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Cognitive functioning in delusions: A longitudinal analysis

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Abstract

Background: this study explored the longitudinal course of the relationship between delusions and different aspects of cognitive functioning.

Methods: deluded patients were compared to psychiatric and non-clinical controls on three tasks: negative priming, a probabilistic judgement task (the 'beads' task), and the pragmatic inference task (PIT). All groups were tested at two time points, once when actively symptomatic, and once when in remission.

Results: deluded individuals exhibited a 'jump-to-conclusions' (JTC) reasoning bias: i.e., they made decisions on the basis of limited evidence and were more likely to revise their estimates when faced with disconfirmatory evidence. This JTC bias remained stable over time, although probability judgments seemed to normalise in remission. No deficits in cognitive inhibition were found on negative priming. The deluded group displayed an excessive self-focus on the PIT at both time points, but did not show a depressive attributional style. Only a small sub-sample, characterised by the "bad-me" type of paranoia [Trower & Chadwick, 1995 Clinical Psychology: Science and Practice, 2, 263–278.], demonstrated depressive schemas when symptomatic, but no longer did so when remitted. Few relationships were found between tasks, suggesting that different areas of functioning are relatively independent. The only measures associated with delusion symptom scores were from the 'beads' task.

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Conclusions: overall these findings suggest that the JTC bias is a stable factor associated with delusional thinking, while the depressive attributional style characteristic of a small sub-sample of paranoid patients fluctuates with delusional course.

Keywords: Delusions; Reasoning; Attributional style; Cognitive inhibition; Paranoia

Introduction

The recent impetus to study symptoms rather than heterogeneous diagnostic categories such as schizophrenia (Bentall, Jackson, & Pilgrim, 1988) has resulted in a plethora of studies looking at delusions from a psychological perspective. Maher (1992) has proposed that delusions arise from the application of normal reasoning processes to abnormal experiences, i.e., delusions are perception-driven. In contrast, Garety and Hemsley (1994) have suggested that delusions are more than statements of experience, and involve an abnormal evaluative judgment arising from reasoning biases.

The ''delusions as explanations of experience'' theory is supported from a number of sources. First, there is a large body of evidence documenting the disruption of information processing in psychotic individuals, leading to a variety of perceptual disturbances (Hemsley, 1993). Studies using negative priming (Tipper, 1985), specifically, have provided fairly robust evidence for Frith (1979)'s suggestion of deficient 'cognitive inhibition' in schizophrenia. The 'negative priming effect' refers to the observation that normal individuals show an increase in reaction time (RT) when asked to name a target which has previously been ignored as a distractor (Tipper, 1985). The negative priming effect has been proposed to be a direct measure of cognitive inhibition[1] (Tipper, Weaver, & Milliken, 1995). Overall there are now 12 studies showing reduced negative priming in psychotic samples on a variety of tasks (Beech, Powell, McWilliam, & Claridge, 1989; McDowd, Filion, Harris, & Braff, 1993; Salo, Robertson, & Nordahl, 1996; Salo, Robertson, & Nordahl, & Kraft, 1997; Laplante, Everett, & Thomas, 1992; David, 1995; Park, Lenzenweger, Puschel, & Holzman, 1996; Moritz et al., 2001; Hoenig, Hochrein, Muller, & Wagner, 2002; McQueen, Galway, Goldberg, & Tipper, 2003), and in positive symptoms specifically (Williams, 1996; Peters et al., 2000). Although a few recent studies have failed to replicate such findings (Moritz, Jacobsen, Mersmann, Kloss, & Andresen, 2000; Baving, Wagner, Cohen, & Rockstroh, 2001; Hoenig et al., 2002; Roesch-Ely, Spitzer, & Weisbrod, 2003), it is likely that these results can all be explained by the critical factors of critical stimulus durations (CSDs) and inter-stimulus interval (ISIs).

Second, there is evidence that delusions occur in a large number of medical and psychological conditions (Maher & Ross, 1984), and that irrational beliefs can be induced in the general

[1]There are alternative accounts of negative priming which do not invoke inhibition: the feature mismatching account (Park & Kanwisher, 1994); the episodic retrieval account (Neill, Valdes, Terry, & Gorfein, 1992); and the temporal discrimination framework account (Milliken, Joordens, Merikle, & Seiffert, 1998). However a discussion of the evidence for and against each model, and their relationships to the cognitive deficit in schizophrenia, is beyond the scope of this paper.

population under anomalous environmental conditions (Zimbardo, Andersen, & Kabat, 1981). Maher (1992)'s theory also makes intuitive clinical sense, as for the individual who comes to the unsurprising conclusion that she is being spied upon by aliens, because the voices she hears declare themselves to be aliens employed by the CIA to spy on her. However, why not arrive at the potentially equally plausible explanation that the voices are a manifestation of a mental disturbance that distorts conscious experience (Freeman et al., 2004)? Indeed, aberrant perceptions do not always lead to delusions, nor are all delusions based on perceptual disturbances (Chapman & Chapman, 1988). Furthermore, perceptions do not exist independently of their interpretations, since cognitive templates actively influence the perceptual search (so-called "Gestalt" or "top-down" processing, Norman & Bobrow, 1976), and as such delusions may at least sometimes shape abnormal experiences rather than the other way round (Slade & Bentall, 1988).

There is now a growing body of evidence demonstrating reasoning and attributional biases in people with delusions (Garety & Freeman, 1999), which challenges Maher's position. Garety and her colleagues (Huq, Garety, & Hemsley, 1988; Garety, Hemsley, & Wessely, 1991) originally demonstrated that deluded people have a "jump-to-conclusions" (JTC) reasoning style on a probabilistic reasoning task (the 'beads' task): they require less information before making a decision, and are more likely to revise their hypothesis in the light of disconfirmatory evidence. These results suggest that limited amounts of information represent sufficient evidence for a hypothesis to be accepted, thereby increasing the likelihood of inaccurate beliefs being formed hastily (Garety, 1991). The greater likelihood of deluded patients to revise their hypotheses is particularly interesting, since it shows that, contrary to popular misconception, they are not characteristically incorrigible if faced with neutral material. Rather, the certainty and incorrigibility traditionally ascribed to delusional beliefs are in fact the normal characteristics of any challenged, strongly held belief, such as religious or scientific beliefs (Alloy & Tabachnik, 1984; Maher, 1992).

An impressive number of studies have since found a JTC bias, using various modifications of the basic paradigm, in deluded and delusion-prone individuals (see Garety & Freeman, 1999, for a review). In the largest study to date, Garety and her colleagues (In Press) found that a JTC bias was present in approximately half of their sample of 100 deluded participants. Most experiments have compared deluded with non-deluded patients, irrespective of diagnosis. In the few studies which have looked specifically at the specificity of JTC to delusions over and above diagnosis, three studies found specific relationships with delusions, but not diagnosis (Garety et al., 1991; McGuire, Junginger, Adams, Burright, & Donovick, 2001; Moritz & Woodward, in press), while two other studies found a JTC bias in patients with schizophrenia, which was not related to the presence of delusions (Mortimer et al., 1996; Menon, Pomarol-Clotet, McKenna, & McCarthy, in press). However, at least two studies have demonstrated the presence of a JTC bias in patients diagnosed with delusional disorder rather than schizophrenia (Fear & Healy, 1997; Conway et al, 2002). In addition, a recent paper comparing psychotic individuals with and without current delusions demonstrated that the JTC bias was indeed specific to the presence of delusions, since diagnostic comparisons in the same sample did not show significant differences on the same tasks (Peters, Thornton, Siksou, Linney, & MacCabe, under review). Garety et al. (in press) further specified that JTC is related to delusional conviction specifically, but not to delusional distress, preoccupation, or disruption to life.

JTC is not a function of impulsiveness, since the psychotic groups adjust the amount of evidence required with a changed probability ratio (Dudley, John, Young, & Over, 1997a; Menon et al. in press; Garety et al., in press). It appears unrelated to a memory deficit (Dudley et al, 1997a; Garety et al., in press), although one study found normalised performance with the presence of a memory aid (Menon et al. in press). Error rates tend to be low, and deluded participants made significantly more errors in only two studies (Fear & Healy, 1997; Young & Bentall, 1997a). The JTC reasoning style appears to be a specific bias in data-gathering rather than a general deficit in reasoning (Bentall & Young, 1996; Young & Bentall, 1997a; Dudley et al., 1997a; Linney, Peters, & Ayton, 1998; Peters et al., under review), and is more pronounced for emotionally salient material (Dudley, John, Young, & Over, 1997b; Young & Bentall, 1997a; McGuire et al., 2001). The specificity of this bias, and the exacerbation of this bias with emotional material, answer two of the criticisms levelled at this body of work by Simpson, Done, & Vallee-Tourangeau (1998), namely that it ignores the fractionation of cognitive abilities and the influence of content on reasoning processes.

Another challenge to Maher's (1992) theory is the large body of work demonstrating attributional biases in delusions (Bentall, Kinderman, & Kaney, 1994; Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001). Kaney & Bentall (1989) originally found that patients with persecutory delusions made excessively external attributions for negative events, but excessively internal attributions for positive events, on the attributional style questionnaire (ASQ; Peterson et al., 1982). This pattern of responding is suggested to represent an exaggerated form of the 'self-serving bias' characteristic of the normal population as a means of maintaining self-esteem. The externalising bias for negative events in paranoid patients is a robust finding which has now been replicated in four other studies (Candido & Romney, 1990; Lyon, Kaney, & Bentall, 1994; Fear, Sharp, & Healy, 1996; Sharp, Fear & Healy, 1997), although the evidence for an internalising bias for positive events is weaker. Attributional differences between deluded and other groups are restricted to self-referent tasks (Young & Bentall, 1997b), and Kinderman and Bentall (1997) have further specified that deluded individuals have a *personalising* bias rather than a general externalising bias for negative events, i.e., they blame other people rather than the situation. Therefore, similarly to the data-gathering bias described above, the attributional bias is of a specific rather than a general nature.

Interestingly, an exaggerated self-serving bias has only been found with measures that assess attributions directly or explicitly. If the attribution task becomes opaque by being disguised as a memory task (such as the pragmatic inference task (PIT); Winters & Neal, 1985), thereby giving an *implicit* measure of attributional style, then some studies have reported that the same individuals who show a self-serving bias using the ASQ change to show a depressive attributional style, i.e., blaming themselves for negative events and attributing positive events to circumstances (Lyon et al., 1994; Krstev, Jackson, & Maude, 1999). This "flipping over" of attributional style depending on whether the measure was transparent or opaque was specific to the deluded individuals, with the attributional style of the depressed and non-clinical comparison groups remaining stable.

These results suggest a discrepancy between overt and covert self-representations in paranoid patients (Bentall et al., 1994). Studies exploring the relationship between self-esteem and persecutory delusions have shown contradictory findings (Freeman et al., 1998; Drake et al., 2004), suggesting the associations are complex and potentially cyclical or dynamic (Kinderman,

Prince, Waller, & Peters, 2003), and depend on the type and timing of measurement (Bentall et al., 2001), as well as the sub-samples of patients examined (Freeman et al., 1998). Trower and Chadwick (1995) have indeed postulated the existence of two forms of paranoia, one characterised by high self-esteem, externalisation of blame, and delusions of persecution (i.e., "poor-me" paranoia), and the other by low self-esteem, internalisation of blame, and delusions of deserved punishment (i.e., "bad-me" paranoia), although so far there have been few empirical studies examining this distinction.

To summarise, there is evidence for a reduction in cognitive inhibition in individuals with positive symptoms of psychosis, and for the presence of a specific data-gathering reasoning bias in deluded people. There is also some suggestion of a discrepancy between implicit and explicit attributional style, with deluded patients showing an implicit depressive attributional style. Garety, Kuipers, Fowler, Freeman, & Bebbington (2001) have recently proposed a cognitive model of the positive symptoms of psychosis which incorporates these three types of disruptions in cognitive functioning. However, there are two major limitations to this body of empirical research.

The first concerns the lack of systematic investigation of the presence of the three forms of pathology in the same patients. This omission is likely to be due to the different theoretical contexts in which each of the above three model has been couched: the negative priming data are part of the large "cognitive deficit" in schizophrenia literature pioneered by Hemsley (1977); the reasoning studies have their roots in the study of decision-making (Kahneman, Slovic, & Tversky, 1982); while the Bentall model originates from the social cognition literature, mostly investigated in depression (Seligman, 1975). However, it is important to explore the connections between the different areas of dysfunction, since they are unlikely to function as isolated factors but may reciprocally influence each other (Bentall, 1999).

The second is due to the research designs employed to date which rule out the possibility of inferring causal relationships between delusions and any of the above cognitive biases. Birchwood (1999) has pointed out that the longitudinal dimension is crucial in disentangling which processes are involved in the formation of delusions and which are merely implicated in their maintenance. Most studies have been carried out on people with active delusions, and it is not possible to determine whether the various biases found have led to the emergence of delusions, or whether delusions recruit those biases once they are activated, in a similar vein to some of the cognitive biases involved in depression (Teasdale & Barnard, 1993). It is therefore crucial to obtain longitudinal data tracing patients through their delusional fluctuations to investigate the formation versus maintenance issue.

This study attempted to address these limitations. First, each patient was tested on multiple tasks, permitting the exploration of potential relationships between information processing deficits, reasoning biases, and implicit attributional style biases, all previously found in isolation in deluded patients. All tasks were chosen specifically because abnormalities of functioning do not predict worse performance (i.e., reduced cognitive inhibition leads to faster RTs in the negative priming condition; a JTC bias is closer to normative Bayesian reasoning), thereby reducing the confounding effects of the generalised performance deficit commonly found in deluded persons. A follow-up was also included to allow the possibility of making tentative causal inferences, with patients being tested both when their delusions were active and after remission.

At baseline, based on the literature reviewed above, it was predicted that deluded individuals would (1) fail to show the usual negative priming effect on a negative priming task; (2) show a JTC

bias on the 'beads' task i.e., ask to see fewer beads before making a decision as to which jar of beads had been chosen, and be more likely to revise their hypothesis in the light of disconfirmatory evidence; and (3) demonstrate an implicit depressive attributional style on an opaque attributional style task such as the PIT. No a -priori hypotheses could be made regarding the potential relationships between negative priming, JTC bias, and attributional style, since this has not been examined in the literature before.

At follow-up, a reversal of the predicted lack of negative priming, the JTC bias, and the depressive attributional style, would suggest that these processes fluctuate with delusional course and may therefore occur as a consequence rather than a cause of delusions. In contrast, the stability of the above cognitive biases with symptomatic remission would be consistent with the conjecture that they may represent stable vulnerability factors potentially implicated in the formation, rather than merely the maintenance, of delusions. The JTC bias has been demonstrated both in healthy delusion-prone (Linney et al., 1998; Colbert & Peters, 2002), and at-risk (Broome et al., 2003) individuals, suggesting that it may be a stable factor. Similarly, a reduction in negative priming has previously been found in schizotypal individuals (Peters, Pickering, & Hemsley, 1994). However, Park, Puschel, Sauter, Rentsch, & Hell (2002) showed that negative priming on a spatial task, absent during the acute psychotic state, was restored at a four-months follow-up. Nevertheless, they also found that a lower negative priming score during the acute psychotic state was associated with increased positive symptoms at follow-up, suggesting that negative priming may be causally related to symptoms. Finally, while Kinderman and Bentall (2000) report some evidence for a link between paranoid ideation in healthy participants and a personalising bias, Martin and Penn (2001) did not find any significant associations between attributional bias and paranoid ideation in a non-clinical population. However, both these studies investigated explicit rather than implicit attributional style, as measured in the present study. Overall, we would therefore predict cautiously that performance on each of the tasks employed in this study would remain stable despite remission in symptoms.

Method

Design

This study involved the longitudinal comparison of three groups of participants on three tasks. Deluded individuals, psychiatric controls, and non-clinical controls were tested at two time points (for the psychiatric groups, once when actively symptomatic (baseline study) and once when in remission (follow-up study)) on negative priming, a probabilistic judgment task (the 'beads' task), and the Pragmatic Inference Task (PIT).

Tasks

Negative priming

The Stroop version of the negative priming task used by Peters et al. (2000) was administered. There were three conditions: the control condition (CC) consisted of a row of coloured Xs; in the Stroop interference condition (SC) the ignored prime (i.e., the colour *word*) was unrelated to the

subsequent target (i.e., the *ink* colour) to be named; in the negative priming condition (PC) the ignored prime *predicted* the following target.

Participants were informed that colour words and Xs would be displayed on the screen, and were instructed to name the ink colour after each exposure. One trial contained nine stimuli presentations, all of the same condition. Each stimulus presentation consisted of the following: a black fixation cross, displayed for 500 (msecs), followed by the stimulus. After 100 ms the pattern mask appeared and remained on display until the subject triggered the voice–key; the next fixation cross was then immediately presented. This was repeated until the participant had been exposed to nine items. At the end of each trial the participant's RT and the number of errors made were displayed on the screen. Three practice trials were followed by six trials in each condition, presented in a fixed random order. Only the RTs for items two to nine in each trial ($n = 48$ for each condition) were included in the analysis, since the first item was not primed. Peters et al. (2000) provide more task details.

'Beads' task

The task used by Garety et al. (1991) was employed. Four jars each containing 100 beads of two different colours were used, two jars in each condition. In the first condition the two jars contained black and yellow beads in equal and opposite proportions (85:15); in the second condition the jars contained green and red beads in the same proportions. In both conditions the two jars were hidden from view, and beads from the selected jar were shown to the participant one at a time in a predetermined order.

In the first condition the participant could see as many beads as they wanted before making their decision as to which jar had been chosen. In the second condition participants were shown 10 beads only. After each bead presentation participants were required to put a mark on an analogue scale as to how likely it was that Jar A had been chosen. Please refer to Garety et al. (1991) for more task details.

PIT

The same task as Lyon et al. (1994) was used. The PIT is presented as a memory test in order to avoid conscious response biasing, and consists of 12 short hypothetical vignettes (6 positive and 6 negative events). The vignettes are all self-referent and both an internal and external locus of causality is implied in each story. The vignettes were recorded on to audiotape for consistency of presentation. After each vignette the participants were required to answer four memory questions presented as multiple-choice items, with only the attributional inference being scored. The attributional item implicitly demands that participants recall what they perceive to be the main contributing factor in the described outcome by selecting one of the two causes (internal and external) given in the story. Please refer to Lyon et al. (1994) for more task details.

Other measures

Psychiatric symptoms in the two psychiatric groups were measured using the Manchester Scale (MS; Krawiecka, Goldberg, & Vaughan, 1977). The MS measures both the presence and severity of symptoms on a five-point scale. Eight symptoms are recorded, falling into three main categories: affective (depression and anxiety); positive (delusions, hallucinations, and incoherence

and irrelevance of speech); and negative (poverty of speech, flattened incongruous affect, and psychomotor retardation).

Delusions and delusional ideation were also measured using the 21-item Peters et al. Delusions Inventory (PDI; Peters, Joseph, Day, & Garety, 2004), and the Delusions Symptom-State Inventory (DSSI; Foulds & Bedford, 1975). Anxiety and depression were measured using the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983), which was originally designed for use in hospital patients. Verbal IQ was assessed using the Quick Test (QT; Ammons & Ammons, 1968).

Participants

Baseline study

Both psychiatric groups were selected from a number of inpatient wards at the Maudsley and the Bethlem Royal Hospitals, and were therefore acutely ill patients. Acute admission units were targeted for the selection of the deluded sample, while the Affective Disorders Unit was targeted for the selection of the psychiatric control group.

For the deluded group, individuals who scored two (i.e., ''moderate severity'') or more on the delusions item of the MS (Krawiecka et al., 1977) were selected, irrespective of diagnosis. The psychiatric control group consisted of inpatients exhibiting depressive and/or anxious symptomatology with no current or previous history of psychosis. Only individuals scoring two or above on either the anxiety or the depression item of the MS were selected, again irrespective of diagnosis. The psychiatrists in charge of their care, who were all experienced clinicians and interviewers, carried out ratings on the MS independently, within a week of the testing session. This information was confirmed by case-note review, carried out by the experimenter (E.P.). None of the patients in either psychiatric group had a history of neurological impairment or alcohol abuse. All participants had normal colour vision, and normal, or corrected-to-normal, acuity. All patients were on a combination of medications at the time of testing; those included neuroleptics, lithium, and antidepressants (only neuroleptics are given in Table 1).

The non-clinical control group was obtained from the Psychology Department Participants Pool. They had all indicated on the demographics form that they had no psychiatric history and were not taking any prescribed drugs at the time of testing. All participants had normal colour vision, and normal, or corrected-to-normal, acuity.

The deluded group consisted of 23 patients, the psychiatric control group comprised 22 patients, and 36 non-clinical individuals participated. There were equal ratios of males in females in the two control groups, but only 2 females were recruited in the deluded group, reflecting the preponderance of male admissions to acute psychiatric wards. Demographic information in the groups is displayed in Table 1.

As expected the deluded group had significantly higher positive symptom MS scores (Mann-Whitney tests: $U = 1.5$, $p < .001$), and significantly lower affective symptom MS scores ($U = 49.5$, $p < .001$) than the psychiatric controls, but did not differ in negative symptom MS scores ($U = 162$, $p > .1$). There was a trend for the deluded group to have spent more time in hospital ($U = 162$, $p = .06$), but no differences between the two psychiatric groups in age of onset of illness ($U = 169$, $p > .1$).

Table 1
Demographic information for the three groups at baseline and follow-up (means and sds (in parentheses))

	Deluded group		Psychiatric controls		Non-clinical controls	
	Baseline ($n = 23$)[a]	Follow-up ($n = 17$)[b]	Baseline ($n = 22$)[c]	Follow-up ($n = 18$)[d]	Baseline ($n = 36$)	Follow-up ($n = 20$)
Age	30.7 (7.2)	29.9 (7.8)	40.9 (13.6)	40.1 (12.9)	27.7 (6.7)	27.9 (6.4)
Verbal IQ	88.1 (13)	87.8 (14.2)	97.4 (13.8)	99.8 (10.2)	107.4 (11.4)	108.6 (10.3)
Gender	21 males 2 females	17 males 0 females	11 males 11 females	8 males 10 females	18 males 18 females	12 males 8 females
PDI (total)	145.9 (69.2)	96.7 (70.3)	87.1 (55.2)	64.3 (57.3)	54.6 (42.4)	43.6 (42.6)
DSSI	15.9 (5.5)	8.1 (6.6)	4.8 (4.5)	4.5 (5.5)	2.3 (4.9)	2.9 (5.3)
HADS (anxiety)	7.6 (2.9)	6.9 (3.9)	13.2 (4.9)	9.8 (5.5)	6.1 (3.7)	5.3 (2.9)
HADS (depression)	6.1 (4.5)	6.7 (4.9)	12.5 (4.8)	7.2 (5.8)	2.9 (2.3)	3.0 (2.2)
Age at onset of illness	25.6 (7.0)	24.8 (7.1)	31.2 (14.1)	32.8 (14.9)	—	—
Time in hospital (weeks)	20.2 (17.6)	17.5 (15.9)	13.4 (18.7)	12.0 (19.5)	—	—
MS affective	1.9 (1.5)	1.5 (1.3)	4.6 (1.7)	1.0 (1.2)	—	—
MS positive	6.1 (2.1)	1.4 (1.7)	0.3 (0.8)	0.0 (0.0)	—	—
MS negative	1.3 (2.0)	0.9 (1.6)	0.7 (1.7)	1.8 (3.2)	—	—
Diagnoses (DSM-IV)	18 Schizophrenia 5 Bipolar/ Schizoaffective	13 Schizophrenia 4 Bipolar/ Schizoaffective	16 Depression 3 anxiety & depression 3 SAD	12 Depression 3 anxiety and depression 3 SAD	—	—
Neuroleptics (chlorpromazine equivalents, in mg)	612.4 (440.7)		—	—	—	—

Legend: Verbal IQ: from Quick Test (Ammons & Ammons, 1968). PDI total: total of Yes/No scores + Distress + Preoccupation + Conviction scales of 21-item Peters et al. Delusions Inventory (Peters et al., 2004). DSSI: Delusions Symptom-State Inventory (Foulds & Bedford, 1975). HADS: Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983). MS affective: Manchester Scale (Krawiecka et al., 1977), depression + anxiety. MS positive: Manchester Scale, delusions + hallucinations + incoherence of speech. MS negative: Manchester Scale, poverty of speech + flattened incongruous affect + psychomotor retardation. SAD: Seasonal Affective Disorder. DSM-IV (APA, 1994).

[a]Three Quick Tests, 2 HADS, 2 DSSI, and 7 PDIs were incomplete or missing for the deluded group at baseline.

[b]Two Manchester Scales, 1 Quick Test and 1 DSSI were incomplete or missing for the deluded group at follow-up.

[c]4 Manchester Scales were incomplete or missing for the psychiatric controls at baseline.

[d]8 Manchester Scales were incomplete or missing for the psychiatric controls at follow-up.

As expected, there were significant differences between the three groups on the Peters et al. Delusions Inventory (PDI; Peters et al., 2004) ($F = 15.8$, d.f. $= 2, 68$, $p < .001$), the delusions symptom-state inventory (DSSI; Foulds & Bedford, 1975) ($F = 53.2$, d.f. $= 2, 76$, $p < .001$), and the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) anxiety ($F = 23.7$, d.f. $= 2, 76$, $p < .001$) and depression ($F = 44.1$, d.f. $= 2, 76$, $p < .001$) scores. The deluded group scored significantly higher on the PDI and the DSSI than both the psychiatric (PDI:

Scheffe = 58.7, $p<.01$; DSSI: Scheffe = 11.1, $p<.001$) and the non-clinical (PDI: Scheffe = 91.3, $p<.001$; DSSI: Scheffe = 13.7, $p<.001$) control groups. The psychiatric control group scored significantly higher on anxiety and depression than both the deluded (anxiety: Scheffe = 5.6, $p<.001$; depression: Scheffe = 6.5, $p<.001$) and the non-clinical controls (anxiety: Scheffe = 7.0, $p<.001$; depression: Scheffe = 9.6, $p<.001$). The deluded group also scored significantly higher than the non-clinical controls on depression (Scheffe = 3.1, $p = .01$).

One-way ANOVAs revealed significant differences between the three groups in age ($F = 14.5$, d.f. = 2, 78, $p<.001$) and verbal IQ ($F = 15.8$, d.f. = 2, 75, $p<.001$), with the psychiatric control group being significantly older than both the deluded (Scheffe = 10.2, $p<.01$) and the non-clinical control (Scheffe = 13.2, $p<.001$) groups, and the non-clinical controls having significantly higher IQ scores than both the deluded (Scheffe = 19.3, $p<.001$) and the psychiatric control (Scheffe = 10.0, $p<.05$) groups.

Follow-up study

All patients were contacted again when they were in remission, usually at or following discharge. Demographic information is displayed in Table 1. For the deluded group only individuals who scored less than two on the delusions item of the MS were considered to be in remission.[2] Six patients in the deluded group were either lost to follow-up or were still deluded, leaving 17 patients. The mean on the delusion item of the MS changed from 3.4 (sd = .73) at baseline to 0.6 (sd = .51) at follow-up, with all patients showing a reduction of 2 or more points (Wilcoxon test: $Z = 3.5$, $p<.001$). As expected, the deluded group scored significantly lower on the overall positive symptom MS scores at follow-up compared to baseline (Wilcoxon test: $Z = 3.3$, $p = .001$) but not on the negative ($Z = 0.9$, $p>.1$) or the affective ($Z = 1.3$, $p>.1$) symptom MS scores.

Four patients were lost from the psychiatric control group, leaving 18 patients. All patients scored less than two on both the anxiety and depression items of the MS[2], apart from 1 patient who still scored 2 on the depression item at follow-up but was still included because his predominant symptom, anxiety, lowered from 3 to 1. All patients showed a reduction of 2 or more points on the affective MS symptom scores. They scored significantly lower on the affective symptom MS scores at follow-up compared to baseline ($Z = 2.6$, $p = .01$) but not on the positive ($Z = 1.0$, $p>.1$) or the negative ($Z = 0.8$, $p>.1$) symptom MS scores.

Sixteen individuals were lost from the non-clinical control group, leaving 20 individuals. The people lost to follow-up were not significantly different from the rest of the sample in age ($t = -.41$, d.f. = 79, $p>.1$), IQ ($t = .14$, d.f. = 76, $p>.1$), or gender distribution ($\chi^2 = 2.2$, d.f. = 1, $p>.1$).

Despite their significant drop in positive symptom MS scores, the deluded group still had significantly higher positive symptom MS scores (Mann-Whitney tests: $U = 30$, $p<.05$) than the psychiatric control group. There was no significant difference between the two groups in affective ($U = 60$, $p>.1$) or negative ($U = 64.5$, $p>.1$) symptom MS scores. There was no longer any difference between the two groups in time spent in hospital ($U = 97$, $p>.1$), although a significant

[2]Where no MS was available remission was determined by case-note review and contacting the psychiatrist in charge of the patient's care.

difference emerged in age of onset of illness ($U = 81$, $p = .05$), with the deluded group having a lower age of onset.

The significant differences between the three groups on the PDI ($F = 15.8$, d.f. $= 2, 68$, $p < .001$), the DSSI ($F = 53.2$, d.f. $= 2, 76$, $p < .001$), and the HADS anxiety ($F = 23.7$, d.f. $= 2, 76$, $p < .001$) and depression ($F = 44.1$, d.f. $= 2, 76$, $p < .001$) scores remained at follow-up. However, all differences between the two psychiatric groups disappeared, with only the differences between the deluded group and the non-clinical controls remaining on the two delusion measures (PDI: Scheffe $= 53.1$, $p < .05$; DSSI: Scheffe $= 5.2$, $p < .05$), and only the difference between the psychiatric and the non-clinical control groups remaining on anxiety (Scheffe $= 4.5$, $p < .01$). The non-clinical controls continued to score significantly lower on depression than both the deluded (Scheffe $= 3.8$, $p < .05$) and the psychiatric control (Scheffe $= 4.2$, $p < .05$) groups.

The significant differences between the three groups in age ($F = 8.9$, d.f. $= 2, 52$, $p < .001$) and verbal IQ ($F = 14.5$, d.f. $= 2, 51$, $p < .001$) remained, with the psychiatric control group continuing to be significantly older than both the deluded (Scheffe $= 10.2$, $p < .01$) and the non-clinical control (Scheffe $= 12.2$, $p = .001$) groups. However, a different pattern of differences was found with IQ, with the deluded group having significantly lower IQ scores than both the non-clinical (Scheffe $= 20.9$, $p < .001$) and the psychiatric (Scheffe $= 12$, $p < .05$) control groups. As at baseline, the ratio of males to females differed only in the deluded group, which consisted of males only.

Procedure

Each participant completed the negative priming task, the 'beads' task, the PIT, the Quick Test, and a number of questionnaires, as described above. Each participant was tested on two occasions, once when they were actively symptomatic (either deluded or depressed/anxious), and once when they were remitted. The presentation of tasks was randomised across participants to counteract potential order effects at baseline, but the order was kept constant for each participant between baseline and follow-up. The mean length of time between the two testing sessions was 17.4 weeks in the deluded group (ranging from 6 to 41 weeks), and 33.4 weeks in the psychiatric control group (ranging from 4 to 68 weeks). Participants in the non-clinical control group were also tested twice, with a mean interval of 35.6 weeks (ranging from 27 to 46 weeks). The shorter interval between testing sessions for the deluded group was due to the shorter admission periods for this patient group, and because their follow-up was prioritised over the non-clinical control group. Patients were either tested in hospital (baseline) or at home (follow-up), while the non-clinical controls were tested at the Institute of Psychiatry.

Results

Separate analyses were conducted at baseline and follow-up on each task to maximise power when patient groups were symptomatic. Due to the significant group differences in age and verbal IQ, univariate or multivariate analyses of co-variances (ANCOVAs or MANCOVAs), co-varying out age and IQ together, were first carried out on the dependent variables to determine the impact

of these differences on test scores and to check for any interactions with group effects. Where significant effects of covariates occurred the relevant variables were covaried out of subsequent analyses. Where no significant effects of covariates were found, either univariate (ANOVAs) or multivariate (MANOVAs) analyses of variances are reported to maximise the power of the analyses (since there were 3 deluded individuals with missing IQ data).

At follow-up 3 (group) × 2 (time) ANOVAs were carried out to investigate change over time on the dependent measures. Only the within-subjects and interaction effects are reported in these analyses, since the main group effects are influenced partly by the participants' performance at baseline, already reported in the baseline sections.

Negative priming

The two dependent measures in this task were reaction times (RTs) and number of errors made, both displayed in Table 2.

Baseline study

One non-clinical control and 2 deluded individuals did not have complete data on this task. RTs were entered into a 3 (condition) × 3 (group) MANCOVA, with IQ as covariate. A significant overall difference in RT was found between the groups ($F = 7.0$, d.f. $= 2, 72$, $p < .01$) with the non-clinical control group having faster RTs than both the psychiatric control ($F = 7.9$, d.f. $= 1, 54$, $p < .01$) and the deluded ($F = 21.9$, d.f. $= 1, 51$, $p < .001$) groups. The two psychiatric groups did not differ from each other ($F = 0.4$, d.f. $= 1, 38$, $p > .1$). The multivariate condition effect was also significant ($F = 4.2$, d.f. $= 2, 71$, $p < .05$). Within-subjects repeated contrasts revealed a significant Stroop (i.e., Stroop condition – control condition) effect ($F = 5.8$, d.f. $= 1, 72$, $p < .05$), while the negative priming (i.e., priming condition – Stroop condition) effect did not quite reach significance ($F = 3.6$, d.f. $= 1, 72$, $p = .06$). There was a trend for the interaction between group and condition ($F = 2.2$, d.f. $= 4, 144$, $p = .075$), and since we had a priori hypotheses of a significant interaction between group and negative priming, but not Stroop, further analyses were carried out. However, neither the interaction between group and the within-subject repeated contrast for the negative priming effect ($F = 1.9$, d.f. $= 2, 72$, $p > .1$), nor for the Stroop effect ($F = 2.2$, d.f. $= 2, 72$, $p > .1$) was significant.

Error rates were low overall, with an overall average of 2%. Errors were entered into a 3×3 MANOVA. A difference in overall errors was found between the groups ($F = 6.3$, d.f. $= 2, 75$, $p < .01$), with the non-clinical control group making fewer errors than both the psychiatric control ($F = 5.6$, d.f. $= 1, 55$, $p < .05$) and the deluded ($F = 10.1$, d.f. $= 1, 54$, $p < .01$) groups. The two psychiatric groups did not differ from each other ($F = 2.9$, d.f. $= 1, 41$, $p = .1$). The multivariate condition effect was also significant ($F = 10.4$, d.f. $= 2, 74$, $p < .001$). Repeated within-subjects contrasts indicated that participants made significantly more errors in the Stroop compared to the control condition ($F = 20.6$, d.f. $= 1, 75$, $p < .001$), but no difference was found between the negative priming and the Stroop conditions ($F = 1.6$, d.f. $= 1, 75$, $p > .1$). The group by condition interaction was also significant ($F = 3.2$, d.f. $= 4, 150$, $p < .05$). Interaction terms from the within-subject repeated contrasts indicated that this interaction was due to a significant group difference in the number of errors made in the Stroop compared to the control condition ($F = 6.9$, d.f. $= 2, 75$, $p < .01$), but not the negative priming compared to the Stroop condition ($F = 0.1$,

Table 2
Means and sds (in parentheses) of RTs and number of errors in the negative priming task in the three groups at baseline and follow-up

	Deluded group				Psychiatric controls				Non-clinical controls			
	RTs (in ms)		Number of errors		RTs (in msecs)		Number of errors		RTs (in msecs)		Number of errors	
	Baseline ($n = 21$)	Follow-up ($n = 17$)	Baseline ($n = 21$)	Follow-up ($n = 17$)	Baseline ($n = 22$)	Follow-up ($n = 18$)	Baseline ($n = 22$)	Follow-up ($n = 18$)	Baseline ($n = 35$)	Follow-up ($n = 20$)	Baseline ($n = 35$)	Follow-up ($n = 20$)
Negative priming condition	929 (171)	951 (143)	3.0 (5.5)	1.8 (1.9)	826 (248)	732 (136)	1.4 (1.8)	1.1 (1.6)	642 (143)	639 (142)	0.6 (1.0)	0.3 (0.6)
Stroop condition	896 (193)	937 (161)	2.7 (3.7)	1.5 (1.7)	792 (246)	693 (116)	1.1 (1.7)	0.8 (1.9)	621 (129)	615 (130)	0.3 (0.5)	0.7 (1.0)
Control condition	698 (175)	757 (186)	0.3 (0.5)	0.5 (0.9)	641 (257)	555 (82)	0.1 (0.3)	0.2 (0.5)	498 (91)	496 (90)	0.2 (0.4)	0.3 (0.6)

d.f. = 2, 75, $p > .1$). Further analyses showed that the significant within-subject contrast interaction was due a smaller difference in error rates between the Stroop and control conditions in the control group compared to both the psychiatric controls ($F = 6.2$, d.f. = 1, 55, $p < .05$) and the deluded ($F = 11.8$, d.f. = 1, 54, $p = .001$). The two psychiatric groups did not differ from each other ($F = 2.7$, d.f. = 1, 41, $p = .1$).

Follow-up study

RTs were entered into a 3×3 MANOVA. A significant overall difference in RT was found between the groups ($F = 29.0$, d.f. = 2, 52, $p < .001$) with the non-clinical control group having faster RTs than both the psychiatric control ($F = 4.6$, d.f. = 1, 36, $p < .05$) and the deluded ($F = 48.8$, d.f. = 1, 35, $p < .001$) groups. The psychiatric control group was also faster than the deluded group ($F = 26.7$, d.f. = 1, 33, $p < .001$). The multivariate condition effect was also significant ($F = 66.5$, d.f. = 2, 51, $p < .001$). Within-subjects repeated contrasts revealed both a significant Stroop (i.e., Stroop condition – control condition) effect ($F = 109.2$, d.f. = 1, 52, $p < .001$) and a significant negative priming (i.e., priming condition – Stroop condition) effect ($F = 7.1$, d.f. = 1, 52, $p = .01$). There was no interaction between group and condition ($F = 1.0$, d.f. = 4, 104, $p > .1$), and this was not looked at further.

Error rates were low overall, with an overall average of 1.6%. They were entered into a 3×3 MANOVA. A difference in overall errors was found between the groups ($F = 3.9$, d.f. = 2, 52, $p < .05$), with the non-clinical control group making fewer errors than the deluded group ($F = 10.6$, d.f. = 1, 35, $p < .01$). The two control groups did not differ from each other ($F = 0.9$, d.f. = 1, 36, $p > .1$), nor did the deluded and psychiatric control groups ($F = 2.3$, d.f. = 1, 33, $p > .1$). The multivariate condition effect was also significant ($F = 7.9$, d.f. = 2, 51, $p = .001$). Repeated within-subjects contrasts indicated that participants made significantly more errors in the Stroop compared to the control condition ($F = 10.1$, d.f. = 1, 52, $p < .01$), but no difference was found between the negative priming and the Stroop conditions ($F = 0.2$, d.f. = 1, 52, $p > .1$). There was a trend for the group by condition interaction ($F = 2.2$, d.f. = 4, 104, $p = .07$). However, separate interaction terms from the within-subject repeated contrasts did not reach significance (Stroop compared to the control condition: $F = 0.8$, d.f. = 2, 52, $p > .1$; negative priming compared to the Stroop condition: $F = 1.6$, d.f. = 2, 52, $p > .1$). Further analyses were therefore not carried out.

Change in negative priming between baseline and follow-up

The amount of negative priming was calculated as RT in priming condition – RT in Stroop condition and entered into a 3 (group) $\times$ 2 (time) ANOVA. There was no overall difference in negative priming over time ($F = 0.2$, d.f. = 1, 51, $p > .1$). The interaction between group and time was not significant either ($F = 0.03$, d.f. = 2, 51, $p > .1$), and these data were not analysed further.

Beads task

The three measures analysed in condition 1 were whether the choice of jar was correct, the number of beads to certainty, and percentage certainty. In condition 2 the variables analysed consisted of initial certainty (mean response to first 3 beads), final certainty (response to bead 10),

Table 3
Means and sds (in parentheses) for the 'beads' task in the three groups at baseline and follow-up

	Deluded		Psychiatric controls		Non-clinical controls	
	Baseline ($n = 22$)	Follow-up ($n = 17$)	Baseline ($n = 22$)	Follow-up ($n = 18$)	Baseline ($n = 36$)	Follow-up ($n = 20$)
Number of beads to certainty (cond. 1)	6.4 (5.1)	5.9 (5.0)	16.6 (8.2)	9.1 (5.1)	10.3 (7.1)	7.3 (5.4)
Initial certainty (cond. 2)	77.8 (15.0)	74.1 (15.3)	68.9 (14.8)	64.2 (16.0)	64.9 (13.8)	67.0 (10.2)
Disconfirmatory evidence (cond. 2)	−26.4 (31.4)	−20.4 (29.8)	−19.2 (30.5)	−10.7 (22.3)	−5.6 (14.2)	−2.7 (12.6)
Final certainty (cond. 2)	88.1 (11.3)	85.5 (22.7)	82.1 (16.5)	78.8 (23.3)	84.9 (19.7)	93.7 (10.8)

and reaction to disconfirmatory evidence (mean of response to bead 9 – bead 8 and of response to bead 4 – bead 3). These variables for both baseline and follow-up studies are presented in Table 3.

Baseline study

One psychiatric control had missing data in condition 1 of this task, and 1 non-clinical control and 1 deluded participant did not complete condition 2. Overall 96% of the total sample chose the right jar, and there was no significant difference between the groups ($\chi^2 = 2.9$, d.f. $= 2$, $p > .1$). One non-clinical control, two psychiatric controls, and none of the deluded group, chose the wrong jar.

A one-way ANOVA revealed a significant difference in number of beads to certainty across the three groups ($F = 11.9$, d.f. $= 2, 76$, $p < .001$). Independent-sample t-tests indicated that the deluded group requested significantly fewer beads than both the non-clinical ($t = 2.3$, d.f. $= 56$, $p < .05$) and psychiatric ($t = 4.9$, d.f. $= 41$, $p < .001$) controls. In turn, the non-clinical control group asked to see fewer beads than the psychiatric control group ($t = 3.1$, d.f. $= 55$, $p < .01$).

All groups had high percentage certainties in the correctness of their choice (deluded group: mean $= 81.1\%$, sd $= 21.7$; psychiatric controls: mean $= 87.1\%$, sd $= 16.4$; non-clinical controls: mean $= 86.7\%$, sd $= 11.8$). There was no significant difference between the groups ($F = 1.0$, d.f. $= 2, 74$, $p > .1$).

The mean responses to each bead in condition 2 for the three groups are illustrated in Fig. 1. A one-way ANOVA revealed a significant difference in initial certainty across the three groups ($F = 5.5$, d.f. $= 2, 76$, $p < .01$). Independent-sample t-tests indicated that the deluded group made significantly initially higher estimates than both the non-clinical ($t = 3.3$, d.f. $= 55$, $p < .01$) and the psychiatric ($t = 2.0$, d.f. $= 42$, $p = .05$) control groups. There was no difference between the two control groups ($t = 1.0$, d.f. $= 55$, $p > .1$). No difference was found between the groups in final certainty (one-way ANOVA: $F = 0.7$, d.f. $= 2, 76$, $p > .1$).

A one-way ANOVA revealed a significant difference in reaction to disconfirmatory evidence between the groups ($F = 5.1$, d.f. $= 2, 76$, $p < .01$). Independent-sample t-tests indicated that the deluded group changed the direction of their estimates significantly more than the non-clinical

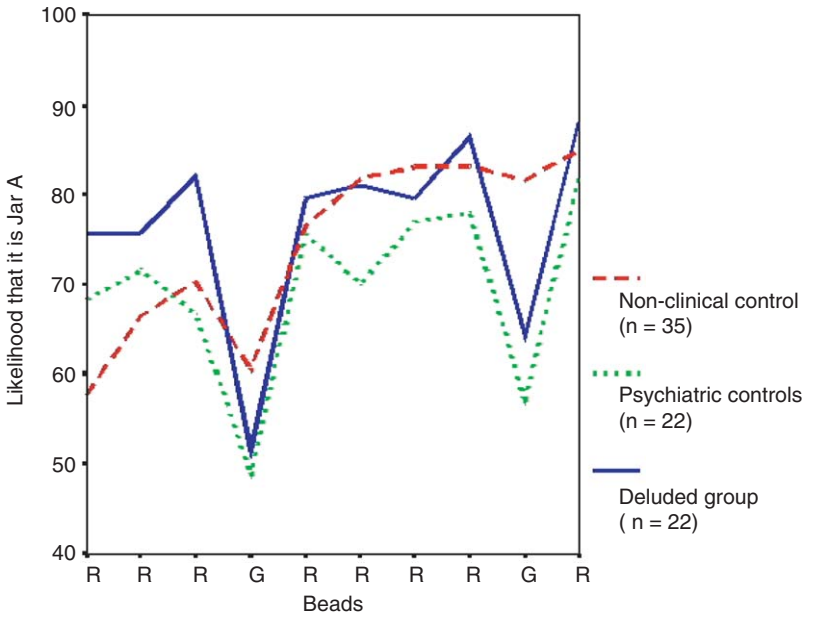

Fig. 1. Mean responses to each bead (R = red bead; G = green bead) in condition 2 in the three groups at baseline.

control ($t = 2.9$, d.f. $= 55$, $p < .01$), but not the psychiatric control ($t = 0.8$, d.f. $= 42$, $p > .1$) groups. There was a trend for the psychiatric control group to also change the direction of their estimate more than the non-clinical control group ($t = 2.0$, d.f. $= 55$, $p = .06$).

Follow-up study

Overall 89% of the total sample chose the right jar, and there was no significant difference between the groups ($\chi^2 = 4.8$, d.f. $= 2$, $p > .05$). Four psychiatric controls, two deluded patients, and none of the non-clinical controls, chose the wrong jar.

A one-way ANOVA showed no significant difference in number of beads to certainty across the three groups ($F = 1.7$, d.f. $= 2, 52$, $p > .1$), nor in percentage certainty in the correctness of their choice ($F = 0.3$, d.f. $= 2, 52$, $p > .1$). All groups had fairly high percentage certainties (deluded group: mean $= 87.9\%$, sd $= 16.0$; psychiatric controls: mean $= 84.6\%$, sd $= 13.5$; non-clinical controls: mean $= 83.5$, sd $= 21.1$).

The distribution of responses to the sequence of beads in condition 2 is represented graphically in Fig. 2. A one-way ANOVA showed no significant difference in initial certainty across the three groups ($F = 2.4$, d.f. $= 2, 52$, $p = .1$), but a trend was found in final certainty (one-way ANOVA: $F = 2.8$, d.f. $= 2, 52$, $p = .07$). Independent t-tests revealed a significant difference between the non-clinical and psychiatric control groups ($t = 2.5$, d.f. $= 36$, $p < .05$), with the psychiatric control group having a lower final certainty. No differences were found between the deluded and the two other groups (compared to non-clinical controls: $t = 1.5$, d.f. $= 35$, $p > .1$; compared to psychiatric controls: $t = -0.9$, d.f. $= 33$, $p > .1$).

A one-way ANOVA revealed a trend in reaction to disconfirmatory evidence between the groups ($F = 2.9$, d.f. $= 2, 52$, $p = .06$). Independent-sample t-tests indicated that the deluded

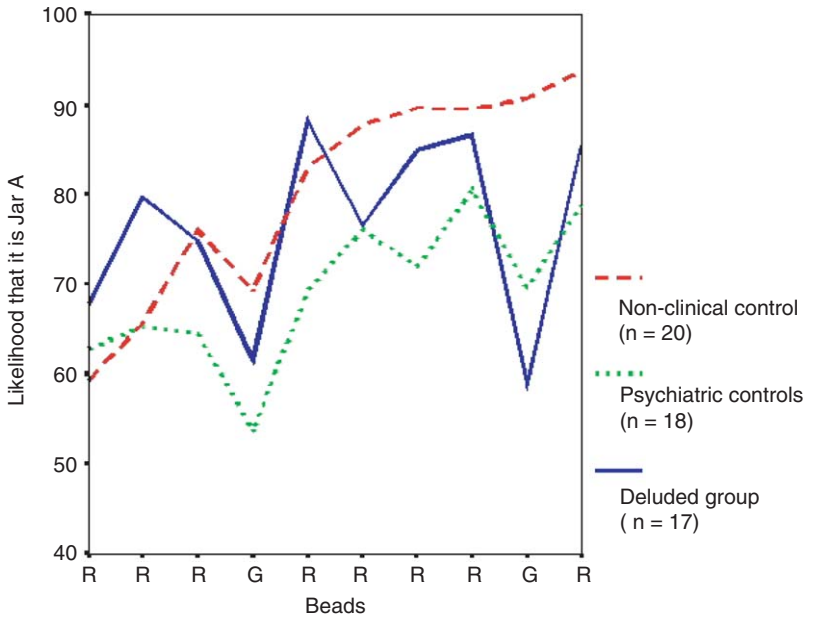

Fig. 2. Mean responses to each bead in condition 2 in the three groups at follow-up.

group changed the direction of their estimates significantly more than the non-clinical control ($t = 2.3$, d.f. $= 35$, $p < .05$), but not the psychiatric control ($t = 1.1$, d.f. $= 33$, $p > .1$) groups. There was no difference between the psychiatric and the non-clinical control groups ($t = 1.3$, d.f. $= 36$, $p > .1$).

Changes on the beads task between baseline and follow-up

A 3 (group) $\times$ 2 (time) ANOVA showed a significant within-subject effect ($F = 16.5$, d.f. $= 1, 51$, $p < .001$), with fewer beads being seen overall at follow-up. The interaction between group and time was also significant ($F = 5.4$, d.f. $= 2, 51$, $p < .01$). Related-samples t-tests showed that both the control groups showed a significant reduction in number of beads seen between baseline and follow-up (non-clinical controls: $t = 2.6$, d.f. $= 19$, $p < .01$; psychiatric controls: $t = 3.9$, d.f. $= 16$, $p = .001$), while the deluded group remained stable ($t = 0.15$, d.f. $= 16$, $p > .1$).

For initial estimates in Condition 2, a 3×2 ANOVA revealed a significant within-subject effect ($F = 4.3$, d.f. $= 1, 52$, $p < .05$), with lower estimates being made overall at follow-up. The interaction between group and time was not significant ($F = 1.0$, d.f. $= 2, 52$, $p > .1$), and this was not examined further. Neither the within-subject effect ($F = 0.01$, d.f. $= 1, 52$, $p > .1$), nor the interaction between group and time ($F = 0.7$, d.f. $= 2, 52$, $p > .1$) was significant for the final estimates, and no further analyses were carried out. Similarly, neither the within-subject effect ($F = 1.6$, d.f. $= 1, 52$, $p > .1$), nor the interaction between group and time ($F = 0.08$, d.f. $= 2, 52$, $p > .1$) was significant for the effect of disconfirmatory evidence, and no further analyses were carried out.

Table 4
Number of internal attributions made on the PIT (means and sds (in parentheses)) in the three groups at baseline and follow-up

	Deluded		Psychiatric controls		Non-clinical controls	
	Baseline ($n = 23$)	Follow-up ($n = 17$)	Baseline ($n = 21$)	Follow-up ($n = 18$)	Baseline ($n = 36$)	Follow-up ($n = 20$)
Positive events	3.09 (1.0)	3.18 (1.5)	2.19 (1.4)	2.50 (1.1)	2.64 (1.3)	2.90 (1.5)
Negative events	3.09 (1.1)	3.12 (1.3)	2.91 (1.0)	2.56 (1.5)	2.31 (1.3)	1.80 (1.4)

PIT

All analyses were conducted on internal attributions only (since the choice of internal and external attributions were mutually exclusive). The mean internal attributions for positive and negative events in the three groups for both the baseline and follow-up studies are given in Table 4.

Baseline study

One psychiatric control participant did not have complete data on this task. A 2 (type of event) × 3 (group) ANCOVA, with age as covariate, revealed a significant main group effect ($F = 3.4$, d.f. $= 2, 76$, $p < .05$). Between-subjects contrasts revealed that the deluded group made significantly more internal attributions overall than the non-clinical control group ($t = 2.5$, d.f. $= 76$, $p = .01$), and there was a trend compared to the psychiatric control group ($t = 1.7$, d.f. $= 76$, $p = .09$). The two control groups did not differ from each other ($t = 0.4$, d.f. $= 76$, $p > .1$). The within-subject effect was significant ($F = 5.7$, d.f. $= 1, 76$, $p < .05$), with more internal attributions being made for negative events overall. The group by type of event interaction was also significant ($F = 5.5$, d.f. $= 2, 76$, $p < .01$). Further analyses revealed that this significant interaction was due to the psychiatric control group making significantly more internal attributions for negative than for positive events compared to both the deluded ($F = 6.6$, d.f. $= 1, 41$, $p = .01$) and the non-clinical control ($F = 11.3$, d.f. $= 1, 54$, $p = .001$) groups. There was no difference between the non-clinical control and the deluded groups ($F = 0.9$, d.f. $= 1, 56$, $p > .1$).[3]

A-posteriori analyses were carried out to investigate the two forms of paranoia postulated by Trower and Chadwick (1995). Following their definitions, patients who had endorsed item 15 of the PDI (''Do you ever feel that you have sinned more than the average person'' were classified as belonging to the ''bad-me'' paranoia group ($n = 8$), while those who had endorsed the persecution

[3]Since Bentall et al.'s (1994; 2001) model pertains specifically to persecutory delusions, a posteriori analyses were carried out including only the deluded patients whose current delusions were of a paranoid content. Fourteen of the 23 patients had answered ''yes'' to the two PDI questions assessing persecution (for those patients with missing data on these two questions ($n = 3$) their paranoid status was determined by case-note review). A 2 × 3 ANCOVA, with both age and IQ as covariates, revealed a similar pattern of results, although the significant main group effect was lost ($F = 0.2$, d.f. $= 2,64$, $p > .1$).

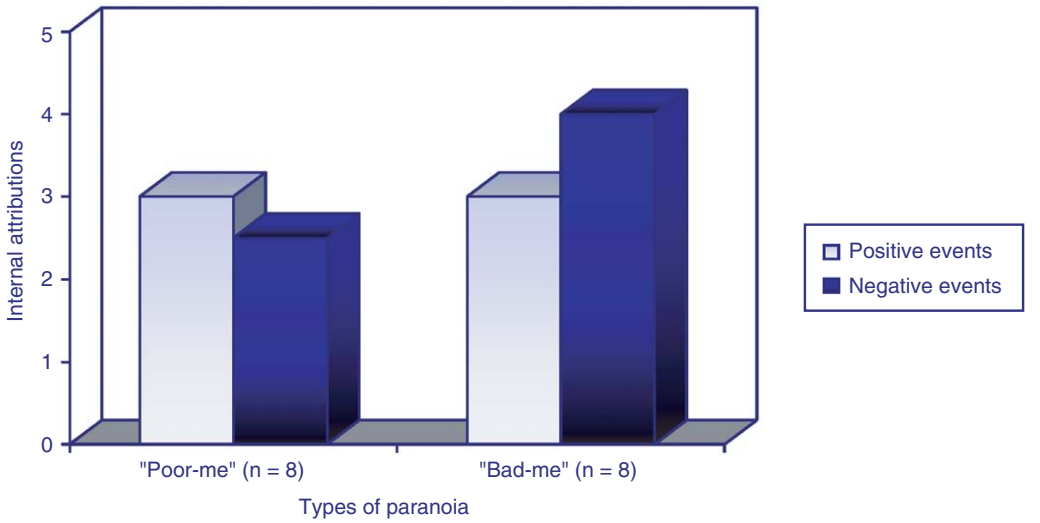

Fig. 3. Internal attributions for positive and negative events in the "poor-me" and "bad-me" paranoia groups at baseline.

items, but not item 15 of the PDI, were classified as belonging to the "poor-me" paranoia group ($n = 8$). The mean internal attributions for positive and negative events in these two groups are displayed in Fig. 3.

As can be seen in Fig. 3 the implicit attributional style of the two groups differed markedly, with the "poor-me" group showing a self-serving bias, while the "bad-me" group displayed a depressive attributional style. A 2×2 ANCOVA, with age as a covariate, revealed that neither the main group effect ($F = 1.6$, d.f. $= 1, 13$, $p > .1$), nor the within-subjects effect ($F = 0.4$, d.f. $= 1, 13$, $p > .1$) was significant. However, the interaction effect was significant ($F = 4.7$, d.f. $= 1, 13$, $p = .05$).[4] Further analyses showed that this interaction was due to the "bad-me" group making significantly more internal attributions for negative events than the "poor-me" group (1-way ANCOVA: $F = 4.9$, d.f. $= 1, 13$, $p < .05$), while there was no difference between the number of attributions made for positive events (1-way ANCOVA: $F = 0.1$, d.f. $= 1, 13$, $p > .1$).

Follow-up study

A 3×2 ANOVA showed a significant main group effect ($F = 4.4$, d.f. $= 2, 52$, $p < .05$). Between-subjects contrasts revealed that the deluded group made more internal attributions than both the non-clinical ($t = 2.9$, d.f. $= 52$, $p < .01$) and the psychiatric ($t = 2.2$, d.f. $= 52$, $p < .05$) control groups. The two control groups did not differ from each other ($t = 0.7$, d.f. $= 52$, $p > .1$).

[4]These analyses were repeated in the other two groups to investigate whether this attributional style was specific to the deluded group. The non-clinical controls had 4 individuals who qualified as "poor-me", and 9 who qualified as "bad-me"; the psychiatric control group had 6 individuals who qualified as "poor-me", and 7 who qualified as "bad-me". Neither the non-clinical controls ($F = 2.9$, d.f. $= 1,10$, $p > .1$), nor the psychiatric controls ($F = .32$, d.f. $= 1,10$, $p > .1$) showed a significant interaction.

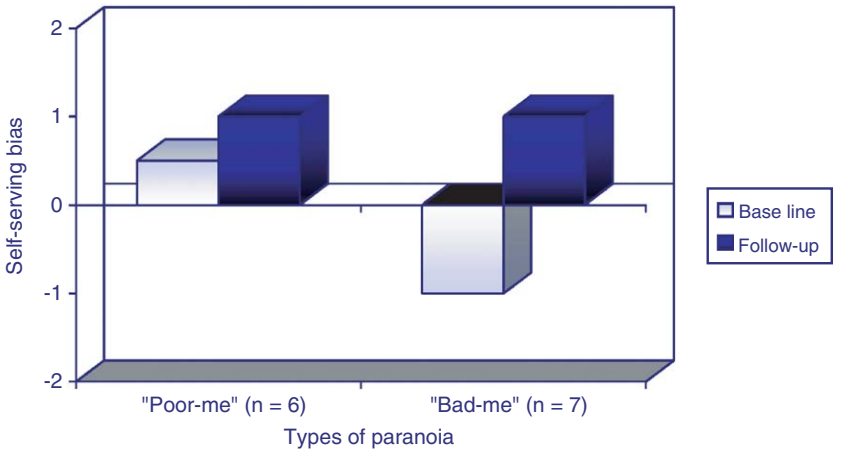

Fig. 4. Self-serving bias in the two paranoid groups at baseline and follow-up.

Neither the within-subject effect ($F = 1.6$, d.f. $= 1, 52$, $p > .1$), nor the group by type of event interaction ($F = 1.6$, d.f. $= 2, 52$, $p > .1$) was significant, and this was not examined further.[5]

Seven of the original 8 patients classified as "bad-me" paranoia, and 6 of the original 8 patients belonging to the "poor-me" paranoid group, had follow-up data. Unlike at baseline, a 2×2 ANOVA on these two groups did not reveal any significant differences (main group effect: $F = 1.6$, d.f. $= 1, 1$, $p > .1$; within-subjects effect: $F = 1.5$, d.f. $= 1, 11$, $p > .1$; interaction: $F = 0.5$, d.f. $= 1, 11$, $p > .1$).

Changes on the PIT between baseline and follow-up

3 (group) $\times$ 2 (time) ANOVAs were carried out to investigate change over time on the self-serving bias (number of internal attributions for negative events – number of internal attributions for positive events). There was a significant within-subject effect ($F = 4.5$, d.f. $= 1, 52$, $p < .05$), with an overall increase in self-serving bias at follow-up. The interaction between group and time was not significant ($F = 0.08$, d.f. $= 2, 52$, $p > .1$).

A further analysis was carried out to compare the change over time in the "poor-me" and "bad-me" paranoid groups. There was no change over time ($F = 1.8$, d.f. $= 1, 11$, $p > .1$), nor interaction with group ($F = 1.1$, d.f. $= 1, 11$, $p > .1$). Although no significant effects were found, presumably because of the small numbers, a graphical illustration of the change over time in self-serving bias in these two groups (Fig. 4) seems to show a bigger increase in self-serving bias in the "bad-me" group.

Relationships between tasks

The relationships between tasks were analysed only at baseline when the patient groups were florid. Only correlations of $p < .01$ were considered to take into account the effects of multiple

[5]A posteriori analyses were carried out including only the persecutory sub-group described above, of whom 11 had follow-up data. A similar pattern of results was found.

testing. Non-parametric correlations (Spearman's rho) were calculated since correlations are more sensitive than ANOVAs to deviations from a normal distribution. Correlations between tasks were carried out for each group separately since it cannot be assumed that similar relationships apply for each group.

Significant correlations were found for only three variable dyads, out of a total of 28. The three significant pairs consisted of number of beads seen in condition 1 of the 'beads' task and final certainty in condition 2 of the same task; initial certainty and reaction to disconfirmatory evidence, both in condition 2 of the 'beads' task; and final certainty in condition 2 of the 'beads' task and self-serving bias in the PIT.

An inverse relationship between the number of beads seen and final certainty was found for the non-clinical control group (rho $= -.44$, $n = 35$, $p < .01$), and there was a trend in the psychiatric control group (rho $= -.42$, $n = 21$, $p = .06$). However, no such effect was found in the deluded group (rho $= -.35$, $n = 22$, $p > .1$). Similarly, an inverse relationship between initial certainty and reaction to disconfirmatory evidence was found for both the psychiatric (rho $= -.68$, $n = 22$, $p = .001$) and the non-clinical (rho $= -.52$, $n = 35$, $p < .01$) control groups, but not the deluded group (rho $= -.25, n = 22, p > .1$). The correlation between final certainty and self-serving bias was significant in the non-clinical control group only (rho $= .43, n = 35, p = .01$), with neither correlation reaching significance in the psychiatric control (rho $= -.13, n = 21, p > .1$) or deluded (rho $= .34, n = 22, p > .1$) groups.

Relationships between tasks and clinical measures

Again the relationships between tasks and clinical measures were only analysed at baseline when the patient groups were florid. To reduce the number of correlations carried out, only the measures where some significant group differences were found were included (number of beads to certainty in Condition 1 of the 'beads' task, initial estimates and reaction to disconfirmatory evidence in Condition 2 of the 'beads' task, and self-serving bias from the PIT). Only correlations of $p < .01$ were considered to take into account the effects of multiple testing. Non-parametric correlations (Spearman's rho) were calculated since correlations are more sensitive than ANOVAs to deviations from a normal distribution. The correlations were carried out for the pooled data set in this instance since there were no reasons to assume different relationships between clinical measures and the dependent variables (analyses including the MS measures were carried out for the two psychiatric groups only).

Only four out of the 28 correlations were significant at the .01 level. The number of beads requested in condition 1 of the 'beads' task was inversely correlated with the positive symptom MS measure (rho $= -.49, n = 39, p < .01$), and positively correlated with the affective MS measure (rho $= .42, n = 39, p < .01$). Further analyses looking at individual symptom measures revealed significant inverse relationships with both delusions (rho $= -.48, n = 39, p < .01$) and incoherence of speech (rho $= -.47, n = 39, p < .01$), and a positive relationship with anxiety (rho $= 0.41, n = 39, p = .01$). No significant associations were found for the hallucinations (rho $= -.24, n = 39, p > .1$) or depression (rho $= .29, n = 39, p > .05$) measures. The number of beads was also inversely related to the DSSI (rho $= -.29, n = 78, p = .01$), while the initial certainty in condition 2 of the 'beads' task was positively correlated with the DSSI (rho $= .39, n = 78, p < .01$).

Discussion

Negative priming

Baseline study

The non-clinical control group was significantly faster and made fewer errors than both psychiatric groups, which did not differ from each other. An overall significant Stroop effect, and near-significant negative priming effect, were found for the RTs. Participants made more errors overall in the Stroop compared to the control, but not the negative priming, conditions. Contrary to our predictions, few interactions were found between condition and group, with no difference being found between the groups either on the Stroop or the negative priming effects. The non-clinical control group showed a significantly smaller difference in error rates between the Stroop and control conditions than both the psychiatric groups, which did not differ from each other.

These findings fail to replicate the 12 previous studies showing reduced negative priming in psychotic samples. However, only two of these studies (Williams, 1996; Peters et al., 2000) showed a specific relationship between current positive symptoms and negative priming. Furthermore, neither Williams nor Peters and her colleagues selected delusional individuals specifically, as was the case in the present study. It is therefore possible that a deficit in cognitive inhibition is related to the general reality distortion syndrome (as described by Liddle, 1987), but not to delusions specifically. Indeed, there is evidence that the general ''positive symptoms'' factor may consist of two separate dimensions, namely a ''cognitive-perceptual'' and a ''paranoid'' factor (Minas, Stuart, Klimidis, & Jackson, 1992). The ''cognitive-perceptual'' factor seems to relate to perceptual and thought disturbances (i.e., hallucinations and first-rank symptoms), while the ''paranoid'' factor is linked to suspiciousness and ideas of reference (i.e., delusions). Cognitive inhibition may be related to the former, but not the latter, factor.

Follow-up study

Similarly to baseline, no significant differences were found between the groups at follow-up either on the Stroop or the negative priming effects, contrary to our predictions. Both control groups were overall significantly faster than the deluded group on the negative priming task, as was the non-clinical compared to the psychiatric control group. The deluded group also made more errors than the non-clinical controls, but did not differ from the psychiatric controls. The two control groups did not differ from each other. Significant Stroop and negative priming effects were found for the RTs. Participants made more errors overall in the Stroop compared to the control, but not the negative priming, conditions. There was no significant change over time between the two testing sessions on negative priming, either as a main effect or as an interaction with group. Therefore the deluded group continue to show normal cognitive inhibition when they are in remission. These finding are in contradiction to the large body of work showing reduced negative priming in schizotypal, non-symptomatic individuals. However, none of these studies selected delusion-prone participants specifically, with the relationship with negative priming being confined to positive symptomatology in general. As discussed above, it is therefore possible that cognitive inhibition is related to perceptual-cognitive disturbances rather than aberrant beliefs.

Beads task

Baseline study

As predicted, the deluded group requested to see significantly fewer beads before making up their mind in Condition 1, and made significantly higher initial estimates in condition 2, than both control groups. There was no difference between the groups in final estimates. There were no differences between the groups in the number of participants who chose the wrong jar, nor in certainty in the correctness of their choice. Therefore, whether the task requires participants to stop the search for evidence, or to make probability estimates on evidence presented to them, deluded individuals are making up their minds more quickly than the other two groups, although all groups reach a common conclusion by the end of the task. These results replicate the numerous previous studies showing a JTC bias in deluded individuals (Garety & Freeman, 1999; Garety et al., in press), and confirm that this is a robust finding.

Previous studies have also shown a disconfirmatory bias in deluded individuals relative to non-clinical controls (Garety et al., 1991), so that they tend to shift their probability estimates downwards when presented with contradictory evidence, rather than sticking to their initial estimates. However, deluded patients have not been differentiated from depressed (Young & Bentall, 1997a) or obsessive-compulsive-disorder (Fear & Healy, 1997) patient groups on this measure. These results were replicated in the present study: the deluded group changed the direction of their estimates when faced with disconfirmatory evidence significantly more than the non-clinical, but not the psychiatric, control groups. There was a trend for the psychiatric control group to also change their estimates more than the non-clinical control group. Depressed/anxious patients also requested to see significantly more information before making up their mind in Condition 1. This pattern of results implies that the greater revision of estimates, and the high degree of cautiousness seen in the psychiatric control group, may be due to a lack of confidence and/or difficulty in making decisions, which are both prominent symptoms of depression and anxiety. Further work disentangling the disconfirmatory bias in deluded and other psychiatric groups is clearly needed to address this issue.

Follow-up study

As was found at baseline, there were no differences between the groups in the number of participants who chose the wrong jar, nor in certainty in the correctness of their choice in condition 1 of the beads task.

Significant within-subject differences between baseline and follow-up in number of beads to certainty in Condition 1, and in initial estimates in Condition 2, would suggest that performance on this task is subject to practice effects, with participants asking to see fewer beads, and making lower estimates at follow-up compared to baseline. No differences were found between the groups in these two measures when the psychiatric groups were in remission. A lack of difference is always problematic to interpret, but one obvious possibility would be that, contrary to our predictions, the deluded group is no longer showing the JTC bias when remitted. There was, however, an interaction between group and change over time in beads

to certainty, such that both the control groups showed a significant reduction in number of beads seen, while the deluded group did not. This is despite the fact that the testing interval between baseline and follow-up was shorter for the deluded than the other two groups. The lack of change in the deluded group is also unlikely to be due to a floor effect, since the mean number of beads asked for by the deluded group at baseline was 6.4, allowing enough room for a potential reduction (bearing in mind that 53% of the 100 patients in the Garety et al. (in press) study made up their mind on the basis of two beads or less, and that the present mean was in fact higher than in a number of other studies).[6] In contrast, no interaction with group was found for initial estimates, with all groups showing the same practice effect. This pattern of results would suggest that the lack of difference on these two measures at follow-up are perhaps not equivalent. We would propose tentatively that for beads to certainty (i.e., the data-gathering part of the task), the deluded group remains stable over time, and therefore continues to show the JTC bias demonstrated at baseline even when they are remitted; the lack of difference between the groups at follow-up in this case is likely to be due to a practice effect in the two control groups. However, for initial estimates (i.e., the probabilistic judgment part of the task), the deluded group seems to be normalising their performance over time, and no longer differs from other groups when in remission.

The results on the two other measures of Condition 2 remained similar to baseline. Thus, the deluded group did not differ from the two other groups in final estimates at follow-up, and they changed the direction of their estimates when faced with disconfirmatory evidence significantly more than the non-clinical, but not the psychiatric, control groups. As proposed above, it is likely that the psychiatric group are revising their estimates due to lack of confidence rather than a JTC bias. The finding that their final estimates are lower than the non-clinical control group would support this explanation. There was no overall change between the two time points, nor was there an interaction with group, on either final estimates or reaction to disconfirmatory evidence. These results suggest that unlike the draws to decision in Condition 1 and the initial certainty in Condition 2, these measures were not subject to a practice effect, and the three groups did not alter their performance over time differentially.

Overall the findings on the beads task indicate that deluded individuals continue to show the JTC bias when they are remitted, although this is more clearly demonstrated when they are asked to gather data or are faced with contradictory evidence, compared to making probability judgments. This pattern suggests that the JTC bias may be implicated in more than just the maintenance of delusions, since it continues to be present after they have abated. Recent findings have also demonstrated that the JTC bias is present in healthy individuals who are high in delusional ideation (Colbert & Peters, 2002), or in help-seekers at high-risk for psychosis (Broome et al., 2003), but who do not show florid signs of pathology. Interestingly, the JTC bias was also more clearly evident with the data-gathering part of the task in the Colbert & Peters study, with no differences being apparent in initial certainty for condition 2. Taken together, these findings are consistent with the proposal that the JTC bias may be involved in the formation of delusional beliefs, rather than delusions recruiting this bias once they are activated.

[6] Garety et al. (1991) had a mean of 2.7; Dudley et al (1997a) had a mean of 2.4.

PIT

Baseline study

Overall more internal attributions were made for negative than for positive events across the three groups. The deluded group made significantly more internal attributions overall than the non-clinical, but not the psychiatric, control groups, suggesting that deluded patients are perhaps more self-focused than the non-clinical participants, attributing both positive and negative events to themselves. This finding would support claims that self-focus is a non-specific process that is common across psychopathologies, including psychosis (Ingram, 1990). A significant interaction was also found between group and type of event, with the psychiatric control group making significantly more internal attributions for negative than for positive events compared to both the deluded and the non-clinical control groups, which did not differ from each other. Therefore while the psychiatric control group exhibited the predicted depressive attributional style, the deluded patients had a similar attributional style to the non-clinical rather than to the depressed/anxious group on this task.

These findings do not replicate previous studies (Lyon et al., 1994; Krstev et al., 1999) demonstrating an implicit depressive attributional bias in deluded patients. One possibility may have been that the current sample was not a purely paranoid group, unlike in previous studies which specifically targeted patients with persecutory delusions. Indeed Sharp et al. (1997) showed that attributional biases were not found in individuals with non-persecutory and non-grandiose delusions. However, the exclusion of patients with non-persecutory delusions did not alter the findings. Although this paranoid sub-group no longer exhibited an excess of internal attributions overall, it is likely that this was due to loss of power rather than excessive self-focus being specific to non-paranoid deluded patients.

Another potential explanation may be that the current sample differed from previous ones in levels of depression. While Lyon et al's and Krstev et al.'s deluded patients showed relatively high levels of depression on two separate versions of the Beck Depression Inventory (BDI; Beck & Beamesderfer, 1974), the current sample did not show significant levels of depression (mean HADS score = 6.1; a score of 8–10 is considered borderline). It is therefore possible that the depressive implicit attributional style found by Lyon, Krstev, and their colleagues was representative of their depressive, rather than delusional, status.

A further option may be that negative self-representations are more likely to be found in some types of paranoia than others. Post hoc analyses of the deluded sample divided into "poor-me" and "bad-me" paranoia (Trower & Chadwick, 1995) by their responses on PDI items confirmed that there was a significant difference in implicit attributional style between those two groups: while the "poor-me" paranoia group displayed a self-serving bias, the "bad-me" paranoia group exhibited the expected depressive attributional style. This interaction was specific to the deluded group, with no such relationships being found in the two control groups. The difference between the two deluded groups, however, was only significant for negative events, with the "bad-me" group making more internal attributions than the "poor-me" group. Both groups made equal numbers of internal attributions for positive events, in line with the literature which suggests that differences between deluded and other groups are clearer for negative, than for positive, events (Garety & Freeman, 1999). This distinction may be due to the greater likelihood of negative events happening to psychotic populations, and therefore rendering such events more salient in experimental tasks.

It should be noted that these post hoc findings are based on small numbers and should therefore be interpreted with due caution. Furthermore, although we picked the PDI items which we felt best reflected the definitions offered by Trower & Chadwick (1995), the method of classification into the two types of paranoia was crude and idiosyncratic to this study, and potentially confounded religious beliefs with paranoia (since the crucial item on the PDI for separating the two groups related to sinning). Further work with a priori, valid distinctions between the two types of paranoia is clearly needed to substantiate the current findings.

Follow-up study

Despite the fact that the psychiatric control group are no longer showing the depressive attributional style they displayed at baseline, there was no differential pattern of change between the groups over the two time points, with all groups showing an increase in self-serving bias.

Similarly to baseline, the deluded group made significantly more internal attributions overall than the two control groups after their delusions had remitted. This was not mediated by type of event, so that both positive and negative events were attributed to the self. These results suggest that the increased self-focus common across psychopathologies (Ingram, 1990) is particularly pronounced in deluded individuals and is stable over time. As at baseline, the exclusion of patients with non-persecutory delusions did not alter the findings.

With the abatement of their delusions the differences between the "poor-me" and "bad-me" paranoid groups disappeared. Although the numbers were too small to demonstrate a significant interaction between group and change over time, these results suggest tentatively that the depressive attributional style shown by the "bad-me" paranoid patients fluctuates with the course of delusions and normalises when their delusions are remitted. This pattern of results reflects that found for the psychiatric control group, which no longer exhibited a depressive attributional style once their pathology had remitted. These findings are also in accordance with the study by Krstev et al. (1999), who reported a much weaker, inverse relationship between self-serving bias and suspiciousness in first-episode patients who had been tested when they were relatively stable, compared to the more chronic, symptomatic individuals tested by Lyon et al. (1994). This would suggest that attributional style is involved in the maintenance of, or occurs as a consequence of, certain paranoid delusions, rather than being causally implicated in their formation.

Relationship between the tasks

The relationships between task measures were only examined at baseline when the two patient groups were florid. Significant correlations were found for only three variable dyads, out of a total of 28, suggesting that the cognitive processes involved in the three tasks are relatively independent. Participants who asked for more beads before making up their mind in choosing a jar in Condition 1 also had lower final estimates in Condition 2. However, this was only true for the two control, and not the deluded, groups. Similarly, the overall significant inverse relationship found between initial certainty and reaction to disconfirmatory evidence was found for both the psychiatric and non-clinical control, but not the deluded, groups. Both of these correlations suggest that in the two control groups individuals' performance on these measures are mediated by confidence in one's decision making: greater cautiousness initially is related to lower levels of final certainty and a greater tendency to revise estimates when presented with contradictory

evidence. However such a link was not found in the deluded group. These results give some credence to the above speculation that the disconfirmatory bias found in both deluded and depressed/anxious individuals originates from different processes.

Relationships between task and clinical measures

Again the relationship between task and clinical measures were only explored at baseline when the patients were in an acute phase. Only four out of the 28 correlations were significant. Three of these significant relationships were with the number of beads seen in condition 1. People showing higher levels of positive symptoms on the MS requested to see fewer beads before choosing a jar. This significant relationship was due to correlations with both the delusion and incoherence of speech, but not the hallucination, symptom measures. The number of beads seen was also inversely related to the delusion-symptom-state-inventory (DSSI; Foulds & Bedford, 1975), but not the 21-item PDI (Delusions Inventory, Peters et al., 2004), which measures less florid symptomatology. In contrast, individuals showing higher levels of affective symptoms on the MS (with a significant contribution from the anxiety,[7] but not the depression, measure) asked to see more beads. The fact that the same measure had relationships in opposite directions with positive and neurotic symptoms would provide further support for the above speculation that different processes are at play in affecting performance on this task in neurotic and psychotic individuals.

Participants scoring highly on the DSSI, but not the PDI, also made higher initial estimates in condition 2. This measure was not related to the MS positive symptom measure, although it was significant at .05 rather than the more stringent .01 level used in this study.

General discussion

The purpose of this study was to investigate the longitudinal course of cognitive biases postulated to be associated with delusional beliefs. At baseline deluded individuals exhibited a JTC reasoning bias: i.e., they made decisions on the basis of limited evidence and were more likely to revise their estimates when faced with disconfirmatory evidence. The JTC bias remained stable over time despite remission, although this was seen more clearly with data-gathering and reaction to contradictory evidence, while probability judgements seemed to normalise in remission. No deficits in cognitive inhibition were found with either active or remitted delusions. The deluded group were excessively self-focused at both time points, but did not show a depressive attributional style. A small sub-sample, characterised by the ''bad-me'' type of paranoia (Trower & Chadwick, 1995), made more internal attributions for negative events than the ''poor-me'' paranoid group at baseline, but not at remission, suggesting that, unlike the JTC bias, attributional style fluctuates with delusional course. There were few relationships between measures on different tasks, suggesting that the different processes measured in this study are relatively independent. A different pattern of correlations on the 'beads' task was found in the

[7]The relationship between anxiety and conservative performance appears somewhat contradictory to the finding of Garety et al (1991) who noted a mixed group of anxious patients demonstrated quite a hasty reasoning style. However the population in the Garety study differed markedly from the present sample, since it consisted of mostly outpatients with specific phobias or GAD, unlike the present more severe inpatient group.

deluded group compared to the two control groups. In the latter groups, performance appeared to be mediated by confidence in one's decision making. No such relationship was found in the deluded patients, suggesting that their performance may be attributable to a different process, hypothesised to be the JTC bias. Similarly, few correlations were found between the tasks and the clinical measures. The only measures associated with delusion symptom scores were initial certainty and number of beads seen, both from the 'beads' task, providing further support for the centrality of the JTC bias in relation to delusions.

This body of results should be viewed within the context of the specific strengths and weaknesses of this study. To our knowledge this is the first study which has examined empirically different aspects of cognitive functioning in operationalised symptomatic and remitted states in the same individuals. First, this approach has allowed us to conclude that there are in fact few connections between different areas of dysfunction previously found in deluded patients. Our findings therefore substantiate the theoretical slant taken by Garety et al. (2001) of a multifactorial route to the formation and maintenance of the positive symptoms of psychosis. They also suggest that a large variability of performance can be found even within a single symptom such as delusions. Some authors have indeed emphasised the importance of identifying delusional content as a possible reflection of the cognitive processes underlying delusional formation and maintenance (Bentall et al., 2001; Trower & Chadwick, 1995). However, while much research has been devoted to persecutory delusions (Bentall et al., 2001; Freeman et al., 2002), other types of delusions have not been investigated. It is likely that delusions with a fantastic or bizarre content (such as having been replaced by a zombie or having a microchip inserted in one's brain) will reflect different types of cognitive abnormalities to those with a more social (e.g., delusions centering around "the position of the self in a social universe" (Bentall et al., 2001), namely persecutory and grandiose) or cultural (delusions reflecting themes which are culturally reinforced, such as religion, telepathy, reference (Peters, 2001)) content. Further research should therefore divide deluded groups into meaningful types or categories to determine whether delusion content is related to types of cognitive biases. We would predict that individuals with bizarre delusions would display the greatest reasoning and information processing biases, since they are furthest away from "normal" beliefs. In contrast, we would predict that social delusions would be more closely intertwined with emotional processes than other types.

Secondly, our approach has also permitted us to make some tentative differentiations between processes which are stable in deluded individuals over time, and potentially may act as a vulnerability towards delusional thinking, and those which appear to be a reflection of delusional content and/or be implicated in the maintenance of delusions (or what Nuechterlein (1987) has called stable vulnerability and transient episode markers). As pointed out by Birchwood (1999), Nuechterlein (1987), and others, the longitudinal dimension is a crucial one to disentangle, for both theoretical and therapeutic reasons. Most studies to date have been carried out on symptomatic participants, and to our knowledge this is the first study to demonstrate that the JTC bias is stable, while a depressive attributional style is more likely to reflect the content of delusions and fluctuate with delusional course. The present study was, however, limited to two time points only, and further work should be devoted to a more thorough longitudinal approach following individuals from the at-risk period, through the acute period, to remission. It would also be valuable to compare the longitudinal course of cognitive biases of those individuals who display good recovery, and those who remain plagued with residual symptoms.

A further strength of this study consists of the research strategy of selecting individuals by virtue of their delusional, rather than diagnostic, status, which has allowed us to make direct links between cognitive factors and the presence and absence of delusions specifically, rather than being limited to post hoc analyses with symptom measures in a heterogeneous sample. One of the limitations of the study, however, regards our inability to compare deluded individuals diagnosed with schizophrenia with those diagnosed with other disorders because of small numbers. Further research will be needed to clarify whether our findings have diagnostic specificity.

Small numbers in the three groups caused by the attrition of participants at follow-up is a clear limitation of this study. This is particularly the case for the post hoc comparisons between "poor-me" and "bad-me" paranoids, which did not find significant changes over time, despite different findings being obtained at baseline and follow-up. It is also possible that the lack of difference between the groups on Condition 1 of the Beads task at follow-up was due to a lack of power; indeed the raw means indicated that the deluded group asked for fewer beads than the other two groups. A greater sample size showing significant differences at both time points would have enabled us to assert more conclusively that the JTC bias remains stable over time. The replication of these findings with a greater sample size would address this issue, and enable the comparisons of different types of delusions, as suggested above.

Another limitation concerns the demographic differences between our three groups. Although the use of an in-patient psychiatric group provided a control for crucial factors such as hospitalisation, use of medication, and non-specific psychiatric deficits, the two psychiatric groups were not matched in age or gender at both time points, and there was a significant difference in IQ between the two groups in the reduced sample at follow-up. The non-clinical group also had higher IQ than both psychiatric groups at both time points, although they did not differ in age to the deluded group. Unfortunately such discrepancies between groups is often a result of selecting representative populations, since psychotic patients hospitalised in acute inner London psychiatric wards tend to be young, male, and of lower IQ than the general population. Although the matching of groups in demographic variables is desirable, it often occurs at the expense of ecological validity. While we recognise that adjusting for demographic differences between the groups statistically is a weaker scientific option, nevertheless it allows us to base our conclusions on a representative selection of patients.

The issue of ecological validity is particularly pertinent since the present findings are likely to have clinical implications. A number of randomised controlled trials (RCTs) have demonstrated the efficacy of cognitive-behaviour therapy (CBT) with individuals with psychosis (see Pilling et al., 2002, for a meta-analysis), but to date only a handful of studies have investigated the specific ingredients of change or reliable predictors of outcome (Garety et al., 1997; in press). The present results would concur with Garety et al.'s (in press) findings which suggest that the assessment and modification of information processing style, specifically the JTC bias, may be just as crucial as addressing the content of delusions to prevent relapse, as has recently been shown in chronic depression (Teasdale et al., 2001). This would also echo other reports that it is not *what* you believe, but *how* you believe it, which predicts psychopathology (Peters, Day, McKenna, & Orbach, 1999a; Peters, Joseph, & Garety, 1999b). In contrast, the identification of negative self-representations in specific sub-types of paranoia, would suggest that the content of delusions could act as a guide as to whether the further exploration of schemas is likely to be necessary to

achieve good therapeutic outcome. These suggestions are testable hypotheses which can be addressed either in RCTs of CBT for psychosis, or in single-case study designs.

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