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INVITED REVIEW

The cognitive neuropsychiatry of delusions: from psychopathology to neuropsychology and back again

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ABSTRACT

Background. The nature of delusions remains unclear despite their interest to researchers and importance in psychopathology. Here we present a review of the contributions from various disciplines, principally cognitive neuroscience, towards a new understanding.

Method. Narrative review of published research.

Results. The main areas of activity revolve around reasoning biases, attributional and attentional biases, theory of mind, and the role of emotion, with each area beginning to be explored using functional neuroimaging techniques. Of heuristic interest are neurological models, which include confabulation and delusional misidentification and the one- *versus* two-stage (perceptual *versus* reasoning plus perceptual) accounts of the latter.

Conclusions. These different approaches are shown to each highlight mechanisms which are suggested to cause, contribute to, or modulate the genesis and form of delusions. Such contributions coupled with traditional phenomenological methods should provide the foundations for a cognitive neuropsychiatry of delusions.

INTRODUCTION

Historically, delusions have been thought of as the *sine qua non* of insanity. In spite of this and recognition of the prominence of delusions within psychopathology, there is no clear understanding of their aetiology. Here we present a digest of recent activity from the cognitive neurosciences as applied to delusions (see Table 1).

THE NATURE OF DELUSIONS

Delusions are a core part of a diagnosis of schizophrenia, with an estimated 60–70% of those diagnosed presenting with delusions of reference or persecution, and approximately 30% with religious delusions or delusions of grandiosity (Wing *et al*. 1974). The first barrier to scientific progress on this topic is the problem of definition. Delusions are generally accepted to be beliefs which (*a*) are held with great conviction; (*b*) defy rational counter-argument; (*c*) and would be dismissed as false or bizarre by members of the same socio-cultural group. A more precise definition is probably impossible since delusions are contextually dependent, multiply determined (David, 1999) and multidimensional (Garety & Hemsley, 1994). Exemplars of the delusion category that fulfil all the usual definitional attributes are easy to find so it would be premature to abandon the construct entirely. Equally, in everyday practice there are patients we regard as deluded whose beliefs in isolation may not meet standard

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Table 1. *Summary of key factors recently explored in the formation and maintenance of delusions*

Domain	Observation/Research area
Reasoning	Jumping-to-conclusions/Data-gathering bias; mild syllogistic reasoning bias
Attribution	Attributional (externalizing) biases for negative events. Attributional 'profiles'
Attention	Selective attention for self-relevant/threatening information, rCBF differences
Theory of Mind	Mentalizing impairments. Neural correlates, e.g. amygdala
Emotion	Dysfunctional emotional mediation of memory/imagined events. Imagined events sensorily richer than true memories. Biased recall. Limbic circuit mediation
Confabulation	'New form' of confabulation in schizophrenics
Neurobiology	State of 'abberant salience' through dysfunctional dopaminergic circuits

delusion criteria. In this way a delusion is more like a syndrome than a symptom.

While most psychologists start with the premise that, delusions are a species of belief, even this has been challenged. Berrios (1991), for example, suggested they might be 'empty speech acts'. More recently the philosopher Gregory Currie (2000) has proposed that they are 'imaginings' which the individual mistakes for beliefs. This is advanced to account for among other reasons, the supposed fact that delusions are rarely acted upon, that they may be incompatible with the person's own prior beliefs, and the difficulty in accepting that they are reasonable interpretations of abnormal perceptions (see below). In fact, although some may not, many patients do indeed act on their delusions (Buchanan & Wessely, 2004), sometimes catastrophically (Krasucki *et al.* 1995), and at times in the form of 'safety behaviours' (Freeman & Garety, 2001) whose connection with the motivating belief may not be obvious.

A cognitive neuropsychiatry of delusions

Cognitive Neuropsychiatry (David, 1993; Halligan & David, 2001) is the explanation of psychiatric phenomena by the methods of cognitive neuropsychology. With delusions, the task is to construct theories of the normal stages involved in belief-formation, and then show how malfunctions produce characteristic psychopathology (Gerrans, 2003*a*, *b*). One set of abnormal beliefs – delusional misidentification – has attracted much 'cross-over' attention in neuropsychology and philosophy. Such beliefs have a strong association with acquired brain dysfunction (Ellis, 1998) so are a 'good bet' in terms of their conforming to extant neuropsychological models of, say, face recognition. Similarly, delusions in Alzhemer's disease may reward more detailed study in relating pathophysiology to psychopathology (Sweet *et al.* 2003; Ballard *et al.* 2004; Shanks & Venneri, 2004).

Fleminger & Burns (1993) in a meta-analysis found a distinction between delusional misidentification of *place* – more common in cases of organic brain disorder – and misidentification of *person* – more common with functional mental disorder. Similar to Benson *et al.* (1976), occipital-parietal damage was highly asymmetric in favour of the right whereas there was no significant asymmetry in fronto-temporal damage. Ramachandran & Blakeslee (1998) have proposed that the left hemisphere assimilates new information into our pre-existing schemas for making sense of the world. The right hemisphere, in particular the right parietal lobe, contrastingly acts as a 'discrepancy detector' which prevents the left hemisphere processes from diverging too far from reality. McKay *et al.* (in press) suggests that such a mechanism leads to a balance or imbalance between *doxastic conservatism* (Langdon & Coltheart, 2000*a*) whereby the individual's existing web of beliefs are maintained, and *observational adequacy*, where first-person experience is accepted as veridical and displaces the contradictory beliefs about the world.

One- *versus* two-stage debate

Experimental studies in delusional misidentification patients have produced a replicable attenuation of the expected physiological and hence affective response to familiar stimuli (Tranel *et al.* 1995) which, authors argue, may lead to an inference that the individual has been replaced (by a 'double') or altered in some possibly bizarre way. Maher (1988) contexualized delusions as normal reasoning subserved by

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aberrant perceptions. Such a 'one-stage' concept is acceptable insofar as delusions are not necessarily associated directly with cognitive deficits (Mortimer *et al*. 1996; cf. thought disorder or negative symptoms; Langdon & Coltheart, 2000*b*). Maher posits that the aberration in perception is sufficient to cause the delusion. However, this cannot explain how such an inference is adopted and maintained as a belief despite its implausibility and despite convincing contradictory evidence, suggesting an extra process or 'stage' is required. Stone & Young (1997) suggest a parsimonious account for the production of a *monothematic* (*versus* polythematic), *circumscribed* (*versus* elaborated) delusion based on a two-stage account (Ellis & Lewis, 2001; see below).

Much has been written about the Capgras delusion, out of proportion to its prevalence or position within psychopathology. Furthermore, while the efforts of cognitive scientists and philosophers have given the field a much-needed boost, the results – a one- *versus* two-stage model – is a modest advance. Such models are in many ways the default of all psychiatric theories since there is no one-to-one correspondence between any aetiology, pathophysiology and clinical disorder in psychiatry, the diathesis-stress account being prototypical. Further, the traditional classification of delusions since Jaspers and following Schneider have included primary and secondary delusions.

REASONING

It has been regularly shown that delusional patients produce a 'jumping-to-conclusions' reasoning style (e.g. Garety & Hemsley, 1994). This is neither a function of impulsive decision making nor a consequence of memory deficit (Dudley *et al*. 1997; Garety & Freeman, 1999). Delusional patients, as well as exhibiting the behaviour noted above, also show a propensity to change their minds very easily with contradictory evidence, behaviour at odds with the unwillingness of patients to allow their beliefs to be disconfirmed in face of contradictory evidence. Garety & Freeman (1999) use the term 'data gathering bias' as the best summary of the data on probabilistic reasoning; others suggest an abnormality in the way hypothesis-generating evidence is treated (Langdon & Coltheart, 2000b). Indeed it has been suggested that the characteristic of delusional reasoning is the unwillingness to admit to any circumstance, which would provide a refutation of the belief (Leeser & O'Donohue, 1999).

In a different reasoning paradigm, Kemp *et al*. (1997) instructed deluded patients to choose between logically fallacious and valid responses on tests requiring judgements of conditionality or syllogistic reasoning. They concluded that controls as well as deluded patients made logical errors, tending to be guided by prior beliefs at the expense of logic, but that there were only minor differences between the groups. The difference was that deluded patients (who had a range neurocognitive deficits ranging from negligible to marked) were slightly more prone to endorse fallacious responses, especially when emotive themes are involved, a finding with a rather small effect size in comparison with the bizarreness of the delusions in question. The authors also highlight that deluded patients are not deluded 'about everything' and perhaps a global deficit in reasoning just is not there to be discovered (see also Young, 2000).

Goel *et al*. (2002) report somewhat contradictory preliminary data which suggest that healthy controls are most accurate on a syllogistic reasoning task when they can mobilize their pre-existing beliefs but not when the beliefs are, on the one hand irrelevant or, on the other hand particularly salient or emotive. By contrast people with schizophrenia failed to 'mobilize the belief-laden reasoning mechanism'. However, since the patients were significantly less accurate compared to controls overall, the statistical interaction noted could equally be due to the *excessive* mobilization of the salient *and* irrelevant beliefs. The interest in this work stems from the cerebral networks that Goel & Dolan (2001) have mapped out in relation to reasoning and belief: activation of the right lateral prefrontal cortex occurs when subjects inhibit responses associated with belief in order to reach the correct solution to a logical reasoning task while there is engagement of ventral medial prefrontal cortex when reason is 'over-ruled' by beliefs (see also Sanfey *et al*. 2003). One might hypothesize an imbalance in these systems in belief formation. We will return to the role of affect in biasing the systems towards belief.

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ATTRIBUTIONAL AND ATTENTIONAL BIAS

Taken together, the findings reviewed above show only subtle differences in reasoning abilities in deluded patients. Kinderman & Bentall (1997) and Bentall (2003), amongst others, postulate instead an extreme form of self-serving attributional style to explain the formation of delusional beliefs, at least when the delusional network has a persecutory nature, without any co-occurring perceptual or experiential anomaly, where a two-stage theory is redundant. Patients with persecutory delusions display an *externality* bias in their causal attributions for bad events, and whilst indeed presenting with different overall attributional 'profiles', non-persecutory patients did not differ from controls on this dimension (Sharp *et al*. 1997). Freeman & Garety (2004) and Beck & Proctor (2002) refine the notion by suggesting a *personalizing* bias: a tendency to blame other people when things go wrong.

Attentional bias has been consistently demonstrated in people with paranoid delusions (Fear *et al*. 1996), and selective attention to self-referential or threatening information may lead an individual to form conclusions about their environment which seem delusional to others (Ullman & Krasner, 1969). Bentall & Kaney (1989) have suggested that biases exist where patients selectively attend to threat-related stimuli and preferentially recall threatening episodes (Kaney *et al*. 1992). The preferential encoding and recall of delusion-sensitive material can be assumed to continually reinforce and propagate the delusional belief (Blackwood *et al*. 2001). Using psychophysiological methods, patients when currently experiencing persecutory delusions have been shown to quickly identify the threatening elements in 'ambiguous' pictures but, rather counter-intuitively, they actually spent less time than controls (without persecutory delusions) reappraising the threatening elements (Phillips *et al*. 2000). Other data suggested that patients may instead search for threatening stimuli in non-threatening areas of the images, i.e. they anticipated threat in inappropriate places.

Blackwood *et al*. (2000) used fMRI to investigate the brain regions which may support the attentional and attributional components of social cognition. Attention to threatening stimuli deemed relevant to 'self' differentially activated a more dorsal region of the left inferior frontal gyrus, which could represent selective biasing or gating of behaviourally relevant information from semantic memory representations, or the mediation of behaviour withdrawal elicited by threatening stimuli. Hypoactivation in these regions, it was hypothesized, would characterize the deluded state, reflecting excessive attention to self-referential information. Hypoactivation of the left precentral gyrus in the symptomatically deluded state could result in the accentuation (failure in inhibition) of the 'self-serving' attributional bias.

More recent work (Blackwood *et al*. 2004) using fMRI has revealed that when deluded patients evaluate potentially negative personal statements they activate less rostral-ventral anterior cingulate cortex – a region noted for self-monitoring – yet show greater increased posterior cingulate gyrus activation in comparison to the normal controls. The authors suggest this underlies impaired self-reflection in the persecutory deluded state.

THEORY OF MIND

Patients with persecutory delusions tend to be impaired on second order 'theory of mind' (ToM) tests where questions are posed to assess the understanding of the mental states of characters within a story (e.g. Frith & Corcoran, 1996; see Frith, 2004 and Lee *et al*. 2004 for reviews). Importantly, patients with passivity features and those in symptomatic remission performed such tasks as well as normal controls, indicating that any ToM deficit is a state rather than a trait variable (Pickup & Frith, 2001). However, good ToM performance may be seen in the face of delusions (Walston *et al*. 2000), and ToM problems have not been found in conversational interactions (McCabe *et al*. 2004) in schizophrenia.

Nevertheless, the advantage for our purposes of work utilizing the ToM concept in schizophrenia or more specifically, delusions (Frith & Corcoran, 1996) is that there is an emerging consensus as to the neural networks involved. Medial frontal systems come up time and again (Frith & Frith, 1999; Lee *et al*. 2004). One study which examined this in people with

schizophrenia (Russell *et al*. 2000) found reduced left medial prefrontal activation with fMRI – however, that experiment may have been confounded by the linguistic demands of processing complex mental state terms in the experimental condition. Finally Blakemore *et al*. (2003) showed that the normal tendency to distinguish contingent from non-contingent movement between two cartoon shapes was lost in schizophrenia and affective disorder patients with persecutory delusions in comparison to those without delusions.

Another part of the 'social brain' circuitry that may be relevant is the amygdala (Fine *et al*. 2001). One patient who acquired bilateral focal damage during childhood has been described who is poor at making important social attributions to others from their appearances (Adolphs *et al*. 1998) and fails to attribute social intent in the normative manner to moving geometrical objects – the mirror image of the ToM disturbance claimed for people with delusions (Heberlein *et al*. 1998). There is consistent evidence for the activation of the amygdala during the 'on-line' adult perception of basic and complex emotional states and for the deleterious effects of early amygdala lesions on emotional perception [for reviews of fMRI studies see Zald (2003) and lesion studies see Adolphs (1999)]. Recent work by Shaw *et al*. (2004) suggests that lesions to the amygdala interfere with ToM reasoning most consistently if acquired relatively early in life – a situation consistent with developmental theories of schizophrenia.

ROLE OF EMOTION IN DELUSIONS

As noted, emotion may 'derail' rationality. The criteria of what qualifies as a truth statement may be relaxed when the topic is related to a deluded person's preoccupations (Rossell *et al*. 1998). Delusions driven by underlying affect (mood-congruent) and those that have no connection to the underlying mood (mood-incongruent), may differ neuro-cognitively. Gibbs & David (2003) propose a significant role of emotion in the aetiology and maintenance of mood-congruent delusions via its effect on memory. Superior recollection of autobiographical episodic memories congruent with present mood, in conjunction with memories (and other mental contents) formed during emotional arousal being resistant to forgetting (e.g. Hamann *et al*. 1999) could reasonably result in persistence of biased recall of mood-congruent memories and beliefs.

Identification of emotionally salient stimuli of relevance to persecutory beliefs has been shown to be associated with amygdala and anterior insula activation, for threat and aversive response (Calder *et al*. 2001) respectively (see also Phillips *et al*. 1998). Neuroimaging studies have demonstrated abnormal volumes in the amygdala, thalamus, as well as the hippocampus and insula in schizophrenic patients (McCarley *et al*. 1999; Wright *et al*. 2000). Inappropriate conjunction of affective tone, mediated by limbic structures, to memories of imagined events could impair reality monitoring and lead to delusions by adding misleading contextual information (Morrison & Haddock, 1997).

A dysfunctional emotional-tagging of imagined events could lead patients to the conclusion that such events actually took place, and the effective mood-congruence formed as a result would readily lend the memory to future recall. Treatment of a co-morbid mood disorder has been shown to reduce delusions (Seretti *et al*. 2000). David & Howard (1994) showed highly emotive delusional memories were rated as perceptually and sensorily richer (across several domains) than memories of *actual* events. Freeman & Garety (2003) also highlight key associations between emotion and the development of delusions which *directly* represent those emotions. By extending the notion of mood congruence beyond an affective valence dimension, anxiety may be related to 'anticipation of danger' leading to perception of threat, and hence persecutory delusions. Such notions have the advantage of encompassing animal models of psychopathology. The aetiology of mood-incongruent delusions is less clear.

A FORM OF SPONTANEOUS CONFABULATION?

Like delusions, spontaneous confabulations (SCs) hold the meaning of reality to patients and motivate them to act. According to Baddeley & Wilson (1986) SCs may be held with great conviction, resistant to persuasion, and preoccupy the patient. They are often bizarre and incoherent and hence indistinguishable from delusional memories and formal thought

disorder (Nathaniel-James & Frith, 1996) except by the clinical context in which they arise (Kopelman *et al*. 1995). SC is believed to reflect damage to the integrity of the frontal lobes in particular the orbitofrontal cortex (Moscovitch, 1989). Damage to a lateral limbic loop connecting the orbitofrontal cortex to the amygdala and dorsolateral nucleus of the thalamus precludes making the distinction between presently on-going and previous information leading to the temporal confusion observed (Schnider *et al*. 1996). Patients with SC are unable to suppress previously activated, yet currently irrelevant memory traces (Schnider & Ptak, 1999). Nathaniel-James & Frith (1996) have argued that schizophrenics present with a 'new form' of confabulation, which entails reorganizing or reconstructing existing material (see also Schnider, 2001).

While confabulation may be a useful linking phenomenon between delusions and the brain, Mattiolo *et al*. (1999) describe a patient who presented with both SC and delusional misidentification. They conclude that the two phenomena were so clearly different in their clinical course with distinct evolutions, that different mechanisms must be involved in their manifestation.

NEUROBIOLOGY

The dopamine hypothesis asserts a primary association of dopamine (DA) and the manifestation of psychotic symptoms. DA is strongly implicated in 'reward' pathways and thus, turns representations of external stimuli into attractive or aversive entities (Kapur, 2003); DA mediates the process of salience acquisition and expression. If a state of dysregulation of DA transmission ensues, then stimulus-independent release of DA could result, leading to 'aberrant assignment of salience to external objects and internal representations' (Kapur, 2003), reminiscent of delusional mood. A delusional network derives from a 'top-down' cognitive explanation of the aberrant salience, and therefore, can explain individual variation from the same neurobiological dysfunction, although other neurotransmitters could be involved.

Antipsychotic medication leads to an attenuation of the salience wrongly attributed to both environmental cues and the individual's own ideas. Clearly, the neurobiology only presents a milieu through which psychological processes operate to change belief structures. The fact that people do recover from delusions may be taken as evidence in and of itself of an abnormal belief construction process (rather than a fixed deficit) and should be incorporated into cognitive neuropsychiatric theories (see Stanton & David, 2000).

Finally, support for a neurobiological contribution to the development of delusions has recently been reported. Zwanzger *et al*. (2002) attempted to treat depression in a patient with rTMS over the left dorsolateral prefrontal cortex. The patient developed severe persecutory psychotic symptoms for the first time following repeated sessions of rTMS, which disappeared between rTMS treatment sessions with medication. Interestingly, the domapinergic action of acute rTMS in healthy volunteers was recently demonstrated (Strafella *et al*. 2001). Future work may aim to see if alteration or amelioration of perceptual input in 'perceptual' delusions can attenuate the delusional belief. Recent work by Hoffman *et al*. (2003) shows that slow (1 Hz) TMS stimulation of the inferior temporal lobe, adjacent to the primary auditory cortex can profoundly reduce auditory hallucinations. If a two-stage model of delusions is correct then perhaps such a process may lead patients to re-evaluate their delusional networks in favour of more 'normal' explanations.

CONCLUSIONS

A cognitive neuropsychiatry of delusions is in its infancy. So far studies tend to be small scale and replications are few. Two approaches appear to be of heuristic value. First, linking disorders of belief to related cognitive deficits – such as dysfunctional memory and recognition, and then linking these to neural systems; and second exploring delusions or delusion-like phenomena in people with known brain disease and using this to infer dysfunction in people with 'idiopathic' disorders. In both there is a sequence from the phenomenology to neuropsychology and neurophysiology and then back, hopefully, to a more scientifically informed understanding of psychopathology.

DECLARATION OF INTEREST

None.

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