Reasoning Anomalies Associated With Delusions in Schizophrenia

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Deluded people differ from nondeluded controls on attributional style questionnaires and probabilistic-reasoning and theory-of-mind (ToM) tasks. No study to date has examined the relations between these 3 reasoning anomalies in the same individuals so as to evaluate their functional independence and potentially inform theories of delusion formation. We did so in 35 schizophrenic patients with a history of delusions, 30 of whom were currently deluded, and 34 healthy controls. Compared with healthy controls, patients showed (a) a jumping-to-conclusions bias and a bias to overadjust when confronted with a change of evidence on probabilistic-reasoning tasks, (b) an excessive externalizing attributional bias, and (c) performance deficits on 3 ToM tasks. Probabilistic-reasoning and ToM measures correlated, while attributional-bias scores were independent of other task measures. A general proneness to delusional ideation correlated with probabilistic-reasoning and ToM measures, while externalizing bias was unrelated to the study measures of delusional ideation. Personalizing bias associated specifically with paranoia across the clinical and nonclinical participants. Findings are consistent with a common underlying mechanism in schizophrenia which contributes to the anomalies on probabilistic-reasoning and ToM tasks associated with delusions. We speculate that this mechanism is impairment of the normal capacity to inhibit “perceived reality” (the evidence of our senses), a capacity that evolved as part of the “social brain” to facilitate intersubjective communication within a shared reality.

Key words: psychosis/reality distortion/social cognition/belief

Introduction

Delusions resist definition according to a set of necessary and sufficient criteria.1–3 Yet these symptoms are a clinical reality, readily recognizable in most instances, and of particular importance in psychotic conditions like schizophrenia. The characteristic features that help define the presence of delusions are incorrigibility and incomprehensibility; delusions are espoused with a degree of conviction that is unwarranted by the evidence to hand and resist revision even when the delusional content is fantastic. A traditional psychiatric approach to incorrigible, incomprehensible delusions is to conceive of these as nonbelief-like statements or kinds of judgments that spring into existence without a meaningful context4; primary delusions, in particular, are described historically as psychologically un-understandable. An alternative approach is to conceive of delusions as resulting from breakdowns in the normal cognitive (ie, mental-processing) system for belief generation and evaluation; this is the cognitive neuropsychological approach.5 On this latter approach, delusions count as beliefs, albeit abnormal beliefs, and can be understood “psychologically,” albeit sometimes only in terms of cognitive-psychological processes that are not directly accessible to consciousness.

The “2-factor theory” developed from the application of cognitive neuropsychology to the study of delusions5–8 proposes that 2 distinct types of cognitive disturbance contribute, in combination, to the generation and adoption of a delusion. The first disturbance explains why an implausible thought with a particular content comes to mind in the first place, thus accounting for the delusional theme. The second disturbance explains why the implausible thought is accepted uncritically as true.

Recent advances in the cognitive sciences have highlighted a number of cognitive disturbances that contribute to the generation of specific delusional themes. Capgras delusion, eg, the delusional belief that a loved one has been replaced by an identical looking impostor, arises when intact visual processing of a familiar face combines with a loss of the normal autonomic (measured using skin conductance responses) sense of familiarity that ought to be triggered by the sight of that face.9–11 This disconnection generates the implausible thought that the person in front of one, despite claiming to be a loved one and recognized as such by other family members, is
a stranger whose face merely resembles that of the loved one. Frith, Blakemore, and colleagues have provided evidence that alien control thoughts come to mind when the internal monitoring of intended actions is disrupted such that actions are experienced with no accompanying sense of agency. A further example is implausible referential ideas, which, several authors have suggested, arise when heightened salience is assigned inappropriately to mundane events in the environment.

Such disruptions contribute to the understanding of delusions but fall short of a full account because they fail to explain the uncritical adoption of the implausible thoughts (generated by the first cognitive disturbance) as true. People with depersonalization disorder, eg, might think it is as if others are controlling their actions but do not believe this to be true. Likewise, people who experience déjá vu might feel as if they have been in a particular situation before but know this cannot be so. Recent reviews have highlighted 3 reasoning anomalies that might contribute to the uncritical adoption of implausible thoughts in deluded people: (a) a jumping-to-conclusions (JTC) bias on probabilistic-reasoning tasks, (b) extreme causal attributional biases, and (c) difficulties with appreciating other people’s perspectives on theory-of-mind (ToM) tasks.

Garety and colleagues pioneered the work on JTC and delusions. In a typical paradigm, participants are shown 2 jars of colored beads (in reverse ratios) and are asked to nominate from which jar a purportedly random series of beads is drawn. The common finding is that deluded people decide after fewer draws than non-deluded people; up to half of deluded participants might decide after 1–2 draws. Bentall and colleagues pioneered the work on attributional biases and delusions. Kaney and Bentall first used an attributional style questionnaire to show that persecutory-deluded people excessively externalize blame for negative events. Kinderman and Bentall later showed that these individuals also excessively blame other people rather than circumstances. Frith pioneered work on ToM and delusions; he proposed that a difficulty with making appropriate inferences of others’ thoughts and intentions (ie, a poor ToM) might explain persecutory and referential delusions. Numerous studies have since demonstrated pervasive ToM deficits in schizophrenia, although results are equivocal concerning a specific link with persecutory/referential delusions (see Brune and Langdon for reviews).

While theorizing about JTC has focused on the contribution of a liberal acceptance (or data-gathering) bias to delusion-proneness (irrespective of delusional theme), theorizing about the attributional-bias and ToM findings has focused, instead, on explaining specific delusional themes. The attributional-bias findings, eg, have been interpreted to support the defensive attribution model, people with specifically persecutory delusions subconsciously defend against activating latent-negative self-beliefs by externalizing blame. Because JTC purportedly associates with a general vulnerability to delusions, while attributional biases and ToM purportedly associate with specific delusional themes, it has been implicitly assumed that the 3 reasoning anomalies are quite distinct, indeed functionally independent. This assumption has not, however, been empirically evaluated.

Moreover, the attributional-bias and ToM anomalies might, just like the JTC bias, promote a general vulnerability to accept implausible thoughts as true (irrespective of delusional theme). Externalizing bias (EB) might, eg, be associated with a general failure to self-correct an initial implausible thought (“God is speaking to me”) with an internalizing attribution (“something wrong with my mind is causing me to hear voices”). Likewise, ToM impairment might reflect an egocentric difficulty with taking on, in imagination, other points of view, thus compromising general reality-testing. If all 3 reasoning anomalies promote a general vulnerability to delusions, as suggested, it seems plausible to hypothesize that they might also be related, and, if related, dependent upon a common underlying mechanism. In order to explore these possibilities and potentially inform theories of delusions, we examined probabilistic reasoning, attributional biases, and ToM in the same samples of people with schizophrenia and healthy controls and employed correlation techniques to examine the relationships of task measures with state and trait ratings of delusional thinking and paranoid ideation.

Methods

Participants

Thirty-five clinical participants took part. Of them, 30 had a Diagnostic and Statistical Manual of Mental Disorder, Fourth Edition (DSM-IV), diagnosis of schizophrenia and 5 were diagnosed with schizoaffective disorder. Patients were recruited from the New South Wales Area Health Services of the Sydney South West and the Hunter Areas and volunteer registers established by the Macquarie Centre for Cognitive Science and the Schizophrenia Research Institute of Australia. Diagnosis was confirmed using the Diagnostic Interview for Psychosis (DIP). The DIP revealed that all patients had a history of marked delusions, although not all patients were acutely delusional at the time of testing. Thirty were currently deluded and 15 had current delusions of moderate-to-severe levels (ie, all 15 were convinced of their delusions with variable impact on the patients’ daily preoccupations and activities). Persecution was the most common theme, being present in 23 of the patients, with loss of boundary delusions being the next most common, present in 17 of the patients. The persecutory delusions were moderate to severe in 10 of these 23 patients. Thirteen of the persecutory-deluded
patients also reported grandiose delusions, while 12 also reported delusions of reference. Among those patients with current nonpersecutory delusions, 5 patients reported loss of boundary delusions and 2 had delusions of guilt. Hallucinations were present in 19 of the patients. Table 1 summarizes the ranges and the mean severity levels of the clinical symptoms.

Thirty-four healthy controls were recruited from the general community and from among mature-age University students to match the patient group on years of age, gender ratio, and IQ estimated using the National Adult Reading Test (NART: see Table 1). Healthy controls were screened using the affective, psychotic, and substance abuse screening modules from the Structured Clinical Interview for DSM-IV Axis 1 Disorders (SCID-I).\(^3^4\) Exclusion criteria for both groups included past history of central nervous system disease or head injury, current substance abuse, and less than 8 years of formal education. All participants were English-speaking and gave written informed consent.

**Materials and Procedure**

**Baseline Measures.** IQ was assessed using the NART, while memory span was assessed using the Wechsler Adult Intelligence Scale-Revised Digits Forward and Backward subtests. The Wechsler Memory Scale-Revised Logical Memories subtests (LMI, LMIII) provided a measure of prose recall. The memory measures were included because previous work suggests reduced memory span contributes to a JTC bias\(^3^5\) and because the verbal eliciting stimuli used in traditional ToM tasks place heavy demands on prose recall.\(^3^6\)

**Probabilistic Reasoning.** There were 2 versions of the “beads task.”\(^1^9\) In the first “decide” version, participants were shown 2 jars of red/green beads in complementary ratios (85:15) and asked to decide from which jar a purportedly random sequence of beads was drawn (McKay, Langdon, Coltheart\(^3^7,3^8\) for details). The number of draws taken to reach a decision was recorded as was decision confidence rated using a 6-point Likert scale (from 50-50 unsure to 100% certain).

In the second “estimate” version, participants were again presented with 2 jars of beads, now blue/yellow, in similar ratios. For each consecutive draw of a total of 20 draws, they now indicated how certain they were the beads came from one or the other jar. This they did by marking a cross anywhere on a 10-cm rating scale ranging from “100% sure jar A” to “100% sure jar B.” The sequence of draws was in fact predetermined (as it was for the decide version); the first 10 trials were consistent with the beads being drawn from jar B, while the next 10 draws were consistent with the beads being drawn from jar A. All ratings were converted to scores ranging from 0 to 100 (indicating certainty in jar B) and ratings at trials 1, 10, and 20 were analyzed. We also calculated “shift in certainty”: the average change in certainty whenever the bead changed color. This was to capture the effect that many patients “overadjusted,” an effect observed previously, particularly in deluded schizophrenic patients\(^2^1,4^0\) (see figure 1 for an illustration).

Three sequences of draws were taken from Garety et al\(^2^1\) and allocated to the decide version, the first 10 trials of the estimate version and the next 10 trials in

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**Table 1. Demographics and Levels of Depression, Delusion-Proneness, and Paranoia for Both Groups, as well as Clinical Demographics and Symptom Ratings for Patients**

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Healthy Controls</th>
<th>Significance Test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong></td>
<td>35</td>
<td>34</td>
<td>(\chi^2) = 0.97</td>
</tr>
<tr>
<td>Males:females</td>
<td>23:12</td>
<td>26:8</td>
<td>(t_{67} = 1.37)</td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td>35.9 ± 10.4 (18–59)</td>
<td>32.0 ± 12.9 (20–58)</td>
<td>(t_{67} = 2.86^{**})</td>
</tr>
<tr>
<td><strong>Depression</strong></td>
<td>7.9 ± 6.6 (0–28)</td>
<td>4.1 ± 4.0 (0–14)</td>
<td>(t_{67} = 3.82^{****})</td>
</tr>
<tr>
<td><strong>Delusion-proneness</strong></td>
<td>9.9 ± 4.3 (2–20)</td>
<td>6.6 ± 2.6 (3–12)</td>
<td>(t_{38} = 4.20^{****})</td>
</tr>
<tr>
<td><strong>Paranoia</strong></td>
<td>56.9 ± 15.7 (29–89)</td>
<td>43.0 ± 11.3 (24–66)</td>
<td></td>
</tr>
<tr>
<td><strong>Clinical demographics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age of illness onset (years)</td>
<td>22.8 ± 6.8 (15–42)</td>
<td>43.0 ± 11.3 (24–66)</td>
<td></td>
</tr>
<tr>
<td>Duration of illness (years)</td>
<td>12.6 ± 9.1 (1.5–41)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Clinical symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delusions</td>
<td>2.4 ± 1.6 (0–5)</td>
<td></td>
<td></td>
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<tr>
<td>Persecutory delusions</td>
<td>1.7 ± 1.6 (0–5)</td>
<td></td>
<td></td>
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<tr>
<td>Hallucinations</td>
<td>1.8 ± 1.9 (0–5)</td>
<td></td>
<td></td>
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<tr>
<td>Bizarre behavior</td>
<td>1.0 ± 1.1 (0–3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive thought disorder</td>
<td>1.5 ± 1.2 (0–4)</td>
<td></td>
<td></td>
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<tr>
<td>Negative symptoms</td>
<td>1.6 ± 0.9 (0–4)</td>
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</tbody>
</table>

*Note: Data expressed as means ± SD (range in parentheses).
*0 = not present; 1 = questionable; 2 = mild; 3 = moderate; 4 = marked; 5 = severe.
**P < .01; ****P < .0005.*
Responses were recorded and scored 0–2 as recommended. Scores were averaged across the 8 stories/set. The second task was a joke-appreciation task based on Happé et al. and comprised 11 ToM cartoons that required appreciation of cartoon characters’ mental states in order to “get the joke” and 11 control cartoons which depicted slapstick or behavioral/situational humor and could be understood without inferring mental states. The 22 cartoons were intermixed and presented in a fixed pseudorandom order with participants instructed to “explain the joke.” Responses were recorded and scored 0–3 as recommended. Scores were averaged across each type of story/joke (having excluded “don’t know” responses). The third task was a picture-sequencing task comprising 4 sets of 4-card pictures stories. False-belief stories test ToM by assessing the capacity to go beyond objective facts so as to infer a character’s mistaken belief, while social-script, mechanical, and capture stories control for logical social-script reasoning, cause-and-effect reasoning, and inhibitory control, respectively. Participants rearrange picture cards to show a logical sequence of events. The order of cards was recorded and scored as recommended. Scores were averaged across each type of story (range 0–6).

**Questionnaires.** The Peters et al. Delusions Inventory (PDI) indexed a general proneness to delusional ideation. The PDI demonstrates good internal consistency and concurrent validity and has been used with both clinical and nonclinical groups. Participants responded “yes/no” to 21 questions and the number of “yes” responses was used to index delusion-proneness (range 0–21). Participants also completed the Paranoia Scale (PS) to index a more specific proneness to paranoid ideation. The PS is a 20-item self-report inventory using a 5-point Likert scale with good internal consistency, test-retest reliability, and construct validity. Total scores range from 20 to 100. The PS was developed for use with non-clinical adults but has since been used to assess paranoid ideation (not necessarily of delusional intensity) in schizophrenia.

**Interviews.** Patients were interviewed using the DIP to confirm diagnosis and the Scales for Assessing Positive and Negative Symptoms of Schizophrenia to rate symptom severity. Because an excessive internalizing, as opposed to an excessive externalizing, bias is associated specifically with depression, we also assessed levels of depression in all the participants using the Hamilton Depression Scale (HDS: range 0–52). The HDS is reported to have good reliability and validity.

**Attributional Biases.** The Internal, Personal and Situational Attributions Questionnaire (IPSAQ) assessed attributional biases. The IPSAQ is a self-report instrument comprising 16 positive and 16 negative events presented to participants in a fixed pseudorandom order. Participants imagine each event, generate the single, most likely reason for that event, and then indicate its primary cause by selecting whether that reason is due primarily to something about themselves (self-attribution), something about other people (external-personal attribution), or something about the situation (external-situational attribution). EB was calculated as percent positive self attributions minus percent negative self-attributions. Personalizing bias (PB) was calculated as percent externally attributed negative events (external-personal or external-situational) that were attributed to other people.

**Theory-of-Mind.** There were 3 ToM tasks. The first comprised 8 ToM stories testing comprehension of story characters’ mental states and 8 control stories which controlled for general comprehension abilities, with the length of stories and the sentence complexities matched across ToM and control conditions. Participants read stories, presented in a fixed pseudorandom order, silently at their own pace, and then turned the page to reveal a question (e.g., “Why did the burglar do that?”). Responses were recorded and scored 0–2 as recommended.
Results

Patients were more depressed than controls and self-reported more paranoia and higher levels of delusion-proneness (see table 1).

Table 2 summarizes IQ, memory, and probabilistic-reasoning results. Group differences in IQ were nonsignificant; however, patients showed significant memory impairments.

In the following analyses, nonparametric statistics are reported whenever distributions were skewed.

Probabilistic-Reasoning Measures

Neither the memory measures nor the IQ were significant predictors of any probabilistic-reasoning measure.

Decide Version. While 4 healthy controls (11.8%) reached a decision after only one draw, the proportion of patients doing so was significantly greater (34.3%), $\chi^2 = 4.91, P = .03$. Because the taking of 2 or fewer draws has also been used as a criterion for extreme JTC, we likewise compared the proportions of patients and controls who decided within 2 draws, and the results were generally similar, $\chi^2 = 3.47, P = .05$. A Mann-Whitney test also revealed a significant group difference in draws to decision, Mann-Whitney $Z = 2.24, P = .03$, with patients, as a group, deciding after fewer draws. Of note, it was the healthy controls with higher PDI scores (indexing a greater propensity to delusional ideation) who decided after fewer draws, $\rho = .51, P = .002$.

The group difference in decision confidence was nonsignificant, $P = .72$.

Estimate Task Results. Certainty ratings at trials 1, 10, and 20 were analyzed using a global test, revealing non-significant group differences, Wilk’s $F = 2.46, P = .07$.

However, while patients’ and controls’ certainty levels at trials 1, 10, and 20 were generally similar, the 2 groups varied significantly in their pattern of responding from trial to trial. In particular, a Mann-Whitney test revealed a highly significant group difference in the shift in certainty, $Z = 4.36, P < .0005$. The median shift in certainty for patients was 28.6% compared with only 4.2% for controls.

Attributional Biases

Table 3 summarizes results. (One patient failed to generate likely causes for a majority of events and was excluded from these analyses.) While neither the memory measures nor the IQ were significant predictors of EB, there was a significant negative correlation with depression in the patients, $r = -.37, P = .03$; patients who were more depressed were more inclined to internalize, rather than externalize, the blame for negative events. While an initial $t$ test revealed a significant group difference in EB,
Table 3. Estimated Marginal Means (± SD) for Attributional-Bias and ToM Scores for Patients and Controls

<table>
<thead>
<tr>
<th>ToM</th>
<th>Attributional Biases</th>
<th>Social-Script MechaCapturer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Picture-Sequential Task</td>
<td></td>
<td></td>
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<tr>
<td>Story-Comprehension</td>
<td></td>
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<tr>
<td>Joke-Appreciation Task</td>
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<tr>
<td>Physical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ToM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical</td>
<td>70.0 ± 30.0</td>
<td>1.27 ± 0.27</td>
</tr>
<tr>
<td>ToM</td>
<td>70.0 ± 30.0</td>
<td>1.27 ± 0.27</td>
</tr>
<tr>
<td>Physical</td>
<td>70.0 ± 30.0</td>
<td>1.27 ± 0.27</td>
</tr>
<tr>
<td>PB</td>
<td>20.6 ± 22.3</td>
<td>1.77 ± 0.27</td>
</tr>
<tr>
<td>Controls</td>
<td>30.0 ± 26.0</td>
<td>0.85 ± 0.39</td>
</tr>
<tr>
<td>ToM</td>
<td>30.0 ± 26.0</td>
<td>0.85 ± 0.39</td>
</tr>
<tr>
<td>Physical</td>
<td>30.0 ± 26.0</td>
<td>0.85 ± 0.39</td>
</tr>
<tr>
<td>PB</td>
<td>4.4 ± 25.9</td>
<td>0.97 ± 0.31</td>
</tr>
<tr>
<td>Controls</td>
<td>25.9 ± 26.0</td>
<td>0.97 ± 0.31</td>
</tr>
<tr>
<td>ToM</td>
<td>25.9 ± 26.0</td>
<td>0.97 ± 0.31</td>
</tr>
<tr>
<td>Physical</td>
<td>25.9 ± 26.0</td>
<td>0.97 ± 0.31</td>
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<tr>
<td>Attributional Biases</td>
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<tr>
<td>FALSE-BELIEF</td>
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<tr>
<td>Social-Script</td>
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<tr>
<td>Mechanical Capture</td>
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<tr>
<td>ToM</td>
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<td></td>
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<tr>
<td>Physical</td>
<td>3.83 ± 1.59</td>
<td>0.52 ± 0.49</td>
</tr>
<tr>
<td>ToM</td>
<td>3.83 ± 1.59</td>
<td>0.52 ± 0.49</td>
</tr>
<tr>
<td>Physical</td>
<td>3.83 ± 1.59</td>
<td>0.52 ± 0.49</td>
</tr>
<tr>
<td>PB</td>
<td>22.3 ± 7.00</td>
<td>1.59 ± 0.49</td>
</tr>
<tr>
<td>Controls</td>
<td>7.00 ± 3.00</td>
<td>1.05 ± 0.49</td>
</tr>
<tr>
<td>ToM</td>
<td>7.00 ± 3.00</td>
<td>1.05 ± 0.49</td>
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<tr>
<td>Physical</td>
<td>7.00 ± 3.00</td>
<td>1.05 ± 0.49</td>
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<td>PB</td>
<td>4.4 ± 25.9</td>
<td>0.97 ± 0.31</td>
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<td>Controls</td>
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<tr>
<td>Physical</td>
<td>25.9 ± 26.0</td>
<td>0.97 ± 0.31</td>
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</table>

With regards PB, patients with better cognitive function, whether indexed by IQ, $r = .43$, $P = .01$, or Digits, $r = .41$, $P = .02$, were more inclined to blame other people rather than circumstances for negative events. An initial $t$ test revealed a nonsignificant group difference in PB, $t_{67} = .76$, $P = .45$, with results remaining nonsignificant when an ANCOVA was used to adjust for IQ and memory, $F_{1,65} = 2.74$, $P = .10$.

**Theory of Mind**

ToM results are summarized in table 3. The designs for the story-comprehension and the joke-appreciation tasks were similar: $(2 \times 2)$ mixed models with 2 levels on the between-factor group (patients vs controls) and 2 levels on the repeated factor story/cartoon type (ToM vs control). Neither IQ nor Digits, $P > .10$, were significant predictors of story-comprehension scores, once LM, indexing prose recall, and word count had been taken into account. The latter 2 variables were significant independent predictors, $P < .03$; the more words the participants generated and the better their verbal comprehension/memory, the higher their story-comprehension scores. LM and word count were therefore included as covariates in an ANCOVA, the results of which revealed a significant main effect of group, $F_{1,65} = 10.72$, $P = .002$, and a near-significant 2-way interaction of group $\times$ story type, $F_{1,66} = 3.79$, $P = .056$. Simple contrasts investigating the 2-way interaction, adjusting for covariates, revealed that the patients’ ToM scores were significantly lower than those of the controls, $P < .0005$, while the scores for the control stories did not differ between groups, $P = .22$.

While the pattern of results was generally as expected, we were concerned that we may have been too conservative in using word count as a covariate. This is because the direction of causation might have been such that a basic difficulty with comprehending stories caused both a poor story-comprehension score and the generation of few words. In other words, by covarying word count, we might have been reducing power by adjusting for another index (perhaps, a less sensitive index) of the primary construct of interest—ie, story-comprehension capacity. We therefore reanalyzed results excluding word count as a covariate and relied solely upon the independent LM measure of verbal comprehension/memory as the covariate. This reanalysis revealed a significant 2-way interaction of group $\times$ story type, $F_{1,66} = 5.96$, $P = .02$.

But we think that the better test of the specificity of any ToM difficulty in the patients on this task is to compare the patients’ and the healthy controls’ ToM scores, while adjusting for their performances on the control stories. This is because the control stories were...
designed specifically to control for baseline comprehension of written stories of matched length and sentence complexity. This ANCOVA revealed a highly significant group difference, $F_{1,66} = 25.20, P < .0005$.

In order to be consistent, we then carried out the same series of analyses for the joke-appreciation task. LM and word count, $P < .01$, and neither IQ nor Digits, $P > .30$, were once again significant predictors of joke-appreciation scores; the more words the participants generated and the better their verbal comprehension/memory, the higher their joke-appreciation scores. An ANCOVA adjusting for these 2 measures then revealed a significant main effect of joke type, $F_{1,66} = 115.35, P < .0005$, and a highly significant 2-way interaction of group $\times$ joke type, $F_{1,66} = 16.97, P < .0005$. Simple contrasts, adjusting for the 2 covariates, indicated that there were group differences on the ToM jokes, $P = .047$, but not on the control jokes, $P = .66$. Results were similar when word count was removed as a covariate; the 2-way interaction was similarly significant, $F_{1,66} = 6.46, P < .01$. As argued above, however, we think that the more telling test of the specificity of the patients’ ToM difficulty on this task is to compare the patients’ and the healthy controls’ scores on the ToM jokes, adjusting for the participants’ performances on the control jokes. This analysis revealed a highly significant group difference, $F_{1,66} = 17.77, P < .0005$.

The design for the picture-sequencing task was a $(2 \times 4)$ mixed model with 2 levels on the between-factor group (patients vs controls) and 4 levels on the repeated factor sequence type (social-script vs mechanical vs capture vs false-belief). Both memory measures were significant predictors of false-belief scores across groups, $P < .005$, while IQ predicted performances on some control sequences (but not the patients), $P < .03$. In order to be conservative, we therefore included both memory measures and IQ as covariates in an initial ANCOVA which revealed the only significant result to be a 2-way interaction of group $\times$ sequence type, $F_{2,62.165.30} = 6.66, P = .001$ (Greenhouse-Geisser corrections are reported because the assumption of sphericity was violated). Simple adjusted contrasts revealed that the patients performed more poorly than the controls on both the false-belief, $P < .0005$, and the capture sequences, $P < .002$, with no significant group differences on the social-script and mechanical sequences, $P > .08$. A post hoc ANCOVA, adjusting for the capture scores, as well as the memory and IQ scores, then revealed that the patients still made significantly more errors than the controls when sequencing the false-belief stories (testing ToM), $F_{1,62} = 8.00, P = .006$. Similarly, when the false-belief scores of the patients and the controls were compared, adjusting for the participants’ performances on the control sequences, the patients continued to show a selective deficit on the false-belief sequences (which test ToM understanding), $F_{1,64} = 14.96, P < .0005$.

Findings thus far are consistent with the co-occurrence, in a general sample of schizophrenia patients who have a history of delusions of (a) a JTC bias and a bias to over-adjust in the face of changing evidence on probabilistic-reasoning tasks, (b) a more extreme externalizing (but not personalizing) attributional bias, and (c) ToM difficulties across 3 tasks. Next, we examine whether there is correlation as well as co-occurrence.

**Interrelationships Between Task Measures and Relations Between Task Measures and Ratings of Delusional Ideation**

First, a composite ToM score was calculated by standardizing and summing z scores for the story-comprehension, joke-appreciation, and false-belief ToM scores. (Results were similar if the ToM story-comprehension score was excluded from this composite measure.) This composite score was then correlated with draws to decision, shift in certainty, EB, and PB. Alpha was set at .01. All results involving EB and PB were nonsignificant. (We also used partial correlations to adjust for levels of depression in the case of EB and to adjust for cognitive function indexed by IQ and Digits scores in the case of PB and results were similar.) There was, however, a significant negative correlation between draws to decision and shift in certainty which was present across groups, $r = -.53, P < .0005$, within patients, $rho = -.53, P = .001$, and within controls, $rho = -.47, P = .005$. Draws to decision also correlated with the ToM score across groups, $r = .44, P < .0005$, and within patients, $rho = .57, P < .0005$, although not within controls, $rho = .14, P = .42$. Examination of scatterplots revealed that the latter null result reflected a tendency to ceiling effects on the composite ToM score in the controls. The correlation between ToM and shift in certainty was also significant across groups, $r = -.50, P < .0005$, although not within either the patients or the controls, $P > .10$.

Correlations between task measures and trait and state levels of delusional ideation were examined next. Somewhat surprisingly, all results involving EB were nonsignificant. There was, however, a tendency for PB to be associated with paranoia across groups, $r = .27, P = .02$, although not within either the patients or the controls, $P > .04$. Draws to decision correlated negatively with levels of delusion-proneness across groups, $r = -.44, P < .0005$, and within controls, $rho = -.51, P = .002$, although not within patients, $rho = -.28, P = .10$. Once again, examination of scatterplots revealed that the latter null result reflected a tendency to ceiling effects for the delusion-proneness measure in the patients. There was also a negative correlation between draws to decision and the clinical ratings of delusions, although this was nonsignificant, $rho = -.27, P = .12$. Moreover, there was a significant negative correlation between ToM and clinical ratings of delusions, $rho = -.44, P = .008$, while the correlation...
with levels of delusion-proneness was also significant across groups, \( \rho = -0.38, P = 0.001 \), although not within either the patients or the controls, \( P > 0.10 \).

Discussion

When compared with controls, a general sample of 35 schizophrenic patients with a history of delusions and 30 reporting current delusions showed (a) a JTC bias and a bias to overadjust certainty in line with current data on probabilistic-reasoning tasks, (b) an extreme externalizing attributional bias, and (c) performance deficits on 3 ToM tasks. While levels of PB did not differ between patients and controls, PB was found to be associated with levels of paranoid ideation across clinical and nonclinical participants. Our findings are thus generally consistent with the results of previous studies which have separately examined each of probabilistic reasoning, attributional style, and ToM. Our findings add to this literature by indicating that the attributional biases are unrelated to the other reasoning anomalies, while the probabilistic-reasoning and ToM anomalies appear related. ToM scores also correlated with ratings of delusional ideation, although it was the draws-to-decision measure that associated most robustly with a general vulnerability to delusions. PB, but not EB, was found to be associated more specifically with levels of paranoia across the clinical and nonclinical participants. While there was no evidence in our clinical and nonclinical samples of an association between EB and a proneness to paranoid ideation, it may have been that our measure of paranoid ideation (the PS) was not specific enough. Freeman,\(^{58}\) eg, have argued that paranoia is a much broader construct, with connotations going well beyond persecution (including, eg, jealousy, ideas of reference, and even grandiosity). Martin and Penn,\(^{55}\) like ourselves, also failed to find evidence of a relationship between EB and paranoid ideation, and these authors suggested that such a relationship may only exist with more specific persecutory paranoid ideation.

Overall, our results suggest that a common underlying mechanism in people with schizophrenia contributes to the probabilistic-reasoning anomalies and the ToM deficits (but not the attributional biases) that have been linked to delusional ideation. Results also suggest that this commonality is not attributable to poor IQ or impaired memory. Theorizing about the role of probabilistic and ToM anomalies in delusion formation thus need to consider mechanisms which are more general than task-specific processes such as setting decision thresholds on probabilistic-reasoning tasks or representing other people’s thoughts as decoupled from reality (purportedly underpinned by medial prefrontal circuitry) on ToM tasks.

As to what this mechanism might be, we draw upon the following:

1. From the delusion literature, that deluded people have a difficulty with overriding the automatic salience of first-person evidence in order to give equal weight to one’s own experience, the views of other people, and third-person general knowledge\(^{5}\);
2. From the ToM literature, that performance deficits on ToM tasks in schizophrenia reflect an egocentric difficulty with representing multiple concurrent, yet potentially discordant, viewpoints of the same here-and-now reality\(^{29,30,56}\); and
3. From Hemsley’s\(^{57–59}\) work, that schizophrenia is characterized by a weakening of the influence of stored memories of past regularities on current perception.

While acknowledging that our ideas are preliminary because they rely on correlational data rather than any direct test of our theory, we speculate that the common underlying mechanism in schizophrenia is a difficulty with inhibiting thoughts that present relatively directly to consciousness as the “perceived reality.” This would explain the tendencies of people with schizophrenia to be swayed more by current data when making decisions on probabilistic-reasoning tasks and the difficulties with going beyond objective facts, as presented on ToM tasks, so as to infer other people’s beliefs. This inhibitory impairment also dissociates from the excessive attributional biases also seen in schizophrenia. While the latter may reflect self-defense mechanisms triggered by perceived threat, the former may reflect brain dysfunction, consistent with recent evidence that a failure to inhibit the “self-perspective” (information presented directly to participants on classic false-belief ToM tasks) causes poor ToM task performances in patients with right-frontal brain injury\(^ {60,61}\) an area of the brain associated with normal belief revision.\(^ {7}\)

In conclusion, we suggest that the probabilistic-reasoning anomalies and the ToM deficits that have been associated with delusions are, in part, functionally related in schizophrenia and depend upon a common impairment of the capacity to inhibit perceived reality. Moreover, we note that this inhibitory impairment cannot be reduced to a generalized frontal dysfunction because ToM deficits are independent of co-occurring executive deficits in schizophrenia.\(^ {62,63}\) We suggest, instead, that specific inhibitory processes for distancing ourselves from perceived reality (“the evidence of our senses”) likely evolved as a part of the “social brain” so as to facilitate the imagining of other subjective viewpoints, thus promoting intersubjective communication within a shared reality.

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