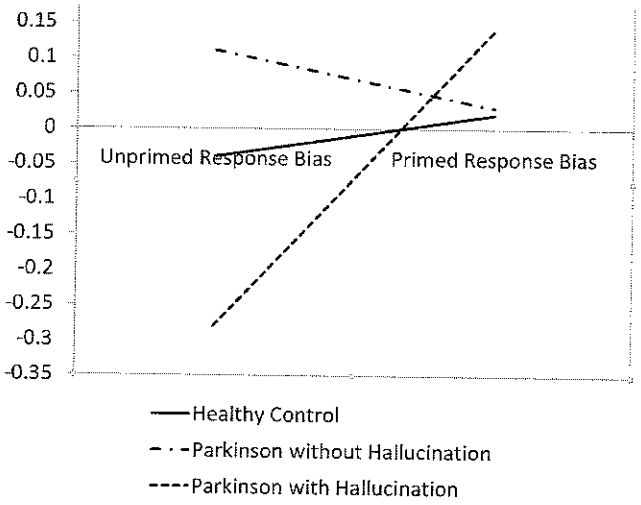

Figure 10. Effects of visual priming on criterion shifts in the pareidolia task in participants with Parkinson's disease with and without visual hallucinations (Bowman *et al.*, data in preparation). When unprimed, the non-hallucinating patients adopted a conservative response bias, avoiding hallucinations at the price of sometimes missing true stimuli. The hallucinating patients adopted a liberal bias, allowing hallucinations to reduce the chance of missing true stimuli. The presentation of a prime appeared to reduce these differences; moving the groups closer towards a neutral response bias.

In addition, the (over)-compensation of compromised networks may provide a bridge between the misattribution models of hallucinations in psychosis and distributed impairment models of hallucinations in Lewy body disease. One key feature of misattribution models is that the internal imagery of hallucinators is particularly vivid and compelling. An hypothesis to explore is that this vividness is a consequence of compensation for impaired visual function in psychosis. In contrast to Lewy body disease, visual and attentional impairments are not associated with visual hallucinations in psychosis (Aleman *et al.*, 2003). And yet, psychosis is partially characterized as a developmental disorder in which cognitive impairments are present (Caspi *et al.*, 2003; Heaton *et al.*, 2001). It may be that it is those people who, in effect, over-compensate for visual impairments who are most prone to vivid imagery and hence visual hallucinations. Teufel *et al.* (2015) have shown that hallucination proneness in psychosis may be associated with a better ability to identify degraded images of people, perhaps because of a greater reliance on prior information. Shine *et al.* (2015) have recently provided bridging evidence that, in Parkinson's disease, internal imagery in people prone to hallucinations may be particularly compelling.

7. Implications of Moving Towards a Compensated Compromised Network Model of Visual Hallucinations

Although the compensated, compromised network is an attractive concept for understanding the genesis of visual hallucinations, it raises significant challenges. In gaining explanatory power through adding in multiple factors which can interact to have contrasting effects, we may have lost testability. What to measure, how to measure it, and how to analyse the interactions are all unresolved questions (Collerton, Mosimann and Perry, 2014). Though we are now able to gather whole-brain data sets tracking visual hallucinations from fMRI, EEG, and MEG (Murphy, Graziadio and Taylor, 2014), our ability to analyse these data and relate them to models of brain function is in its infancy. Perturbing brain networks using non-invasive stimulation techniques such as transcranial magnetic stimulation (TMS) may offer experimental opportunities to further understand visual dynamics. For example, TMS applied to the visual system has suggested functional alterations in visual cortical excitability, perhaps as a result of a loss of top-down inhibition (Taylor, Firbank and O'Brien, 2015), which may contribute to the propensity of patients with dementia with Lewy

bodies to hallucinate more frequently and severely (Taylor *et al.*, 2011). Combining investigative approaches (e.g. combining TMS with EEG, pharmacological-fMRI, pharmacological-EEG, etc.) may provide further perspectives; for example electro-neural signatures in EEG for psilobycin induced (via 5HT 2A receptor activation) hallucinatory states in healthy individuals have been observed which may help our understanding of pathological hallucination-prone groups (Kometer *et al.*, 2013).

Recognition that the brain as a whole, and the visual system as one aspect of it, is a complex dynamic system, together with the hypothesis that visual hallucinations reflect the end result of dynamic compensation of impairments, puts a high emphasis on understanding and modelling complex dynamic systems in order to give a better insight into the phenomenology of visual hallucinations. As an additional benefit of this approach, aspects of the functioning of complex systems are relevant to understanding the temporal characteristics of hallucinatory episodes.

The idea that a compromised but compensated network is responsible for hallucinations is not new (Peláez, 2000), but it has gained recent popularity following widespread media reports that Google has created an hallucinating neural network (Figure 11, on p. 227). However, looking at these images, the nature of the generated images varies considerably. This suggests that it is not enough to have a functioning network to understand hallucinations, one must also understand how the properties of that network change to allow particular hallucinatory outputs.

8. Formalizing the Properties of Hallucinating Networks

A substantial body of work has shown how understanding the properties of the early visual cortex can cast light on the character of simple geometric visual hallucinations (Cowan, 2014). The organization of cortical connectivity within these areas constrains the possible modes of variation across them in mathematically describable ways that account deterministically for the limited number and particular character of these types of hallucination.

However, the complexity of visual processing beyond the early visual cortex suggests that less deterministic models may be needed to model neuronal dynamics (Tsuda, 2001; Kanamaru, Fujii and Aihara, 2013). We argue above that it is highly likely that hallucinating

networks are states of veridical networks. Exploring the adaptive functionality of the network in maintaining accurate information flow (veridical perception) while aspects of it are compromised may lead to new insights into how hallucinations arise. From a computational perspective, the purpose of vision is to provide an interpretation of an object within a context rather than to describe it exactly (Marr, 1982). In order for the interpretation process to succeed, several different types of information need to be dynamically reconciled (Tsuda, 1984; Érdi and Tsuda, 2002). Thus, time-based expectations from the medial prefrontal cortex and the orbito-frontal cortex are integrated with prototypical three-dimensional models within the default mode network and scene information from the parahippocampal place area; all within shifting attentional focus modulated by the dorsal attentional network. The overall system is optimized to allow functional perception even when incoming information is partial or contradictory.

The structure of this overall system can be modelled by creating processing networks or modules based upon cortical and subcortical areas and linked by information flows across known connecting tracts. Clinical and experimental methods can gather evidence on how these components may be stressed by disease processes. Thus, in-vivo Diffusion Tensor Imaging can highlight changes in connecting tracts, while Voxel Based Morphometry of structural MRIs will show changes within specific brain areas. These structural changes can then be related to variation found in functional imaging, including fMRI and EEG during hallucinations. Neuropathological and clinical studies can relate neurotransmitter changes to the risk of hallucinations. However, we have already seen how structural and functional changes can be distributed widely across networks. How can we know which changes are relevant to hallucinations?

Modelling hallucinatory networks is in its initial stages (Series, Reichert and Storkey, 2010; Fujii *et al.*, 2015; Tsukada *et al.*, 2015a,b) but is showing promising results in terms of exploring the relationship between the capacity to generate hallucinatory images and impaired and preserved function in networks. Thus, Tsukada *et al.* and Fujii *et al.* made a computational model of the prefrontal-inferotemporal cortex (PFC-IT) complex. In this, the PFC network consists of pyramidal cells and fast-spiking GABAergic interneurons which receives both contextual and visual image inputs, and the IT network consists of a recurrent network of pyramidal cells with fast-spiking GABAergic interneurons which receives index information from the PFC. The numerical results suggest that partial deficits of visual informa-

tion in the PFC give rise to incorrect index information that does not match to contextual information coming from visual input, which in turn brings about the occurrence of a mismatched visual image in IT — an hallucination.

A minimal model comprises coupled modules corresponding to the early visual cortex (primarily receiving and processing visual input), the later visual cortex (interpreting and perceiving), and associated frontal and medial temporal cortex (which provide mnemonic and contextual information). Sparse dysfunctions within modules or tracts can be modelled by pruning of virtual synapses (Suemitsu and Nara, 2005; Soma *et al.*, 2015) and the effects of this on the dynamics of information flow in other parts of the system captured. If there is a relative under-activity of sensory processing, information in other parts of the system will be more influential in perceptual output. For example, there is generally an inverse relationship between activity in attentional and stored information networks — the dorsal attentional and default mode networks; with dopaminergic, cholinergic, and other neurotransmitter networks modulating their balance. The risk of hallucinations in Lewy body disorders is closely associated with disturbances in these neurotransmitters as well as in connecting white matter tracts (leading to pruned synaptic connectivity between various functional modules), raising the possibility that a breakdown in the relationship between modules within the system may account for why people hallucinate rather than fail to perceive.

Modelling the dynamics of hallucinatory episodes needs the network to manifest certain properties, particularly meta-stability and transitory dynamics which match the timescale of hallucinatory episodes. Analogies may be drawn with models of associative memory networks in which information stored in recurrent synaptic connections is able to be retrieved from partial current information. This process can be conceptualized as the dynamic convergence of the system to a particular state (an attractor) which corresponds to a specific memory (Hopfield, 1982; Amari, 1972; Anderson, 1972). These dynamics reflect the components of the system and their connectors; providing a means of relating sparse structural changes to changes in functional state. For instance, a mnemonic network modulated by short-term changes in plasticity has a variety of dynamical states and can both converge on a specific memory state and transit from one memory state to another (Katori *et al.*, 2011; 2013; see also Tsuda, 1992, and Adachi and Aihara, 1997, for an example of transitory memory dynamics). These transitive dynamics are a necessary

feature of perception as well (Tsuda, 2001; 2015). In the ever-changing visual world, objects must both be seen and also rapidly replaced by other perceptions. Modelling studies suggest that these dynamics are sensitive to some aspects of structure, particularly the balance between the strength of recurrent connections and of feed-forward sensory connections (Katori, in press).

Thus modelling may highlight specific aspects of structural change within networks which can then be investigated to see if, in practice, they are associated with hallucinations.

Developing these models further so that they can make testable predictions is the next stage. Empirically, for example, we could further stress compensated systems by modulating the visual information that they have to work with — as in the pareidolia task. Combining these manipulations with novel analytic methods for network-wide activity data such as multi voxel pattern analysis or graph theory may allow specific predictions of theories to be tested.

9. Wider Implications

Beyond our interest in visual hallucinations, developments within this field illustrate a wider shift from impairment-based models of disease or injury to a resilience-based model of brain function in which it is always trying to do its best to maintain function with the capability that it has at any single point. What from one perspective is an aberrant symptom is, from another, a worthwhile price for maintaining a higher degree of functionality than would otherwise be possible.

10. Conclusions

There has been a shift in our understanding of visual hallucinations over the last twenty years or so. It has been recognized that these phenomena are disorders of the distributed visual perceptual system, modulated by links to other systems. Increasing load on these systems, especially core object recognition and attention, decreases robustness and increases the chance of hallucinations, but impairment alone does not create hallucinations. The mechanisms by which the brain dynamically compensates for limited function may be a key mechanism. Thus, hallucinatory episodes may reflect the dynamics of the visual system. Context influences content and response.

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Colour Figures

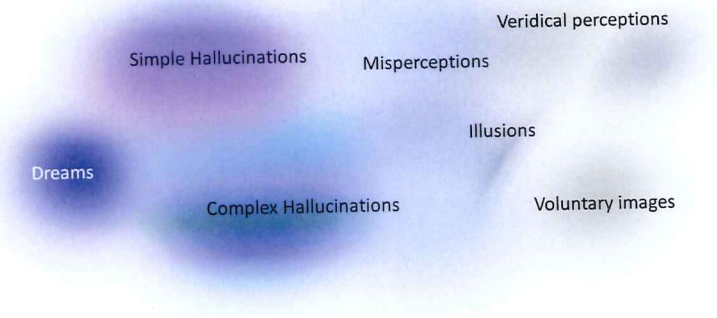

Figure 1. The boundaries of visual experiences are indistinct, with each merging into others.

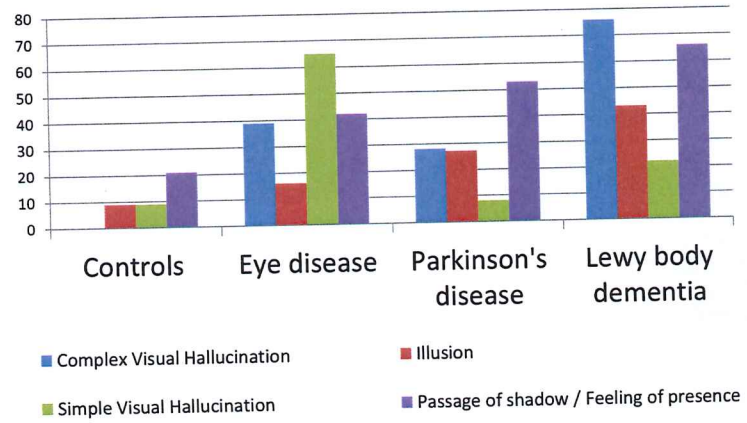

Figure 2. Example of categorization of visual experiences in healthy controls and people with eye disease, Parkinson's disease, and Lewy body dementia (including Parkinson's disease dementia and dementia with Lewy bodies). Figures are percentages of each group who report each type of experience (Urwyler et al., 2015).

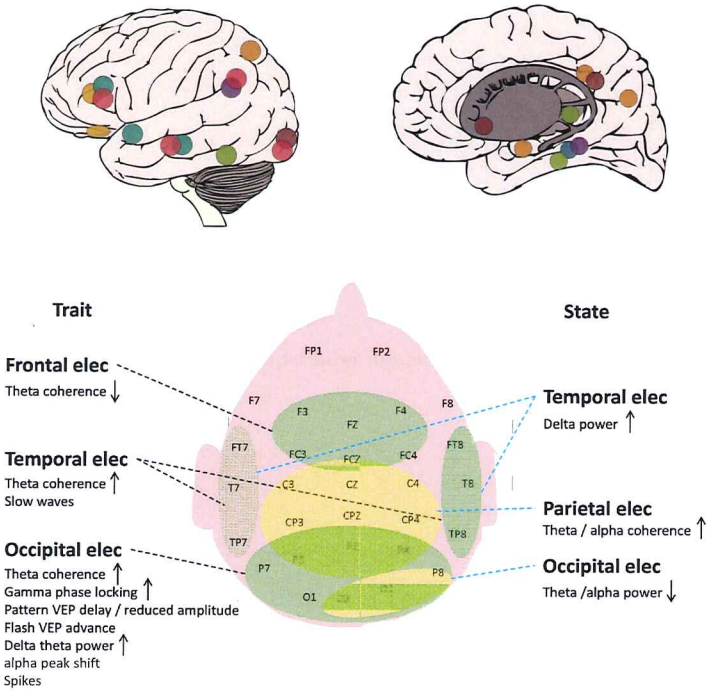

Figure 8. Composite figures illustrating activity change associated with hallucinations in functional imaging (top, Meppelink, 2014) and electrophysiological (bottom, Ffytche, unpublished data, reproduced with permission) measures across disorders.

Figure 11. Examples of 'hallucinatory' images created from a trained neural network. The upper pair (original on left) illustrates the replacement of a visual environment with a panoramic 'hallucination' in which a replacement scene is created. The lower pair illustrates a single image in the context of a relatively preserved background.