

## Review

## Brain Mechanisms of Reality Monitoring

Jon S. Simons,<sup>1,\*</sup> Jane R. Garrison,<sup>1</sup> and Marcia K. Johnson<sup>2</sup>

**Reality monitoring processes are necessary for discriminating between internally generated information and information that originated in the outside world. They help us to identify our thoughts, feelings, and imaginations, and to distinguish them from events we may have experienced or have been told about by someone else. Reality monitoring errors range from confusions between real and imagined experiences, that are byproducts of normal cognition, to symptoms of mental illness such as hallucinations. Recent advances support an emerging neurocognitive characterization of reality monitoring that provides insights into its underlying operating principles and neural mechanisms, the differing ways in which impairment may occur in health and disease, and the potential for rehabilitation strategies to be devised that might help those who experience clinically significant reality monitoring disruption.**

**How Do We Know What Is Real?**

Thinking back over your life, you can often find yourself mentally transported back in time, reliving a past episode, sometimes in vivid detail. Except – how do you know that you were actually there when the event originally took place? How can you be sure that you are remembering a faithful representation of what happened, as opposed to an event you might have previously imagined, or a story told to you by someone else? In short, how do you determine whether your memories are real?

One prominent theory, the Source Monitoring Framework, proposes that there are decision processes involved in making attributions about the origin of information that comes to mind, including discriminating information that was generated by internal cognitive functions, such as thought and imagination, from information that was derived from the outside world by perceptual processes ('reality monitoring' [1,2]). According to this framework, memories do not contain propositional tags that directly specify their source. Instead, we make attributions about the origin of a mental experience by considering its features in light of assumptions about the characteristics that tend to be associated with various sources. For example, a person might infer that an apparent 'memory' rich in visuoperceptual detail is likely to be real ('I can remember what the dentist's office looked like' [3]) whereas one comprising mainly traces of internally generated thoughts might instead have been imagined ('I could remember I had a very specific reason for making the association'; 'I made the decision by knowing what my train of thought was during the exercise' [4]).

Reality monitoring errors tend to involve misidentifying internally generated events as being real, for example misattributing particularly vivid imaginations to perception, or assuming that the absence of memory for cognitive operations indicates that a memory is unlikely to have been self-generated [4], although misattributions in the other direction also occur, such as in cryptomnesia [5]. Similarity between potential sources increases the likelihood of source errors [6]. For example, misattribution errors are more common for auditory than visual stimuli,

## Trends

Mounting evidence identifies anterior prefrontal cortex as playing a key role in reality monitoring, the ability to distinguish internally from externally generated information.

Individual differences in reality monitoring performance in healthy volunteers are associated with variability in functional activity and structural morphology in this region of the brain.

Differences are also seen in clinical conditions such as schizophrenia, in which people sometimes experience disturbed awareness of what is real.

Dysfunction in anterior prefrontal cortex increases vulnerability to misidentify internally generated information as external in origin, providing a possible neurocognitive basis for psychiatric symptoms such as hallucinations.

<sup>1</sup>Department of Psychology, University of Cambridge, Cambridge, UK  
<sup>2</sup>Department of Psychology, Yale University, New Haven, CT, USA

\*Correspondence: [jss30@cam.ac.uk](mailto:jss30@cam.ac.uk) (J.S. Simons).

perhaps because ‘inner speech’ and real speech tend to be more similar than visual imagery and actual visual perceptions and, hence, are more vulnerable to confusion [7]. It is important to note that the features that are activated when a ‘target’ memory is cued are determined by processes engaged during encoding of the event (e.g., the quality of feature binding), during the interval between the initial event and the act of ‘remembering’ (e.g., the number of reactivations), and during retrieval (e.g., the cue eliciting the memory). In addition, features from other events can be activated at any point, potentially influencing the characteristics of the remembered event [8]. In addition to the phenomenal qualities of mental experiences, reality monitoring may also involve explicit retrieval of supporting or conflicting information, and may be influenced by prior knowledge, beliefs, and motives [9]. Thus, there are multiple factors operating during encoding and retrieval, as well as in the intervening period, that can produce source misattributions in healthy individuals, and multiple ways that processes can be disrupted in clinical populations.

In the past few years a number of laboratories around the world have explored the brain mechanisms underlying reality monitoring processes using cognitive neuroscience methods including functional brain imaging of healthy volunteers and studies of neurological, psychiatric, and developmental disorders, as well as of normal aging [10]. The aim has been to understand how the brain supports our capacity to determine the sources of mental experiences, including distinguishing what is real from what we have imagined, an ability that is vital for maintaining confidence in our memories, and in understanding ourselves as individuals in the world with a past and a future. In characterizing how these processes might be instantiated in the brain, we can better understand the way in which they may break down in disorders such as schizophrenia, in which a person’s relation to reality can be altered in ways that disrupt their everyday functioning.

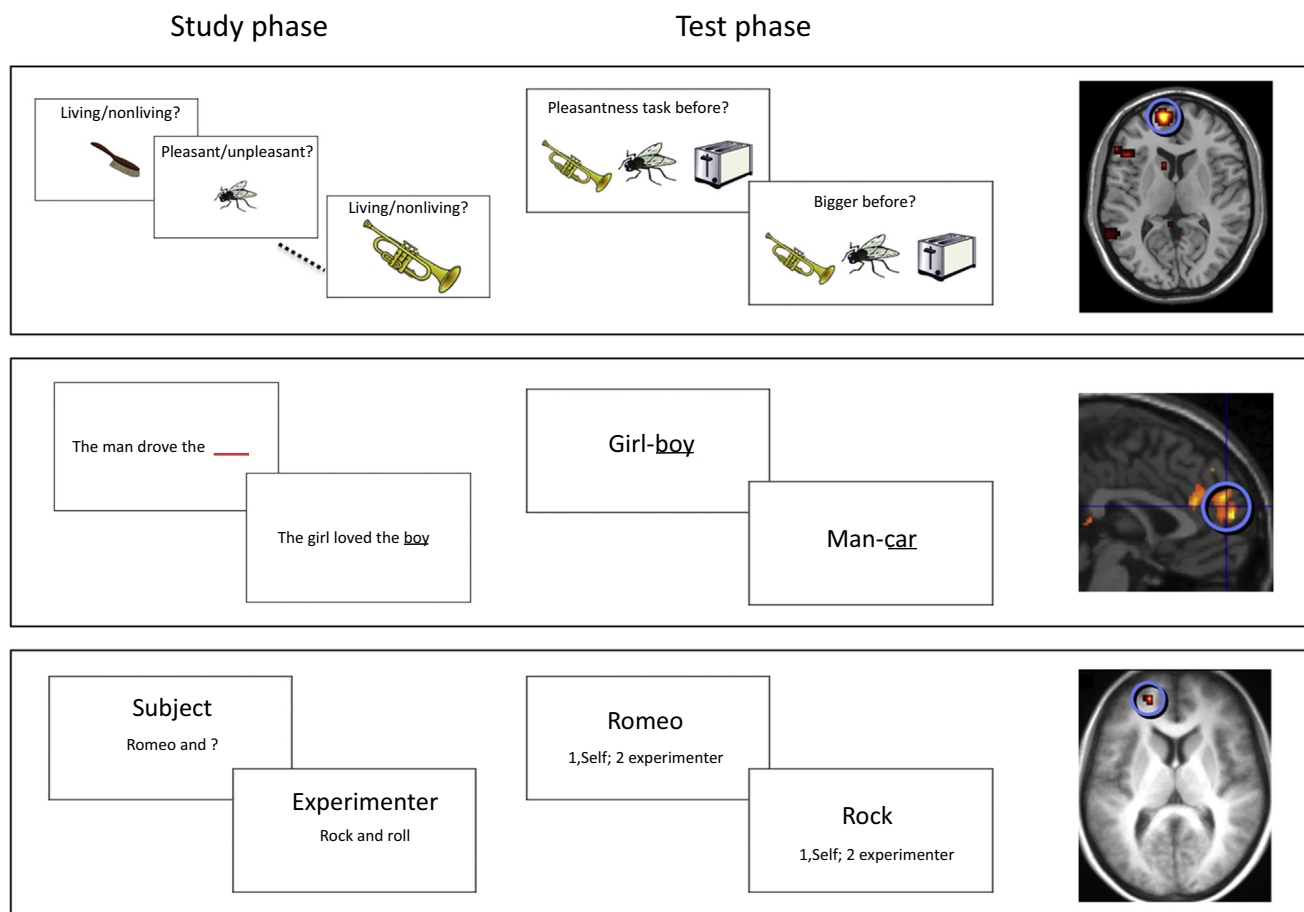
### Anterior Prefrontal Cortex (PFC) and Reality Monitoring

Neuropsychological and neuroimaging studies have identified a network of brain regions involved in the recollection of source information, which include prefrontal, medial temporal, and parietal cortices [10–12]. In broad terms, regions of PFC are thought to provide cognitive control over the encoding and retrieval of feature representations that are bound together in a spatial frame by the hippocampus and further integrated by parietal cortex into a first-person perspective that supports the subjective experience of remembering [9,13]. The role of the PFC appears to be particularly crucial for source memory, and lesions to the frontal lobes typically cause severe difficulties with the recollection of such contextual details even when old/new item recognition is unimpaired [14,15]. Distinct prefrontal regions may make separable functional contributions to source memory [11], with ventrolateral PFC subregions linked to the specification of retrieval cues and the maintenance of retrieved information, and the dorsolateral PFC exhibiting activity during post-retrieval monitoring [16–18].

One region that has emerged as playing a key role in reality monitoring is the anterior PFC, an area right at the front of the brain that, in relative terms, is roughly twice as large in the human brain than in even the great apes [19]. It has lower cellular density and higher dendritic complexity than comparable cortical regions [20], and is thought to be among the last areas to achieve myelination [21], enabling nerve cells to transmit information more rapidly and facilitating more complex cognitive abilities. As such, although the functions performed by this area are not well understood, they have generally been considered likely to be among the ‘higher’ levels of human complex cognition [21–24]. The role played by the anterior PFC in memory has been difficult to characterize. Several neuroimaging experiments published in the early 2000s reported activation in this region during the recollection of source details [16,18,25,26], but this was not consistently observed [17,27,28]. An absence of anterior PFC activity could of course always be attributable to insufficient experimental power or to

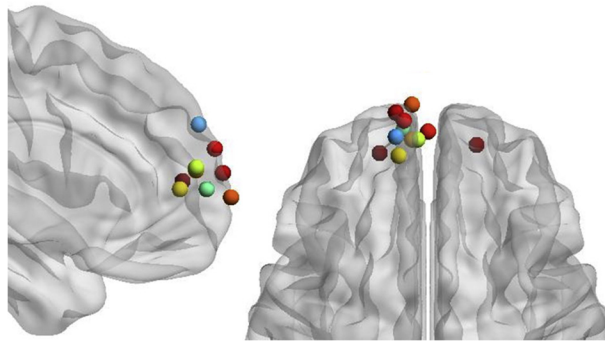
susceptibility in the fMRI signal owing to the proximity of the anterior PFC to the sinus area. However, another hypothesis is that the discrepancy between studies might have arisen because the types of information that participants were being asked to remember differed according to whether the information had been generated internally or externally at the time of encoding [29].

In the past decade or so this hypothesis has been tested by a number of experiments from different laboratories that have manipulated the recollection of internally generated and externally derived source details (Figure 1). Although there is some variability in the patterns of activity elicited by such tasks, these studies have consistently identified the medial aspect of the anterior PFC in particular as exhibiting differential activity during the retrieval of internal versus external aspects of context (Figure 2). For example, medial anterior PFC responses distinguish recollection of the encoding task undertaken compared to remembering where on the screen [29–31] or when in time [32,33] stimuli were presented, or remembering the size in which they were displayed [25,34]. The same region is involved in remembering whether verbal phrases were previously presented in full on the screen (e.g., ‘bacon and eggs’), or whether a word was missing which participants needed to imagine (e.g., ‘bacon and ?’) to complete the phrase



Trends in Cognitive Sciences

**Figure 1. Three Examples Illustrating the Variety of Reality Monitoring Tasks and Activity Elicited in the Anterior Prefrontal Cortex.** (Top panel) Memory for encoding task versus stimulus size [34] (fMRI image courtesy of Ian G. Dobbins). (Middle panel) Remembering whether words were seen or imagined [35] (fMRI image reprinted, with permission, from Elsevier). (Bottom panel) Distinguishing between word pairs read aloud by the participant herself versus the experimenter [37] (fMRI image reprinted, with permission, from MIT Press).

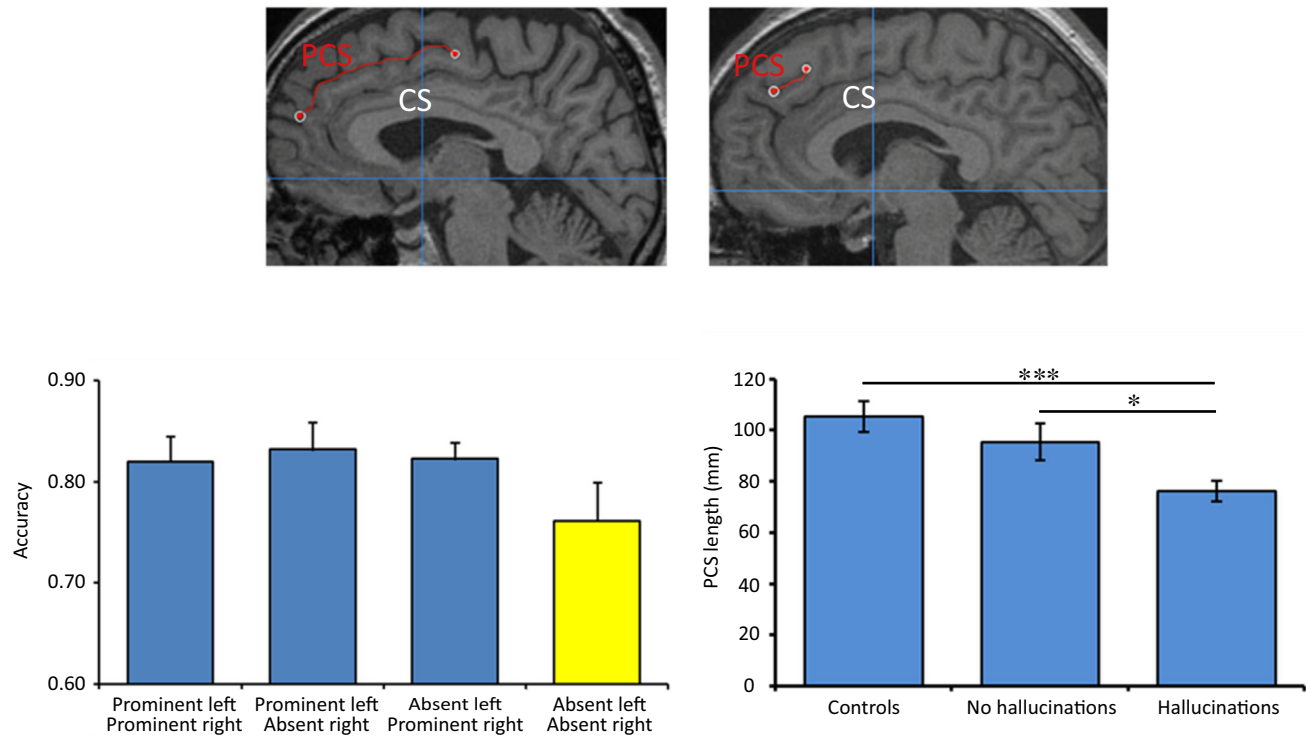


Trends in Cognitive Sciences

Figure 2. Locations of Medial Anterior Prefrontal Cortex Activity Reported by 12 fMRI Studies of Reality Monitoring in Healthy Volunteers.

themselves [30,35]. Likewise, medial anterior PFC is differentially engaged during recollection of whether a visual object was previously seen or imagined by participants [36], as well as when judging whether oneself or another person performed a particular action with stimuli [37–39]. The sensitivity of this region to reality monitoring distinctions is apparent regardless of whether words, faces, or objects are being remembered [29,32,34,36], suggesting that the effect is independent of stimulus type. Moreover, medial anterior activation has been observed irrespective of whether the ‘internal’ or ‘external’ condition is associated with lower recollection accuracy and longer response times, or vice versa, or whether such behavioral factors are matched between conditions [29,32], suggesting that an account in terms of differential task difficulty is unlikely to be sufficient. Possible explanations of the observed activity include that medial anterior PFC represents records of cognitive operations involved in self-generation, or is involved in self-referential processing or self-representation, or that the region operates as a gateway, biasing attention between self-generated and externally generated information, irrespective of whether that information is experienced currently or in the past [21,40].

Not everyone exhibits perfect reality monitoring performance, of course. Performance varies considerably even in apparently healthy individuals, with some people reliably able to distinguish internally from externally generated stimuli whereas others perform closer to chance levels [41]. Consistent with the functional neuroimaging evidence discussed above, recent research points to these individual differences as having a specific brain structural basis in the medial PFC. One structural landmark in this region of the brain that exhibits considerable morphological variability within the general population is the paracingulate sulcus (PCS), which lies dorsal and parallel to the cingulate sulcus (Figure 3). The PCS, which is prominent in roughly half of the normal population, is a tertiary sulcus, one of the last structural folds to develop before birth and, due to a combination of genetic and environmental influences, varies considerably in size between individuals [42]. This brain structure variation appears to be linked to reality monitoring ability: healthy, apparently cognitively intact, adults whose MRI scans indicate bilateral absence of the PCS are significantly less accurate in reality monitoring decisions than people with a prominent PCS on at least one side of the brain [41]. PCS reductions are typically associated with increased grey matter volume in the surrounding anterior cingulate cortex [43] and, consistent with this observation, voxel-based analyses reveal that reality monitoring performance correlates negatively with medial PFC volume [41]. Reduced sulcal folding and increased surrounding cortical volume may reflect weakened local and long-range connectivity, according to theories of morphogenesis [44,45]. These findings thus suggest an explanation for individual differences in reality monitoring ability as reflecting, in part, variations in connectivity between the medial PFC and other cortical regions involved in processing the sensory/perceptual and other features that constitute our memories.



Trends in Cognitive Sciences

**Figure 3.** Paracingulate Sulcus (PCS) and Reality Monitoring. (Top panel) The PCS (shown in red), located in the medial prefrontal cortex (PFC) dorsal to the cingulate sulcus (CS), differs in length considerably between individuals (reprinted, with permission, from Nature Publishing Group). (Bottom left panel) Reduced reality monitoring performance in healthy volunteers in whom PCS is absent in both hemispheres [41]. (Bottom right panel) PCS length differentiates hallucination status in patients with schizophrenia, as well as distinguishing patients with schizophrenia from healthy control participants [78] (reprinted, with permission, from Nature Publishing Group). Asterisks indicate statistical significance.

### Reality Monitoring in Schizophrenia

One of the applications of the work seeking to characterize the brain mechanisms of reality monitoring has been to inform understanding of the cognitive dysfunction seen in clinical conditions such as schizophrenia. Although schizophrenia can vary in its presentation, among the positive symptoms often observed are hallucinations, such as hearing voices when none are present. For example, a person with schizophrenia might imagine a voice conveying a specific message, and misidentify that voice as being real, coming from another person. Hallucinations also occur in other psychiatric conditions including bipolar disorder, major depressive disorder, borderline or schizotypal personality disorder, post-traumatic stress disorder, and dissociative identity disorder [46–48], but affect between 60% and 80% of patients with a schizophrenia diagnosis [49,50]. Auditory and visual hallucinations are the most common forms, with a prevalence of around 59% and 27% in schizophrenia, respectively [51], but olfactory, tactile, somatic, and gustatory hallucinations have also been reported [52–54]. Activity associated with hallucinations is often observed in sensory processing areas, such as the superior temporal gyrus during auditory hallucinations and extrastriate cortex during visual hallucinations [55], suggesting that separable modality-specific impairments contribute to different types of hallucinations across patients. One possibility is that hallucinations primarily reflect unusually vivid internally generated experiences represented in one or more of these modality-specific processing areas, experiences that are so vivid that they seem to be external events. In addition, it is possible that hallucinations may in part result from a more central difficulty in discriminating between perceived and imagined information, perhaps

because self-generated events have attenuated or missing records of the cognitive or motor operations that produced them [56].

Despite the interpretational complexities inherent in studying a heterogeneous syndrome such as schizophrenia, a number of predictions have been tested that arise from the hypothesis linking disrupted reality monitoring with hallucinations, and the assumption that some common processes are involved in distinguishing reality from imagination both retrospectively and in real time. One prediction is that individuals with schizophrenia should be impaired on seen versus imagined memory tasks that have been shown to elicit anterior PFC activity in healthy volunteers. Of 20 studies of reality monitoring in schizophrenia surveyed for this article (Table 1), involving a total of 700 patients and 505 controls, 19 studies reported impaired reality monitoring in the patients, including four studies in which the deficit was found to be specific to reality monitoring, with item recognition memory being preserved [57–60]. A second prediction, if hallucinations result from misidentifying imagined stimuli as being real, is that an externalization bias should be observed in schizophrenia during reality monitoring performance, with more errors on self-generated than externally generated trials, and that this bias should be greater in patients with schizophrenia who experience hallucinations than in those who do not. Of the four studies reporting a specific reality monitoring impairment in schizophrenia, three noted that the deficit only (or disproportionately) involved the misidentification of internally generated stimuli as having been externally generated [58–60]. Furthermore, all five studies that compared reality monitoring in patients with and without hallucinations, and measured externalization bias, observed a greater likelihood of misidentifying internally generated stimuli as being real in the patients who experienced hallucinations ([57,61–64], see also [65,66] for previous reviews highlighting such associations).

A third prediction is that the anterior PFC region activated in healthy volunteers during reality monitoring performance should be among the areas that are dysfunctional in patients with schizophrenia. Consistent with this prediction, the anterior PFC region linked to reality monitoring in healthy volunteers overlaps closely [30] with one of the areas that consistently exhibit reduced activity in patients with schizophrenia compared with controls during performance of a range of cognitive tasks [67–71]. Moreover, lower anterior PFC activity in healthy individuals during reality monitoring correlates with proneness to psychosis and schizotypal trait expression [37], an effect that is also observed in adolescents at risk of developing schizophrenia [72], suggesting its potential as a possible marker in young people of those with heightened likelihood of converting from prodromal to full psychosis [73].

A fourth prediction is that healthy volunteers who exhibit reduced levels of activity in anterior PFC should make more of the externalization misattribution errors often observed in schizophrenia. Accordingly, analysis of activity in the anterior PFC across participants during reality monitoring performance has revealed a significant negative correlation with the likelihood of mistakenly endorsing imagined items as having been seen [30]. A fifth prediction is that patients with schizophrenia should exhibit disproportionately reduced activity in anterior PFC during performance of reality monitoring tasks, an effect that has been reported in three studies to date [60,74,112], and which may be partly ameliorated by cognitive training interventions [74] (although this latter possibility requires replication in larger samples). A sixth prediction, if hallucinations in schizophrenia are attributable to overstimulation of sensory processing areas and reality monitoring dysfunction, is that altered functional connectivity should be observed between posterior sensory cortices and anterior PFC. Accordingly, several studies have reported impaired functional integration between superior temporal cortex and medial regions of anterior PFC associated with misattribution by patients with schizophrenia of their own speech as that of somebody else [75,76]. Finally, based on the specific structural basis for reality monitoring identified in the PCS [41], hallucinations in schizophrenia should be



Table 1. Studies of Reality Monitoring in Schizophrenia

Patients with Schizophrenia versus Healthy Controls							
Study	Task	Subjects		Impairment in		Increased	Refs
		Patients (n)	Controls (n)	Recognition memory	Reality monitoring	Externalization bias	
Anselmetti <i>et al.</i> (2007)	Word-stem completion	45	54	Yes	Yes	Yes	[99]
Bentall <i>et al.</i> (1991)	Word-stem completion	22	22	–	Yes	No	[62]
Brebion <i>et al.</i> (2000)	Semantic association	40	40	–	Yes	–	[63]
Brunelin <i>et al.</i> (2007)	Word recognition	15	15	–	Yes	–	[64]
Brunelin <i>et al.</i> (2008)	Word recognition	30	24	–	Yes	–	[100]
Fisher <i>et al.</i> (2008)	Sentence completion	91	30	Yes	Yes	Yes	[101]
Franck <i>et al.</i> (2000)	Word recognition	17	17	–	Yes	Yes	[102]
Harvey <i>et al.</i> (1988)	Word recognition	26	25	–	Yes	–	[103]
Keefe <i>et al.</i> (2002)	Word-stem completion	29	19	Yes	Yes	Yes	[104]
Moritz <i>et al.</i> (2003)	Semantic association	30	21	Yes	Yes	No	[105]
Moritz <i>et al.</i> (2005)	Semantic association	30	15	Yes	No	No	[106]
Nienow and Docherty (2004)	Sentence completion	52	52	Yes	Yes	Yes	[107]
Seal <i>et al.</i> (1997)	Semantic association	21	15	No	Yes	No	[57]
Stephane <i>et al.</i> (2010)	Sentence recognition	39	26	No	Yes	Yes	[58]
Subramaniam <i>et al.</i> (2012)	Sentence completion	31	16	–	Yes	Yes	[74]
Szoke <i>et al.</i> (2009)	Semantic association	54	41	–	Yes	No	[108]
Vinogradov <i>et al.</i> (1997)	Sentence completion	26	21	No	Yes	Yes	[59]
Vinogradov <i>et al.</i> (2008)	Sentence completion	8	8	No	Yes	Yes	[60]
Waters <i>et al.</i> (2004)	Object pairing	43	24	Yes	Yes	No	[109]
Woodward <i>et al.</i> (2007)	Sentence completion	51	20	Yes	Yes	No	[61]
Total subjects		700	505				
Patients with Schizophrenia: Hallucinations versus Non-Hallucinations							
Study	Task	Subjects		Impairment in		Increased	Refs
		Hall. (n) <sup>a</sup>	Non-hall. (n) <sup>a</sup>	Recognition memory	Reality monitoring	Externalization bias	
Bentall <i>et al.</i> (1991)	Word-stem completion	22	16	–	No	Yes	[62]
Brebion <i>et al.</i> (2000)	Semantic association	22	18	–	No	Yes	[63]
Brunelin <i>et al.</i> (2006)	Word recognition	30	31	No	Yes	Yes	[110]
Seal <i>et al.</i> (1997)	Semantic association	10	11	–	No	Yes	[57]
Waters <i>et al.</i> (2006)	Object pairing	19	24	No	Yes	–	[111]
Woodward <i>et al.</i> (2007)	Sentence completion	16	35	No	No	Y;1;1;es	[61]
Total subjects		119	135				

<sup>a</sup>Hall., hallucinations; non-hall., non-hallucinations.

associated with differences in PCS morphology, among other areas. Providing support for this prediction, a quantitative measure of PCS length has been shown to be capable of distinguishing not only patients with schizophrenia from healthy controls [77], but also patients who have experienced hallucinations from patients with no history of such symptoms (Figure 3) [78]. The PCS reduction in patients with hallucinations appears to be evident irrespective of the sensory modality in which they were experienced (e.g., auditory, visual, tactile, olfactory), consistent with the observation described above that healthy volunteers exhibit anterior PFC activity during reality monitoring regardless of the type of stimuli being remembered [29,32,34,36].

### Reality Monitoring and Hallucinations

These findings provide evidence that reality monitoring impairment may be a key component of the cognitive basis for the hallucinations experienced by many patients with schizophrenia. However, it is not only people with a mental health diagnosis who experience hallucinations. Approximately 1% of the general population report hearing voices when no speaker is present, but do not seek professional help or receive a clinical diagnosis [79]. Phenomenally, the auditory hallucination experience tends to be similar in clinical and non-clinical voice-hearers in terms of loudness, involvement of others' voices, number of voices, and so forth, but can differ in frequency, duration, and content, with patients typically experiencing negative voices whereas non-clinical individuals are sometimes more likely to report neutral or pleasant content to their hallucinations [80–82]. Recent interest has focused on whether there might be a common neurocognitive basis for hallucinations in clinical and non-clinical groups. If reduced reality monitoring ability is part of the explanation for the hallucinations observed in schizophrenia, can it also account for the symptoms experienced by non-clinical voice-hearers?

Evidence consistent with a common underlying mechanism comes from one study of reality monitoring in healthy volunteers which found that participants who were prone to experience hallucinations were more likely than other participants to misidentify self-generated words as having been spoken by the experimenter [83]. However, two more recent studies involving healthy individuals with a proneness to hallucinations found no evidence of an impairment in source or reality monitoring, or of an increased externalization bias, in such non-clinical voice-hearers [84,85]. Evidence from neuroimaging suggests common hallucination-related activity in auditory processing areas such as the superior temporal gyrus in clinical and non-clinical groups [86], and a correlation between the auditory hallucination proneness scores of non-clinical participants and activity in superior temporal gyrus when imagining voices they subsequently misidentified as being heard [87]. Clinical and non-clinical voice-hearers also have similar aberrant microstructure of the arcuate fasciculus connection between frontal and temporal cortices [88,89], but there is so far no evidence as to whether hallucinations experienced by non-clinical individuals reflect the same pattern of anterior PFC dysfunction that has been observed in patients with schizophrenia.

It may well be that there is more than one route by which hallucinations might occur in clinical and non-clinical groups. Hallucinations are sometimes experienced spontaneously in healthy individuals during periods of sensory deprivation [50], auditory hallucinations can follow the onset of deafness [90], and visual hallucinations can occur following visual cortex injury [91]. These observations fit with the proposal that hyperactivation of sensory processing cortices might provide the perceptual content for hallucinatory experiences [92]. Consistent with this proposal is evidence that, in healthy individuals during periods of silence, there is spontaneous random activity in speech-sensitive auditory processing areas within the superior temporal gyrus, together with associated activation in the anterior cingulate region of the medial PFC [93]. Thus, it is possible that a hallucination is initiated by spontaneous random activity in sensory processing areas, such as the superior temporal gyrus in the case of auditory hallucinations, reflecting spontaneous 'inner speech' and/or remembered speech, which may occur with greater intensity or frequency during periods of stress or heightened emotion [81]. In healthy individuals who do not experience hallucinations, such sensory activity may be correctly identified as being internally generated by reality monitoring processes supported by anterior medial PFC, and experienced as an imagined voice. In patients with schizophrenia who experience hallucinations, spontaneous sensory hyperactivity may be even more intense, accompanied by hypoactivation of medial PFC regions (consistent with studies of connectivity [75,76]), resulting in a reality monitoring impairment that leads to failure to recognize the activity as self-generated and to the experience of a hallucination. In non-clinical voice-hearers, the spontaneous activity in sensory processing areas may either be of such intensity, or be



sufficiently unusual in character (perhaps due to emotional stress, trauma, or tiredness [81]), that an otherwise intact reality monitoring system fails to recognize the stimuli as generated internally, and a sporadic hallucination is experienced. A multifactor model such as this (see also [92,94]) can also explain why some participant groups, such as older adults [95], people with developmental disorders such as autism [96], and healthy volunteers with bilateral PCS absence [41], may exhibit reductions in performance on reality monitoring tasks but do not apparently experience hallucinations. These findings may be less compatible with single factor accounts, such as those involving prediction error (e.g., [97]).

### Concluding Remarks

There is still much to discover about the brain mechanisms underlying reality monitoring, and the ways in which they may fail in health and disease, impairing the accuracy of judgments about what is real (see Outstanding Questions). Reality monitoring processes are fundamental for maintaining an understanding of the self as a distinct, conscious agent interacting with the world, perceiving and interpreting external information relating to events happening around us and generating our own thoughts and imaginations and responses. Generally, people are able to keep the 'internal' and 'external' worlds distinguished sufficiently from one another to function under everyday circumstances but, occasionally, anyone may confuse real experiences with those they might have been told about or imagined or dreamt. Some individuals face a greater difficulty in reality monitoring, perhaps reflecting specific individual differences in the general population or, in more severe cases, dysfunction caused by neurological or psychiatric disease.

The evidence from cognitive neuroscience research to date converges to suggest that the anterior PFC region of the brain is central to reality monitoring ability and disability. It is a key component of brain networks that are engaged when distinguishing internally and externally generated information, and its disruption is associated with misattributions of reality, confusing internally generated information with events taking place in the outside world. It appears to play an important, and previously underappreciated, role in the psychotic experiences that characterize disorders such as schizophrenia. The processes underlying reality monitoring are, similarly to many higher cognitive functions, complex [9], but progress has been made by laboratories around the world in recent years leading to a greater understanding of the brain regions, especially the anterior PFC, that subserve reality monitoring mechanisms. An important next challenge will be to apply this knowledge to the development of cognitive training techniques and other rehabilitation interventions aimed at enhancing the ability to distinguish between real and imagined experiences in people whose sense of reality may be disturbed. Preliminary evidence suggests that such an approach might have potential [74,98], but researchers are only at the beginning of this road and there is much further work to do before it can be determined whether the potential is real.

### Acknowledgments

Preparation of this article was supported by a James S. McDonnell Foundation Scholar Award to J.S.S., and by the University of Cambridge Behavioural and Clinical Neuroscience Institute, funded by a joint award from the Medical Research Council and the Wellcome Trust.

### References

1. Johnson, M.K. and Raye, C.L. (1981) Reality monitoring. *Psychol. Rev.* 88, 67–85
2. Johnson, M.K. et al. (1993) Source monitoring. *Psychol. Bull.* 114, 3–28
3. Johnson, M.K. et al. (1988) Phenomenal characteristics of memories for perceived and imagined autobiographical events. *J. Exp. Psychol. Gen.* 117, 371–376
4. Johnson, M.K. et al. (1981) Cognitive operations and decision bias in reality monitoring. *Am. J. Psychol.* 94, 37–64
5. Macrae, C.N. et al. (1999) Contexts of cryptomnesia: may the source be with you. *Soc. Cognit.* 17, 273–297
6. Johnson, M.K. et al. (1988) The consequences for memory of imagining in another person's voice. *Mem. Cognit.* 16, 337–342

### Outstanding Questions

Although much has been learned in recent years about the mechanisms of reality monitoring, and the way they may be impaired in neurological and psychiatric disorders, there is much still to be discovered. In particular:

Which regional and interregional brain mechanisms, and related cognitive processes, might explain the intriguing observations that poor reality monitoring performance in healthy individuals is associated not only with reduced anterior PFC activity but also with PCS reductions and greater volume of surrounding cortex?

What are the common and distinct components of neural activity associated with reality monitoring tasks that require a distinction between self and other, and internal source monitoring between self-generated activities (e.g., imagined and performed)?

Do hallucinations experienced by non-clinical individuals without a psychiatric diagnosis reflect a similar pattern of anterior PFC dysfunction and reduced PCS length to that observed in patients with schizophrenia? Are similar functional and structural markers also present in prodromal individuals who subsequently progress to a diagnosis of schizophrenia?

What can explain the subjective differences between hallucinations typically experienced by patients with schizophrenia and by non-clinical individuals with a proneness to psychosis? Why are some people with hallucinations fully aware that their experiences are erroneous perceptions, but others are not?

Does the range of findings concerning hallucinations require a multiple factor explanation, or can they be explained equally fully by more parsimonious single factor accounts, such as prediction error?

What are the similarities and differences in dysfunction in the brain mechanisms underlying reality monitoring processes that lead to hallucinations, confabulations, and delusions?

Can interventions that involve cognitive training, brain stimulation, or neuro-feedback be developed according to

7. Garrison, J.R. *et al.* (2017) Monitoring what is real: the effects of modality and action on accuracy and type of reality monitoring error. *Cortex* 87, 108–117
8. Lyle, K.B. and Johnson, M.K. (2006) Importing perceived features into false memories. *Memory* 14, 197–213
9. Johnson, M.K. *et al.* (2012) The cognitive neuroscience of true and false memories. In *True and False Recovered Memories: Toward a Reconciliation of the Debate* (Bell, R.F., ed.), pp. 15–52, Springer Science + Business Media
10. Mitchell, K.J. and Johnson, M.K. (2009) Source monitoring 15 years later: what have we learned from fMRI about the neural mechanisms of source memory? *Psychol. Bull.* 135, 638–677
11. Simons, J.S. and Spiers, H.J. (2003) Prefrontal and medial temporal lobe interactions in long-term memory. *Nat. Rev. Neurosci.* 4, 637–648
12. Rugg, M.D. and Vilberg, K.L. (2013) Brain networks underlying episodic memory retrieval. *Curr. Opin. Neurobiol.* 23, 255–260
13. Moscovitch, M. *et al.* (2016) Episodic memory and beyond: the hippocampus and neocortex in transformation. *Annu. Rev. Psychol.* 67, 105–134
14. Janowsky, J.S. *et al.* (1989) Source memory impairment in patients with frontal lobe lesions. *Neuropsychologia* 27, 1043–1056
15. Simons, J.S. *et al.* (2002) Recollection-based memory in frontotemporal dementia: implications for theories of long-term memory. *Brain* 125, 2523–2536
16. Rugg, M.D. *et al.* (1999) The role of the prefrontal cortex in recognition memory and memory for source: an fMRI study. *NeuroImage* 10, 520–529
17. Henson, R.N.A. *et al.* (1999) Right prefrontal cortex and episodic memory retrieval: a functional MRI test of the monitoring hypothesis. *Brain* 122, 1367–1381
18. Dobbins, I.G. *et al.* (2002) Executive control during episodic retrieval: multiple prefrontal processes subservise source memory. *Neuron* 35, 989–996
19. Semendeferi, K. *et al.* (2001) Prefrontal cortex in humans and apes: a comparative study of area 10. *Am. J. Phys. Anthropol.* 114, 224–241
20. Jacobs, B. *et al.* (2001) Regional dendritic and spine variation in human cerebral cortex: a quantitative golgi study. *Cereb. Cortex* 11, 558–571
21. Burgess, P.W. *et al.* (2005) The gateway hypothesis of rostral prefrontal cortex (area 10) function. In *Measuring the Mind: Speed, Control, and Age* (Duncan, J., ed.), pp. 217–248, Oxford University Press
22. Koechlin, E. *et al.* (1999) The role of the anterior prefrontal cortex in human cognition. *Nature* 399, 148–151
23. Ramnani, N. and Owen, A.M. (2004) Anterior prefrontal cortex: insights into function from anatomy and neuroimaging. *Nat. Rev. Neurosci.* 5, 184–194
24. Badre, D. (2008) Cognitive control, hierarchy, and the rostro-caudal organization of the frontal lobes. *Trends Cogn. Sci.* 12, 193–200
25. Ranganath, C. *et al.* (2000) Left anterior prefrontal activation increases with demands to recall specific perceptual information. *J. Neurosci.* 20, RC108
26. Kahn, I. *et al.* (2004) Functional–neuroanatomic correlates of recollection: implications for models of recognition memory. *J. Neurosci.* 24, 4172–4180
27. Nyberg, L. *et al.* (1996) General and specific brain regions involved in encoding and retrieval of events: what, where, and when. *Proc. Natl. Acad. Sci. U. S. A.* 93, 11280–11285
28. Suzuki, M. *et al.* (2002) Neural basis of temporal context memory: a functional MRI study. *NeuroImage* 17, 1790–1796
29. Simons, J.S. *et al.* (2005) Anterior prefrontal cortex and the recollection of contextual information. *Neuropsychologia* 43, 1774–1783
30. Simons, J.S. *et al.* (2006) Discriminating imagined from perceived information engages brain areas implicated in schizophrenia. *NeuroImage* 32, 696–703
31. Gilbert, S.J. *et al.* (2010) The scale of functional specialization within human prefrontal cortex. *J. Neurosci.* 30, 1233–1237
32. Simons, J.S. *et al.* (2005) Distinct roles for lateral and medial anterior prefrontal cortex in contextual recollection. *J. Neurophysiol.* 94, 813–820
33. Turner, M.S. *et al.* (2008) Distinct roles for lateral and medial rostral prefrontal cortex in source monitoring of perceived and imagined events. *Neuropsychologia* 46, 1442–1453
34. Dobbins, I.G. and Wagner, A.D. (2005) Domain-general and domain-sensitive prefrontal mechanisms for recollecting events and detecting novelty. *Cereb. Cortex* 15, 1768–1778
35. Vinogradov, S. *et al.* (2006) Brain activation patterns during memory of cognitive agency. *NeuroImage* 31, 896–905
36. Kensinger, E.A. and Schacter, D.L. (2006) Neural processes underlying memory attribution on a reality-monitoring task. *Cereb. Cortex* 16, 1126–1133
37. Simons, J.S. *et al.* (2008) Separable forms of reality monitoring supported by anterior prefrontal cortex. *J. Cogn. Neurosci.* 20, 447–457
38. Brandt, V.C. *et al.* (2014) Did I turn off the gas? Reality monitoring of everyday actions. *Cogn. Affect. Behav. Neurosci.* 14, 209–219
39. Metzrak, P.D. *et al.* (2015) Functional brain networks involved in reality monitoring. *Neuropsychologia* 75, 50–60
40. Johnson, M.K. (2016) Cognitive neuroscience: applied cognitive psychology. *J. Appl. Res. Mem. Cogn.* 5, 110–120
41. Buda, M. *et al.* (2011) A specific brain structural basis for individual differences in reality monitoring. *J. Neurosci.* 31, 14308–14313
42. Armstrong, E. *et al.* (1995) The ontogeny of human gyrfication. *Cereb. Cortex* 5, 56–63
43. Fornito, A. *et al.* (2008) Variability of the paracingulate sulcus and morphometry of the medial frontal cortex: associations with cortical thickness, surface area, volume, and sulcal depth. *Hum. Brain Mapp.* 29, 222–236
44. Van Essen, D.C. (1997) A tension-based theory of morphogenesis and compact wiring in the central nervous system. *Nature* 385, 313–318
45. Ronan, L. *et al.* (2014) Differential tangential expansion as a mechanism for cortical gyrfication. *Cereb. Cortex* 24, 2219–2228
46. Siegel, R.K. (1984) Hostage hallucinations. Visual imagery induced by isolation and life-threatening stress. *J. Nerv. Ment. Dis.* 172, 264–272
47. Ross, C.A. *et al.* (1990) Schneiderian symptoms in multiple personality disorder and schizophrenia. *Compr. Psychiatry* 31, 111–118
48. Skaf, C.R. *et al.* (2002) Psychotic symptoms in major depressive disorder are associated with reduced regional cerebral blood flow in the subgenual anterior cingulate cortex: a voxel-based single photon emission computed tomography (SPECT) study. *J. Affect. Disord.* 68, 295–305
49. Wing, J.K. *et al.* (1974) *Measurement and Classification of Psychiatric Symptoms*, Cambridge University Press
50. Slade, P.D. and Bentall, R.P. (1988) *Sensory Deception: A Scientific Analysis of Hallucination*, Johns Hopkins University Press
51. Waters, F. *et al.* (2014) Visual hallucinations in the psychosis spectrum and comparative information from neurodegenerative disorders and eye disease. *Schizophr. Bull.* 40, S233–S245
52. Mueser, K.T. *et al.* (1990) Hallucinations in schizophrenia. *Acta Psychiatr. Scand.* 82, 26–29
53. Kopala, L.C. *et al.* (1994) Olfactory hallucinations and olfactory identification ability in patients with schizophrenia and other psychiatric disorders. *Schizophr. Res.* 12, 205–211
54. Shergill, S.S. *et al.* (2001) Modality specific neural correlates of auditory and somatic hallucinations. *J. Neurol. Neurosurg. Psychiatry* 71, 688–690
55. Zmigrod, L. *et al.* (2016) The neural mechanisms of hallucinations: a quantitative meta-analysis of neuroimaging studies. *Neurosci. Biobehav. Rev.* 69, 113–123
56. Frith, C.D. and Done, D.J. (1989) Experiences of alien control in schizophrenia reflect a disorder in the central monitoring of action. *Psychol. Med.* 19, 359–363

theoretical hypotheses about disrupted functions to improve reality monitoring ability and, if so, can they be demonstrated to reduce the incidence of hallucinations in people who experience them?

57. Seal, M.L. *et al.* (1997) Deficits in source monitoring in subjects with auditory hallucinations may be due to differences in verbal intelligence and verbal memory. *Cognit. Neuropsychiatry* 2, 273–290
58. Stéphane, M. *et al.* (2010) Evaluation of speech misattribution bias in schizophrenia. *Psychol. Med.* 40, 741–748
59. Vinogradov, S. *et al.* (1997) Clinical and neurocognitive aspects of source monitoring errors in schizophrenia. *Am. J. Psychiatry* 154, 1530–1537
60. Vinogradov, S. *et al.* (2008) Deficit in a neural correlate of reality monitoring in schizophrenia patients. *Cereb. Cortex* 18, 2532–2539
61. Woodward, T.S. *et al.* (2007) Source monitoring biases and auditory hallucinations. *Cognit. Neuropsychiatry* 12, 477–494
62. Bentall, R.P. *et al.* (1991) Reality monitoring and psychotic hallucinations. *Br. J. Clin. Psychol.* 30, 213–222
63. Brébion, G. *et al.* (2000) Positive symptomatology and source-monitoring failure in schizophrenia – an analysis of symptom-specific effects. *Psychiatry Res.* 95, 119–131
64. Brunelin, J. *et al.* (2007) Impaired verbal source monitoring in schizophrenia: an intermediate trait vulnerability marker? *Schizophr. Res.* 89, 287–292
65. Waters, F. *et al.* (2012) Self-recognition deficits in schizophrenia patients with auditory hallucinations: a meta-analysis of the literature. *Schizophr. Bull.* 38, 741–750
66. Brookwell, M.L. *et al.* (2013) Externalizing biases and hallucinations in source-monitoring, self-monitoring and signal detection studies: a meta-analytic review. *Psychol. Med.* 43, 2465–2475
67. Andreasen, N.C. *et al.* (1996) Schizophrenia and cognitive dysmetria: a positron-emission tomography study of dysfunctional prefrontal-thalamic-cerebellar circuitry. *Proc. Natl. Acad. Sci. U. S. A.* 93, 9985–9990
68. Callicott, J.H. *et al.* (2003) Abnormal fMRI response of the dorsolateral prefrontal cortex in cognitively intact siblings of patients with schizophrenia. *Am. J. Psychiatry* 160, 709–719
69. Whalley, H.C. *et al.* (2004) fMRI correlates of state and trait effects in subjects at genetically enhanced risk of schizophrenia. *Brain* 127, 478–490
70. Fox, M.D. *et al.* (2005) The BOLD onset transient: identification of novel functional differences in schizophrenia. *Neuroimage* 25, 771–782
71. MacDonald, A.W. *et al.* (2005) Specificity of prefrontal dysfunction and context processing deficits to schizophrenia in never-medicated patients with first-episode psychosis. *Am. J. Psychiatry* 162, 475–484
72. Lagioia, A.-L. *et al.* (2011) Neural correlates of reality monitoring during adolescence. *Neuroimage* 55, 1393–1400
73. Cannon, T.D. (2015) How schizophrenia develops: cognitive and brain mechanisms underlying onset of psychosis. *Trends Cogn. Sci.* 19, 744–756
74. Subramaniam, K. *et al.* (2012) Computerized cognitive training restores neural activity within the reality monitoring network in schizophrenia. *Neuron* 73, 842–853
75. Mechelli, A. *et al.* (2007) Misattribution of speech and impaired connectivity in patients with auditory verbal hallucinations. *Hum. Brain Mapp.* 28, 1213–1222
76. Wang, L. *et al.* (2011) Aberrant connectivity during self–other source monitoring in schizophrenia. *Schizophr. Res.* 125, 136–142
77. Fornito, A. *et al.* (2006) Morphology of the paracingulate sulcus and executive cognition in schizophrenia. *Schizophr. Res.* 88, 192–197
78. Garrison, J.R. *et al.* (2015) Paracingulate sulcus morphology is associated with hallucinations in the human brain. *Nat. Commun.* 6, 8956
79. Johns, L.C. *et al.* (2004) Prevalence and correlates of self-reported psychotic symptoms in the British population. *Br. J. Psychiatry* 185, 298–305
80. Daalman, K. *et al.* (2011) The same or different? A phenomenological comparison of auditory verbal hallucinations in healthy and psychotic individuals. *J. Clin. Psychiatry* 72, 320–325
81. Johns, L.C. *et al.* (2014) Auditory verbal hallucinations in persons with and without a need for care. *Schizophr. Bull.* 40, S255–S264
82. Woods, A. *et al.* (2015) Experiences of hearing voices: analysis of a novel phenomenological survey. *Lancet Psychiatry* 2, 323–331
83. Larøi, F. *et al.* (2004) The effects of emotional salience, cognitive effort and meta-cognitive beliefs on a reality monitoring task in hallucination-prone subjects. *Br. J. Clin. Psychol.* 43, 221–233
84. McKague, M. *et al.* (2012) Source monitoring and proneness to auditory-verbal hallucinations: a signal detection analysis. *Cognit. Neuropsychiatry* 17, 544–562
85. Garrison, J.R. *et al.* (2016) Testing continuum models of psychosis: no reduction in source monitoring ability in healthy individuals prone to auditory hallucinations. *Cortex* Published online November 22, 2016. <http://dx.doi.org/10.1016/j.cortex.2016.11.011>
86. Allen, P. *et al.* (2012) Neuroimaging auditory hallucinations in schizophrenia: from neuroanatomy to neurochemistry and beyond. *Schizophr. Bull.* 38, 695–703
87. Sugimori, E. *et al.* (2014) Brain mechanisms underlying reality monitoring for heard and imagined words. *Psychol. Sci.* 25, 403–413
88. de Weijer, A.D. *et al.* (2013) Aberrations in the arcuate fasciculus are associated with auditory verbal hallucinations in psychotic and in non-psychotic individuals. *Hum. Brain Mapp.* 34, 626–634
89. McCarthy-Jones, S. (2015) Reduced integrity of the left arcuate fasciculus is specifically associated with auditory verbal hallucinations in schizophrenia. *Schizophr. Res.* 162, 1–6
90. Thewissen, V. *et al.* (2005) Hearing impairment and psychosis revisited. *Schizophr. Res.* 76, 99–103
91. Kölmel, H.W. (1985) Complex visual hallucinations in the hemianopic field. *J. Neurol. Neurosurg. Psychiatry* 48, 29–38
92. Allen, P. *et al.* (2008) The hallucinating brain: a review of structural and functional neuroimaging studies of hallucinations. *Neurosci. Biobehav. Rev.* 32, 175–191
93. Hunter, M.D. *et al.* (2006) Neural activity in speech-sensitive auditory cortex during silence. *Proc. Natl. Acad. Sci. U. S. A.* 103, 189–194
94. Jones, S.R. and Fernyhough, C. (2009) Rumination, reflection, intrusive thoughts, and hallucination-proneness: towards a new model. *Behav. Res. Ther.* 47, 54–59
95. Henkel, L.A. *et al.* (1998) Aging and source monitoring: cognitive processes and neuropsychological correlates. *J. Exp. Psychol. Gen.* 127, 251–268
96. Cooper, R.A. *et al.* (2016) Reality monitoring and metamemory in adults with autism spectrum conditions. *J. Autism Dev. Disord.* 46, 2186–2198
97. Fletcher, P.C. and Frith, C.D. (2008) Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. *Nat. Rev. Neurosci.* 10, 48–58
98. Mammarella, N. *et al.* (2016) Self-generation and positivity effects following transcranial random noise stimulation in medial prefrontal cortex: a reality monitoring task in older adults. *Cortex* Published online November 15, 2016. <http://dx.doi.org/10.1016/j.cortex.2016.11.005>
99. Anselmetti, S. *et al.* (2007) Psychopathological and neuropsychological correlates of source monitoring impairment in schizophrenia. *Psychiatry Res.* 150, 51–59
100. Brunelin, J. *et al.* (2008) Selective source monitoring impairment in patients with schizophrenia compared to healthy and major depressive disorder subjects. *Eur. Rev. Appl. Psychol.* 58, 105–110
101. Fisher, M. *et al.* (2008) Self and other in schizophrenia: a cognitive neuroscience perspective. *Am. J. Psychiatry* 165, 1465–1472
102. Franck, N. *et al.* (2000) Confusion between silent and overt reading in schizophrenia. *Schizophr. Res.* 41, 357–364
103. Harvey, P.D. *et al.* (1988) Cognitive deficits and thought disorder: a retest study. *Schizophr. Bull.* 14, 57–66

104. Keefe, R.S.E. *et al.* (2002) Source-monitoring deficits for self-generated stimuli in schizophrenia: multinomial modeling of data from three sources. *Schizophr. Res.* 57, 51–67
105. Moritz, S. *et al.* (2003) Source monitoring and memory confidence in schizophrenia. *Psychol. Med.* 33, 131–139
106. Moritz, S. *et al.* (2005) Confidence in errors as a possible basis for delusions in schizophrenia. *J. Nerv. Ment. Dis.* 193, 9–16
107. Nienow, T.M. and Docherty, N.M. (2004) Internal source monitoring and thought disorder in schizophrenia. *J. Nerv. Ment. Dis.* 192, 696–700
108. Szöke, A. *et al.* (2009) Correlations between cognitive performances and psychotic or schizotypal dimensions. *Eur. Psychiatry* 24, 244–250
109. Waters, F.A.V. *et al.* (2006) The 'who' and 'when' of context memory: different patterns of association with auditory hallucinations. *Schizophr. Res.* 82, 271–273
110. Brunelin, J. *et al.* (2006) Source monitoring deficits in hallucinating compared to non-hallucinating patients with schizophrenia. *Eur. Psychiatry* 21, 259–261
111. Waters, F.A.V. *et al.* (2004) Context memory and binding in schizophrenia. *Schizophr. Res.* 68, 119–125
112. Garrison, J.R. *et al.* (2017) Reality monitoring impairment in schizophrenia reflects specific prefrontal cortex dysfunction. *Neuroimage Clin.* 14, 260–268