Confidence in Errors as a Possible Basis for Delusions in Schizophrenia

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Abstract: In two previous studies, it was observed that schizophrenic patients display increased confidence in memory errors compared with controls. The patient group displayed an increased proportion of errors in their knowledge system, quantified as the percentage of high-confident responses that are errors. The latter phenomenon has been termed *knowledge corruption* and is put forward as a risk factor for the emergence of delusions. In the present study, knowledge corruption was analyzed separately for different aspects of memory errors. A source-monitoring task was used, for which participants (30 schizophrenic patients with past or current paranoid ideas and 15 healthy controls) were asked to provide associates for each of 20 prime words. Later, participants were required to recognize studied words among distractor words, judge the original source, and provide a confidence rating for the most recent decision. Schizophrenic patients displayed greater confidence in memory errors compared with controls. Knowledge corruption was observed to be significantly greater in schizophrenic patients relative to controls for false-positive and false-negative judgments. It is proposed that reliance on false knowledge represents a candidate mechanism for the emergence of fixed false beliefs (i.e., delusions).

Key Words: Schizophrenia, memory, knowledge corruption, delusions.

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Delusions are perhaps the most prominent feature of schizophrenia. Jaspers (1973) has described three core clinical characteristics of delusions that have remained essential for all current definitions of delusions: certainty, imperviousness (incorrigibility), and impossibility or falsity of content (Sedler, 1995; Spitzer, 1990). Whereas consensus is readily obtained among clinicians for estimating the certainty and imperviousness of a belief, diagnostic problems exist with respect to the third criterion. For example, different cultures and subcultures vary in their views of normality (Al-Issa, 1977; Murphy, 1967), which may lead to an overestimation of psychotic disorders in some migrant populations (Haasen et al., 2000). Furthermore, many delusions (*e.g.*, one's activities are being monitored by the secret service), although highly improbable, often cannot be completely disproved (Spitzer, 1989). To infer presence of delusional ideation, the clinician frequently adopts simple heuristics such as exploring additional psychiatric symptoms (*e.g.*, hallucinations or formal thought disorder), consistent reoccurrence of suspiciousness after stressful life events, and/or incongruence between belief and behavior. To illustrate, if a person claims to be the son of God but engages in criminal activities and shows additional patterns of positive or disorganized symptomatology, this is a clear indication (but not proof) that the claim is delusional. A fourth core feature of primary delusions (*i.e.*, delusions proper) according to Jaspers is that the origin of the belief must defy understanding (Walker, 1991). Beliefs that fulfill the three mentioned core criteria for delusions but emerge understandably from, for example, strong affects, hallucinations, or the person's personality are judged either as delusion-like or overvalued ideas according to Jaspers.

To date, rivaling cognitive hypotheses exist on how fixed, false beliefs emerge. Among the most promising approaches are the jumping to conclusions hypothesis (Garety and Freeman, 1999; Huq et al., 1988), the attributional styles hypothesis (*e.g.*, Bentall, 1994), and the perceptual abnormality hypothesis (Maher, 1974, 1999). These theories can convincingly account for some aspects of delusional thinking but have limited explanatory power for the entire range of paranoid/delusional behavior (Garety and Freeman, 1999). This underscores the possibility that delusions may stem from multiple pathogenetic causes.

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We have recently proposed an additional cognitive dysfunction that might underlie the establishment of fixed, false beliefs (Moritz and Woodward, 2002; Moritz et al., 2003). The basic assumption is that schizophrenic patients with past or present paranoid ideas share a significant discrepancy between objective and subjective reliability of information stored in memory. We have claimed that paranoid schizophrenic patients are poor at disentangling valid from invalid memory information and display a stronger tendency to trust incorrect memory information, whereas normal participants are more cautious in their evaluation of incorrect memory information. Overconfidence in errors in schizophrenia is thought to arise from the impaired ability to cast doubt on fallible information, whereas healthy subjects are able to attach so-called *not trustworthy* tags to such mental representations. In the initial studies (Moritz and Woodward, 2002; Moritz et al., 2003) a source-monitoring task was used, which required participants to provide associates for each of 20 prime words. Later, participants were instructed to recognize studied words among distractor words, to recall the source if words were classified as having been studied, and to rate their confidence in the source judgment. It became evident that the number of high-confident false memory responses (subjective knowledge but invalid) expressed as a percentage of the overall number of high-confident memory responses (subjective knowledge, both valid and invalid) was increased in schizophrenic patients relative to healthy controls. Using the definition of *knowledge* as statements or hypotheses held as accurate with high confidence (Hemsley and Garety, 1986, p. 52), this increased presence of inaccurate memories held with high confidence is referred to as *knowledge corruption*.

It is proposed that knowledge corruption predisposes subjects to reality distortion because perception, evaluative inferences, and problem solving all heavily rely on previous knowledge (Frith and Dolan, 1997; Stapel and Koomen, 2000). When corrupted, the knowledge framework may not provide an adequate basis for reality assessment and for navigation in the external environment because it offers incorrect interpretations of current input.

Experimental studies have demonstrated that knowledge corruption can also be induced in healthy participants by task manipulations such as the Deese-Roediger-McDermott paradigm (Roediger and McDermott, 1995; Roediger et al., 2001). However, the knowledge system of healthy individuals is largely reliable and represents a consensually experienced picture of the world (Reisberg, 2001, p. 211).

It needs to be emphasized that excessive knowledge corruption in paranoid schizophrenia cannot be regarded as a mere consequence of decreased memory performance alone. To illustrate, a person who has severe problems encoding and recalling information but is cognizant of this problem, and therefore mistrusts the validity of his or her memory responses, would not suffer from knowledge corruption because conviction for incorrect (pseudo-) memories is central to the concept. Although memory performance is severely disturbed in schizophrenia (Aleman et al., 1999; Moritz et al., 2001), this cannot satisfactorily explain the emergence of positive schizophrenic symptoms, because other neurological and psychiatric groups such as demented patients (Heaton et al., 1994) are even more disturbed in this domain but do not necessarily display delusions.

In the second study (Moritz and Woodward, 2002), it was demonstrated that patients with schizophrenia committed more recognition errors and displayed greater confidence in these errors compared with healthy controls. At the same time, patients were somewhat underconfident in responses that were in fact correct. Thus, knowledge corruption in schizophrenia stems from two sources: more memory errors and a decreased ability to cast doubt on such errors. Substantial aspects of this theory await further empirical testing. Therefore, at present, our claim should be considered a working hypothesis and a framework for further research.

AIMS OF THE STUDY

For the present study, we investigated whether knowledge corruption in schizophrenia is confined to specific aspects of the memory system or reflects a more generalized memory dysfunction. In previous studies (Moritz and Woodward, 2002; Moritz et al., 2003), knowledge corruption scores were collapsed across different types of memory errors. This was done because there were insufficient response trials for some memory conditions. Therefore, the question of whether knowledge corruption in schizophrenia is present for only particular types of memory errors has remained unresolved. We analyzed knowledge corruption separately for the following types of memory errors: false-negative errors (*i.e.*, nonrecognition of a word which was previously heard or said), false-positive errors (*i.e.*, recognition of a word not previously heard or said), and source attribution errors (*e.g.*, misattribution of a self-generated word to another person). It was expected that knowledge corruption would occur for all aspects of memory.

METHODS

Participants

Thirty schizophrenic inpatients from a psychiatric long-term institution participated in the study. Patients displayed paranoid ideas either at the time of assessment or at admission. Diagnoses relied on DSM-IV criteria and were determined by a registered psychiatrist. Exclusion criteria were an Axis I diagnosis other than schizophrenia, severe substance abuse, any form of documented or suspected brain damage, or diabetes. All patients received atypical neuroleptic medication at the time of testing. Sociodemographic and psycho-

pathological characteristics of the sample are displayed in Table 1.

Psychopathological symptoms were assessed using the Signs and Symptoms of Psychotic Illness (SSPI) rating scale (Liddle et al., 2002). The reality distortion syndrome comprised the symptoms of delusions and hallucinations. The psychomotor poverty syndrome was composed from items tapping underactivity, flattened affect, anhedonia, and poverty of speech. The disorganized syndrome included scores for inappropriate affect and formal thought disorder. At the time of cognitive assessment, 23 patients displayed at least mild but definite delusional ideas as assessed with the SSPI delusion item.

Seventeen controls were drawn from hospital staff and the general population. Control participants were screened for brain damage and mental illness. Two participants were excluded because of severe concussion sustained in the past followed by unconsciousness for at least 30 seconds.

None of the participants had participated in either of the two earlier studies that used a related paradigm. Participants were paid CND$5 for participation per session, which also involved other experimental tasks. Written informed consent was obtained after the procedures had been fully explained to the participant. The study was approved by the local ethics committee.

Procedure

The experimental procedure contained a learning and a recognition trial. In the learning phase, the experimenter read 20 words drawn from the Kent-Rosanoff association test to the participant. These words were identical to the stimuli administered in the two previous studies. After each prime, the participant had to provide a close associate (always single words, no names, no repetition of previous words). Subsequent to the last item, participants were told that they later had to recognize the studied items (*i.e.*, generated by either experimenter or participant). The recognition phase started 10 minutes after the learning phase had been terminated.

In the recognition phase, a list of 80 words was read to the participant. Four different stimuli types were used (presented in random order):

(1) Twenty words from the Kent-Rosanoff association test (experimenter-generated words)

(2) Twenty associates produced by the participant in response to 1 (self-generated words)

(3) Twenty new words with no associative relation to words from conditions 1 or 2

(4) Twenty new words that were related but never identical to words from 1 or 2 (*e.g.*, if the prime word was *bread* and the participant associated the word *butter,* the related new word could be *cheese*). There were always 2 item options for each new-related stimulus in case the participants' association (*i.e.*, condition 2) matched the first item option. The related new words were highly frequent associations to the prime words according to the German translation and norm study of the Kent-Rosanoff association test (Russell, 1970).

After each presented word, the participant was instructed to provide three responses:

(1) Recognition: new or studied word.

(2) Source attribution: If the word was recognized as being studied, it had to be determined who had produced it: the experimenter or the participant.

(3) Memory confidence: Participants were instructed to rate on a 4-point scale how confident they were concerning the origin of the information (experimenter, participant or new; 1 = don't know; 2 = rather uncertain; 3 = rather certain; 4 = convinced). No feedback on whether responses were correct or incorrect was given.

The present experiment contained one important modification in comparison with the forerunner experiments. The two previous studies presented only 20 distractor items, thereby producing floor effects in many healthy participants. Equation of trials across conditions allowed us to avoid floor effects and to assess knowledge corruption directly for all

TABLE 1. Sociodemographic and Psychopathological Characteristics of the Samples: Means and SDs

Variable	Healthy participants ($N = 15$)	Schizophrenic participants ($N = 30$)	Statistics
Age, year 5	37.67 (12.47)	37.30 (10.16)	$t(43) = 0.11$; $p > .9$
Sex (male/female)	8/7	19/11	$\chi^2(1) = .42$; $p > .6$
Premorbid intelligence (NART IQ)	110.64 (6.79)	102.01 (9.04)	$t(43) = 3.26$; $p = .002$
Number previous hospitalizations	—	7.03 (3.36)	—
Length of illness, years	—	16.95 (8.86)	—
Chlorpromazine equivalent dosage, mg	—	671.60 (494.40)	—
SSPI total	—	17.33 (7.35)	—

conditions (experimenter-generated items, self-generated items, new items).

Strategy of Data Analyses

As for the previous studies, the main dependent variable was the number of responses for which participants gave the highest confidence ratings (*i.e.*, confidence rating score = 4). Two main indices, which are related but provide nonredundant information, were calculated. First, we computed the percentage of occasions on which participants rated responses as high in confidence (the confidence in responses index). This was computed separately for correct and incorrect responses. Second, we computed the percentage of high-confident responses that were errors (the knowledge corruption index). The latter index, as mentioned, measures the degree to which false information intrudes into the knowledge system (*i.e.*, what one believes to be factual). If participants made no high-confident responses at all, the knowledge corruption index was set to zero.

RESULTS

Sociodemographic Characteristics and Error Analysis

As can be seen in Table 1, the samples did not differ regarding either gender distribution or age. However, patients had significantly lower intelligence scores. Most patients were chronic, with an average of seven previous hospitalizations. Table 2 displays memory errors for schizophrenic patients and controls across all four conditions. Misattribution errors (*e.g.*, a self-generated response is misattributed to the experimenter) occurred comparably often in both samples. For both studied conditions (original source: experimenter or self), patients significantly more often judged items as new. For the new-unrelated condition, patients showed a significant bias to judge never-presented words as experimenter-generated.

Confidence in Responses

The following analyses were conducted to inspect whether schizophrenic patients showed greater confidence in erroneous responses than controls. A two-way mixed ANOVA was calculated with response type (correct, incorrect) as the within-subject variable and group (schizophrenic, control) as the between-subject variable. The number of high-confident responses divided by the overall number of responses per condition served as the dependent variable (the confidence in responses index). Because of low error rates in the new conditions (leading to a division by zero in the confidence in responses index), the related and unrelated new items were collapsed to a single condition. The effect of error type achieved significance for all analyses ($p < .001$), showing that for all participants, confidence was far greater when a response turned out to be correct versus incorrect. Put differently, all participants mistrusted incorrect responses to a greater degree than correct responses. The group effect failed to reach significance ($p > .05$) in all analyses when correct and incorrect responses were combined, meaning that patients and controls gave confident ratings to a comparable extent. The interaction of group X response type achieved significance for the new condition ($F = 5.69$; $p = .02$) and the experimenter-generated condition ($F = 10.30$; $p = .003$). The interaction of the self-generated condition ($F = 0.00$; $p > .9$) failed to reach significance. Except for self-generated items, there was a tendency for schizophrenic participants to display a lower percentage of confident responses when the response was actually correct (a trend, however, emerged for experimenter-generated words, with schizophrenic patients being somewhat underconfident for correct responses) and a

TABLE 2. Means and SDs of Source Monitoring and Recognition Errors (*t*-Test Differences Displayed in the Cells of the Schizophrenic Participants)[a]

	Number of responses, original source			
Group and attributed source	**Experimenter, mean (SD)**	**Self, mean (SD)**	**New-related, mean (SD)**	**New-unrelated, mean (SD)**
Healthy participants				
Experimenter	17.20 (2.54)	3.67 (5.00)	1.67 (2.09)	0.33 (1.05)
Self	1.53 (2.00)	14.06 (5.31)	0.46 (1.13)	0.27 (0.59)
New	1.27 (1.39)	2.27 (2.09)	17.87 (2.83)	19.40 (1.59)
Schizophrenic participants				
Experimenter	12.90 (4.57)***	3.77 (3.52)	3.13 (4.55)	2.77 (4.42)**
Self	1.90 (2.87)	11.06 (4.56)*	0.60 (0.97)	0.40 (0.77)
New	5.20 (3.71)***	5.17 (3.88)***	16.27 (4.78)	16.83 (4.87)***

[a]Underlined values designate correct source identification of previously presented words; all other values represent memory errors. *$p \leq .1$; **$p \leq .01$; ***$p \leq .005$, two-tailed.

higher percentage of confident responses when the response was incorrect compared with controls (at least $p < .05$; Figure 1).

Knowledge Corruption

In our final set of analyses, we determined the extent of knowledge corruption in both samples (Table 3). A two-way mixed ANOVA was calculated with response type (correct, incorrect) as the within-subject variable and group (schizophrenic, control) as the between-subject variable. The number of high-confident errors divided by the overall number of high confidence responses (the knowledge corruption index) served as the dependent variable. Significant knowledge corruption was detected for all conditions except for source memory errors (*i.e.*, self-generated but judged as experimenter-generated and vice versa) and misattribution of a new word to the self. Knowledge corruption was stronger for false-negative responses ($p < .001$), but a significant difference was also measured for false-positive responses ($p = .03$).

Comparing Table 3 with Table 2, it is apparent that knowledge corruption was revealed at least in all those conditions in which patients produced more errors than controls. Importantly, patients not only made more errors but also were more confident that their erroneous responses were correct (Figure 1). As a result, group differences in knowledge corruption were stronger than would be expected from solely analyzing accuracy scores.

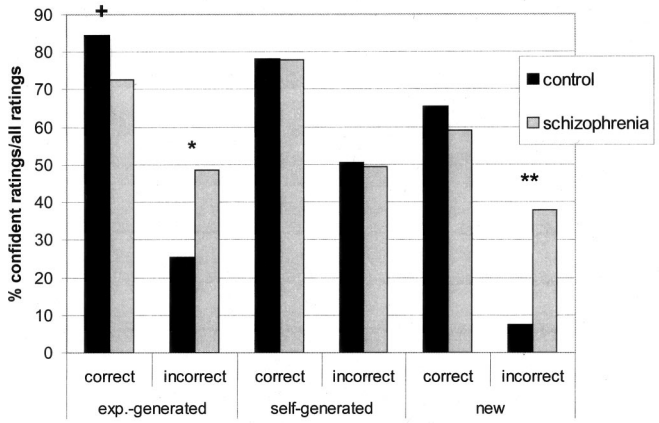

FIGURE 1. Confidence in responses index ([# high-confident responses/all responses] * 100) separated by response type (correct, incorrect). Except for the self-generated condition, the interaction of group by response type achieved significance. Because of low levels of error rates, the related and unrelated new conditions were collapsed to one condition. $+p = .1$; $*p < .05$; $**p < .001$; two-tailed independent samples *t*-tests.

Relationship Between Knowledge Corruption With IQ, Psychopathology, and Medication

All correlations between the severity of hallucinations and the dependent variables were not significant. Specifically, there was no correlation ($r = .05$; NS) with the tendency to misattribute self-generated items to the experimenter. Delusions were correlated with a greater percentage of high-confident incorrect responses relative to the overall number of incorrect false-negative responses (*i.e.*, the confidence in responses index; $r = .41$; $p = .02$) but were not correlated with any knowledge corruption parameter. Positive symptomatology did not correlate with any parameter. Formal thought disorder was associated with a tendency to judge new-related information as studied ($r = .43$; $p = .03$) and specifically to attribute new-related information to the experimenter ($r = .36$; $p = .05$). Neuroleptic dosage did not significantly correlate with any knowledge corruption index. However, a significant negative correlation emerged between neuroleptic dosage and number of (correct and incorrect) high-confident ratings ($r = –.47$; $p = .009$). No correlation emerged between intelligence and any of the parameters. Intelligence was correlated with neither knowledge corruption nor confidence in errors ($p > .2$).

DISCUSSION

For the third time, data have been collected in line with the hypothesis that patients diagnosed with schizophrenia put more trust in false memory responses than controls. This result strengthens the claim that schizophrenic participants, unlike controls, have difficulties attaching not trustworthy tags to erroneous responses (Moritz and Woodward, 2002), leading to the observed pattern of overconfidence in incorrect responses. As outlined in the introduction and the Methods section, the combination of increased error rates with overconfidence in errors leads to excessive knowledge corruption in schizophrenia. In the present study, knowledge corruption in controls occurred on 9% of responses, whereas the corresponding rate for schizophrenic patients was approximately 21%. This phenomenon was not moderated by severity of delusions nor any other syndromatic patterns. Patients were significantly more often highly confident for incorrect memories than controls.

Although it may be expected that a cognitive deficit underlying the formation of delusions would correlate with the severity of delusions, it can also be argued that knowledge corruption is a trait marker of schizophrenic delusions that persists over the different stages of the disease and is thus observable even in patients with a past history of delusions. So far, we have demonstrated knowledge corruption in schizophrenic patients relative to controls. Although there is at least some indirect evidence that other psychiatric groups—like OCD patients (MacDonald et al., 2001; Zitterl

TABLE 3. Degree of Knowledge Corruption in Patients and Controls (Cell Entries Are % of All High-Confident Responses That Are Errors, SD in Parentheses)

Item type	Healthy participants	Schizophrenic participants	Statistics (2-tailed), $df = 43$
1. EG items misjudged as SG	1.47 (2.35)	1.57 (2.52)	$t = 0.13$; $p > .8$
2. EG items misjudged as new	0.45 (1.02)	4.48 (4.80)	$t = 4.40$; $p < .001$
3. SG items misjudged as EG	4.27 (8.62)	3.86 (4.49)	$t = 0.21$; $p > .8$
4. SG items misjudged as new	1.37 (1.61)	4.55 (4.48)	$t = 3.46$; $p = .001$
5. New-related items misjudged as SG	0.18 (0.72)	0.48 (0.94)	$t = 1.17$; $p > .2$
6. New-related items misjudged as EG	0.50 (1.49)	2.68 (5.27)	$t = 2.10$; $p = .04$
7. New-unrelated items misjudged as SG	0.00 (.00)	0.23 (.71)	$t = 1.75$; $p = .09$
8. New-unrelated items misjudged as EG	0.31 (.84)	3.04 (7.00)	$t = 2.11$; $p = .04$
Summary statistics			
All EG items (1+2)[a]	1.92 (2.56)	6.05 (5.83)	$t = 3.29$; $p = .002$
All SG items (3+4)	5.64 (8.40)	8.41 (6.02)	$t = 1.27$; $p > .2$
All new-related items (5+6)	0.69 (2.18)	3.16 (5.40)	$t = 2.18$; $p = .035$
All new-unrelated items (7+8)	0.31 (0.84)	3.27 (7.35)	$t = 2.17$; $p = .038$
False-positive responses (sum 5–8)	1.00 (2.88)	6.43 (12.47)	$t = 2.23$; $p = .03$
False-negative responses (2+4)	1.82 (2.07)	9.03 (8.06)	$t = 4.60$; $p < .001$
Source attribution errors (1+3)	5.74 (9.17)	5.43 (5.69)	$t = 0.14$; $p > .8$
Total knowledge corruption	8.56 (10.49)	20.89 (18.56)	$t = 2.84$; $p = .007$

EG, experimenter-generated; SG, self-generated.
[a]The numbers in brackets designate sums of scores, *e.g.*, 1+2 means the sum of EG items misjudged as SG (*i.e.*, 1) and EG items misjudged as new (*i.e.*, 2).

et al., 1999)—may show normal or sometimes even largely decreased confidence in their memory, this hypothesis needs explicit empirical testing.

Unlike our previous work (Moritz and Woodward, 2002; Moritz et al., 2003), the present study addressed whether knowledge corruption affects different types of memory errors to the same degree or whether it is confined to special types of errors (false-positive, false-negative, source memory). Our results again show that, overall, patients display greater knowledge corruption than controls. Specifically, patients showed a tendency to misjudge new information as externally generated and to judge studied information as new; knowledge corruption was significantly greater in patients for both studied and new items. In a recent study conducted with the false memory paradigm, greater knowledge corruption was observed for false-negative responses in patients, whereas the difference for false-positive responses achieved trend level when baseline performance was controlled for (Moritz et al., 2004).

Inaccurate knowledge may trigger a distortion in the interaction with reality because the way we perceive and judge our environment is through an interaction of current input with previous knowledge. To illustrate, if person A is convinced that person B owes her $1,000 although the correct amount was $10, this can have serious consequences (false-positive knowledge corruption). Similarly, if you are absolutely positive that your best friend did not invite you to his party (although he actually did), this may pose a serious threat to your friendship (false-negative knowledge corruption). Whereas both false-positive and false-negative memory errors occur in healthy people, our research shows that for most incorrect judgments, healthy persons are not convinced that they are true. Casting doubt on judgments is thus a powerful adaptive mechanism and enables a person to perform further reality checks.

Presently, we are attempting to replicate this finding and to determine factors that moderate knowledge corruption in schizophrenia, because not all memory components seem to be affected. In particular, it seems that schizophrenic patients do not display knowledge corruption for self-generated items. Such items probably generate a number of distinct retrieval cues (*e.g.*, remembering the sound of one's own voice while speaking) that set self-generated apart from other memory material. The data suggest that these strong cues are accessible even to schizophrenic patients.

In line with our previous study (Moritz et al., 2003), schizophrenic patients with symptoms of formal thought disorder were biased to report that new-related information (*i.e.*, new words that were associated with both prime words and self-generated associates) were old (recognition error). This result can be interpreted as further evidence for the hyperpriming theory of formal thought disorder (*e.g.*, Moritz et al., 2003; Spitzer, 1989). That is to say, the activation elicited by the studied items preactivates related and even

remote associations in these patients more than in controls. The greater than normal activation of related items thereby surpasses the recognition threshold, producing the illusion that these words have actually been heard.

Future research is needed to explore the effects of medication on knowledge corruption. Whereas some patients continue to experience full-blown or attenuated paranoid ideas under neuroleptics, there is extensive evidence that neuroleptic agents are powerful in reducing existent delusions and in preventing further episodes (*e.g.*, Naber et al., 2002). Therefore, when a patient is guarded against the presence or emergence of delusions or hallucinations with the administration of neuroleptic agents, knowledge corruption should be less pronounced if it underlies delusions. An ameliorating effect of higher neuroleptic doses on knowledge corruption was found in one study (Moritz et al., 2003), but this could not be confirmed in another investigation (Moritz and Woodward, 2002). The present study found that higher neuroleptic doses made participants generally less confident in their judgments. This inconclusive pattern of results may have occurred because medication was not manipulated independently. Participants were medicated by their psychiatrists according to clinical judgment, and all patients in this study were on antipsychotic medication. Thus, severity of psychopathological symptoms and medication are confounded. To shed light on this issue, analyses with drug-naive patients versus medicated patients may be especially instructive.

Furthermore, it remains to be determined empirically whether delusional patients not diagnosed with schizophrenia also express excessive knowledge corruption, and whether schizophrenic patients with no clear systematized past or present delusions are free of this deficit. As long as the disturbance is found in many schizophrenic patients of all subtypes, it cannot be ruled out that knowledge corruption is a causal factor for symptoms other than delusions. As outlined, the main claim that knowledge corruption underlies schizophrenic delusions is still speculative at this point. We are currently performing a study on unmedicated first-episode schizophrenic subjects and psychometric high-risk subjects (*i.e.*, subjects scoring high on measures of psychosis-proneness/schizotypy) to explore whether knowledge corruption is a predisposition rather than a consequence of schizophrenia. In disagreement with the view that knowledge corruption is a mere consequence of the illness, there was no relationship to symptomatology and illness duration. Investigation of unaffected first-degree relatives may be another important avenue to investigate the vulnerability status of knowledge corruption.

The present findings complement previous research on metamemory processes in schizophrenia. There is evidence (Danion et al., 1999; Huron et al., 1995) that patients with schizophrenia have difficulty recalling memory episodes vividly, reflected by fewer remember responses relative to controls in a memory paradigm that asked patients to judge whether participants only knew, or clearly remembered that an item had been previously presented. The association between remember-know and confidence ratings in schizophrenia remains to be established. In healthy participants, high-confidence judgments are typically accompanied by conscious recollections (*i.e.*, remember responses) of previous episodes (Yonelinas, 2001). Patients with schizophrenia, in contrast, may have a more lax criterion for high memory confidence. Feelings of knowing may be judged by schizophrenic patients as sufficient evidence that an event has actually occurred. Such an approach would be maladaptive, because remember experiences are better discriminators for true memories than know experiences (Reisberg, 2001). In this respect, overconfidence in memory errors may be a special case of the jumping to conclusions bias typically exhibited by schizophrenic patients in probabilistic reasoning tasks (Garety et al., 1991) whereby firm decisions are reached on the basis of little evidence.

For future research, it seems crucial to explore the possible neuronal underpinnings of metacognitive deficits in schizophrenia. It seems particularly worthwhile to pursue the hypothesis that response confidence is related to error-related cortical activity, which is typically associated with the anterior cingulate cortex (Alain et al., 2002). A number of recent studies have presented physiological evidence that the neural system involved in processing errors exhibits distinct patterns of activation after correct versus incorrect responses in healthy controls, but shows similar patterns after correct versus incorrect responses for schizophrenic patients (Bates et al., 2002; Laurens et al., 2003; Mathalon et al., 2002). One study has demonstrated that this reduced distinction between errors and correct responses is enhanced in paranoid patients (Mathalon et al., 2002). In other words, the neural systems of patients with schizophrenia appear to be less sensitive to errors relative to controls. These physiological studies corroborate our finding of a reduced behavioral distinction between correct and incorrect responses for schizophrenic patients.

Apart from factors that may predispose a subject to delusions, we are currently investigating characteristics that differentiate currently deluded schizophrenic patients from schizophrenic patients without delusional ideas. In a recent study, we presented participants with pictured scenes that were displayed successively. Participants were asked to rate the plausibility of interpretations at each stage of the sequence. We demonstrated that patients with current delusions show a bias against disconfirmatory evidence (Woodward et al., 2004). Interestingly, currently deluded but not nondeluded schizophrenic patients tended to persist with their initial interpretations, even when these were no longer supported by subsequent evidence. Thus, a predisposition to delusions (*e.g.*, knowledge corruption, attributional style) may become symptomatic when subjects are no longer able to correct their assessments in light of contradicting evidence.

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