

Jumping to Conclusions, Neuropsychological Functioning, and Delusional Beliefs in First Episode Psychosis

M. Aurora Falcone^{*1,2}, Robin M. Murray², Benjamin D. R. Wiffen², Jennifer A. O'Connor², Manuela Russo³, Anna Kolliakou², Simona Stilo^{2,4}, Heather Taylor², Poonam Gardner-Sood², Alessandra Paparelli², Fatima Jichi⁵, Marta Di Forti², Anthony S. David², Daniel Freeman⁶, and Suzanne Jolley¹

¹Department of Psychology, Institute of Psychiatry, King's College London, London, UK; ²Department of Psychosis Studies, Institute of Psychiatry, King's College London, London, UK; ³Department of Psychiatry, Mount Sinai School of Medicine, New York, NY; ⁴Department of Health Service and Population Research, Institute of Psychiatry, King's College London, London, UK; ⁵Department of Biostatistics, King's College London, Institute of Psychiatry, London, UK; ⁶Department of Psychiatry, University of Oxford, Warneford Hospital, Oxford, UK

*To whom correspondence should be addressed; Department of Psychosis Studies, PO 52, Institute of Psychiatry, De Crespigny Park, London SE5 8AF, UK; tel: +44-(0)207-848-0100, fax: +44-(0)207848-0287, e-mail: aurora.falcone@kcl.ac.uk

Background: The “jumping to conclusions” (JTC) data-gathering bias is implicated in the development and maintenance of psychosis but has only recently been studied in first episode psychosis (FEP). In this study, we set out to establish the relationship of JTC in FEP with delusions and neuropsychological functioning. **Methods:** One hundred and eight FEP patients and 101 age-matched controls completed assessments of delusions, general intelligence (IQ), working memory (WM), and JTC (the probabilistic reasoning “beads” task). **Results:** Half the FEP participants jumped to conclusions on at least 1 task, compared with 25% of controls (OR range 2.1 to 3.9; 95% CI range 1.5 to 8.0, P values $\leq .02$). JTC was associated with clinical, but not nonclinical delusion severity, and with neuropsychological functioning, irrespective of clinical status. Both IQ and delusion severity, but not WM, were independently associated with JTC in the FEP group. **Conclusions:** JTC is present in FEP. The specific association of JTC with clinical delusions supports a state, maintaining role for the bias. The associations of JTC with neuropsychological functioning indicate a separable, trait aspect to the bias, which may confer vulnerability to psychosis. The work has potential to inform emerging interventions targeting reasoning biases in early psychosis.

Key words: psychosis/delusions/reasoning/jumping to conclusions/neuropsychology

Introduction

The jumping to conclusions (JTC) data-gathering bias is the most comprehensively studied of the reasoning biases

associated with psychosis.^{1–4} It is hypothesized to lead to hasty decision making, acceptance of incorrect ideas, and the failure to consider alternative explanations and hence to the formation and maintenance of delusional beliefs.^{5–14} The importance of the JTC bias is that it is modifiable, and therefore understanding its components has the potential to improve interventions and, consequently, clinical outcomes.^{15–23}

The JTC bias is usually assessed by a probabilistic reasoning task, the “beads task,”²⁴ in which participants are asked to request information in the form of colored beads drawn from one of 2 jars in order to make a decision about their jar of origin. A tendency to JTC has been operationally defined by both the number of draws a respondent requests before making their decision and by how quickly they rate themselves to be certain. Recent studies and reviews have argued for the superiority of a categorical definition of JTC as a decision made after viewing fewer than 3 beads.^{3,25} When assessed in this way, the bias is reliably found in around 50% of people with delusions and is associated with delusional ideation in the general population, psychosis vulnerability, and change in symptomatology,^{1–5,24–30} supporting its hypothesized role in the development and maintenance of the clinical disorder. Findings are mixed with regard to the association of JTC with other reasoning biases.^{5–7,10,13,14} The majority of studies suggest a specific association of JTC with delusion severity.^{3,15}

There are also indications of a link between JTC and neuropsychological functioning, which has been reported for both clinical psychosis and nonclinical participants. Garety and colleagues³¹ found that verbal intelligence

and JTC were significantly associated, as did Moritz and colleagues¹⁴ on their own JTC task variant. Lincoln and colleagues³² found that the association of the bias with delusions was rendered nonsignificant when IQ was controlled. Corcoran and colleagues³³ found that the lower the IQ score, the hastier the data gathering in a clinical group with psychotic depression. In 2 large factor-analytic studies including clinical and nonclinical participants,^{34,35} both neuropsychological functioning and JTC loaded on the same factor, and van Dael and colleagues³⁶ reported lower IQ scores in those who jumped to conclusions among nonpsychotic relatives of patients with schizophrenia and controls with high levels of psychotic experiences. There is emerging evidence to suggest that JTC is particularly associated with worse performance on working memory (WM) tasks^{26,37,38} and with impaired executive functioning.^{35,39,40}

JTC has been less comprehensively studied in first episode psychosis (FEP) with mixed findings regarding associations of the bias with delusions and neuropsychological functioning.^{41–45} So and colleagues⁴² found a strong JTC bias in a FEP group of 30 people with current clinician-rated delusions, compared with 30 nonclinical controls. Dudley and colleagues⁴³ tested 77 patients from early psychosis services and found that while 47% showed the JTC bias, neither the rate of JTC nor hastiness in data gathering differed between the 25 participants with current distressing delusions, and those without. JTC predicted delusional persistence over time,⁴⁴ in line with a small inpatient study,⁴¹ but was not associated with neuropsychological functioning in a subset ($n = 29$, 9 JTC) at follow-up.⁴⁵ As both delusions and neuropsychological functioning are associated with JTC in persistent psychosis, a large, controlled investigation of their associations with the bias in an FEP group, using standardized ratings of delusions, is warranted. This was the aim of the current study.

We tested the following hypotheses:

1. The FEP group will be more likely to JTC than non-psychotic, age-matched, general population controls;
2. JTC will be associated with severity of delusions in the FEP group and delusional ideation in the control group;
3. JTC will be associated with neuropsychological functioning, both general intelligence and WM, irrespective of clinical status.

We also carried out an exploratory analysis to determine the independence of the associations of the JTC bias with delusions/delusional ideation and with neuropsychological functioning.

Methods

Participants

Participants were recruited and assessed as part of the Genetics and Psychosis (GAP) study, which was designed

to identify genetic and environmental factors associated with psychosis.^{46–48} Ethical approval for the study was obtained from the Institute of Psychiatry and South London and Maudsley NHS Foundation Trust Research Ethics Committee. All study participants gave informed written consent to enter the research. The study recruited across 4 London boroughs, each with similar sociodemographic profiles. FEP participants were recruited from inpatient wards and community mental health teams. Controls were recruited by randomized house visits, newspaper advertisement, and leaflets distribution. Inclusion criteria for the FEP group were: a current diagnosis of FEP; within 6 months of first contact with services; current psychotic symptoms, experienced for at least 7 days. Exclusion criteria for both groups were: a history of moderate or severe learning disabilities, or current IQ < 70; insufficient command of English to complete assessments; a history of previous contact with mental health services for the presence of psychosis; age outside the range 18–65 years. For the FEP group, a primary diagnosis of alcohol or substance dependency or a known organic cause of psychosis were additional exclusion criteria. Controls were excluded if their scores on the Psychosis Screening Questionnaire⁴⁹ indicated current psychosis but included if they indicated psychotic-like experiences that did not reach the threshold of clinical significance.

Measures

Demographic data were collected from self-report, supplemented, for FEP participants, by the clinical record.

JTC: the probabilistic reasoning “beads” task²⁴ was employed to assess participants’ tendency to JTC. Two neutral versions of the task were administered, with beads in 85:15 and 60:40 ratios. For the first, “easy,” ratio task, participants were shown a jar with 85 orange and 15 black beads (the “Mainly Orange” jar) and a jar with 85 black and 15 orange beads (the “Mainly Black” jar), on a computer screen. The jars were then removed from view and the participants told that one of the jars had been selected by the computer. The participant was asked to request as many colored beads as she/he would like to see before deciding from which of the 2 hidden jars the beads were being drawn. Requested beads were left visible as memory aid. Following the 85:15 task, participants completed the more difficult version of the task, with beads in the ratio 60:40 (“Mainly Blue” vs “Mainly Red” jars). Draws for the 85:15 task followed the pattern: OOOBOOOBBOOOBBOOOOO, where O = orange and B = black; and for the 60:40 task: BRRBBRBBRRBBBBRRRBRB, where B = blue and R = red. The key variable employed was a dichotomous rating of JTC based on the number of beads requested before making a decision, with fewer than 3 beads classified as JTC.²⁴ A dichotomous rating, using the established convention of fewer than 3 draws to indicate JTC, was

preferred over a continuous measure for 2 reasons: (1) the number of draws is not normally distributed in a continuous scale, as the information value of each single new bead differs according to the color of the bead and the sequence employed; and (2) the dichotomous scoring method has been shown to have a better model fit in predicting change in delusion conviction.²⁵

Neuropsychological functioning: general intelligence was assessed using a brief version of the Wechsler Adult Intelligence Scale—Third Edition (WAIS-III).⁵⁰ The WAIS-III is widely used, extensively validated, and reliable. A standardized set of 5 tasks, representing each of the identified indices of the full test (Information, Verbal Comprehension Index; Block Design and Matrix Reasoning, Perceptual Organization Index; Digit Symbol Coding, Processing Speed Index; Digit Span, Working Memory Index) was used in the current study to give a prorated intelligence quotient (IQ). As the beads task involves the manipulation of visually presented data, WM was assessed using the spatial WM task of the Wechsler Memory Scale.⁵¹

Clinical delusions were assessed using the delusions item of the Positive Symptoms subscale of the Positive and Negative Syndrome Scale (PANSS).⁵² The PANSS is a 30-item, 7-point (from 1 = symptom absent to 7 = extreme severity) clinical rating of the symptoms associated with schizophrenia. Ratings were based on symptomatology reported over the week preceding assessment.

Nonclinical delusional ideation was rated using the Psychosis Screening Questionnaire (PSQ).⁴⁹ The PSQ scale was employed to ensure that control participants were not currently psychotic but provides severity ratings of the occurrence of 5 categories of psychotic symptoms (hypomania, thought interference, persecutory delusion, unusual experiences, and auditory hallucinations) over the year preceding assessment on a 3-point scale (0 No, 1 Unsure, 2 Yes).

Design and Procedure

The GAP study employed a cross-sectional case-control design with a large battery of biological, clinical, social, and neuropsychological measures. All GAP participants with JTC and IQ data were included in the current study. Assessments commenced within 3 months of consent. The probabilistic reasoning task was usually administered on the same day as the neuropsychological tasks and always before them. A face-to-face diagnostic interview was carried out for all FEP participants by trained researchers and was supplemented by scrutiny of clinical records. Diagnoses were made using OPCRIT⁵³ according to Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria.⁵⁴ Ten correct test diagnoses were required for researchers to reach reliability. Interrater reliability for diagnostic assessments was very high (Cronbach's alpha = .97). All raters were trained

to a criterion of reliability, for each specific measure, by experts in its design and administration.

Analyses

Data analysis was conducted using the Statistical Package for the Social Sciences Version 20.0 (IBM, 2011). Preliminary chi-square and *t* test analyses were employed to examine differences in age, gender, ethnicity, and neuropsychological functioning between the FEP and control groups. The main hypotheses were tested by 3 series of binary logistic regression analyses, with the dichotomous rating of JTC as the dependent variable (JTC = 1; no JTC = 0). The first series tested hypothesis 1, with clinical status as the independent variable (FEP = 1; Control = 0), entered firstly alone, then controlling for gender and ethnicity, as these differed significantly between the FEP and control groups. The second series of regressions tested hypothesis 2; severity of delusions/delusional ideation was entered as a continuous independent variable in 2 separate analyses for the FEP and control groups. In the third regression series, IQ and WM were entered as continuous independent variables, separately and then together, for FEP and control groups combined controlling for clinical status, gender, and ethnicity, then for the FEP and control groups separately. Finally, for the exploratory analysis, delusion severity was entered with both IQ and WM in a backward regression, to assess their independent associations with JTC. Analyses were repeated for each task (85:15 and 60:40) separately.

Results

Demographic and Clinical Characteristics

The study sample comprised 108 FEP and 101 control participants. Demographic characteristics are shown in [table 1](#). Diagnoses for the FEP group were predominantly schizophrenia spectrum (Schizophrenia, *n* = 21, 19%; Delusional Disorder, *n* = 3, 3%; Schizoaffective Disorder, *n* = 15, 14%; Other Psychotic Disorder, *n* = 34, 31%), with just under a third meeting criteria for an affective psychosis (Manic Psychosis, *n* = 18; 17%; Depressive Psychosis, *n* = 17, 16%). JTC rates did not differ according to type of diagnosis on either task (Schizophrenia spectrum vs Affective diagnosis; χ^2 values < 2.5, *df* 1, *P* values > .1). The mean PANSS delusions score in the FEP group was 2.8 (SD 1.6, *n* = 99). The range of delusion scores (1–6) indicated a good spread of symptomatology, with a third of participants scoring above 3, indicating the presence of current delusions. Mean PSQ scores for the control group were: PSQ Total 2.3 (SD 3.5, *n* = 93, possible range 0–10); PSQ delusions 0.4 (SD 0.8, *n* = 95, possible range 0–2).

Hypothesis 1: The first episode group will be more likely to JTC than controls, and JTC will be associated with the severity of delusions/delusional ideation

Table 1. Demographic and Cognitive Characteristics of the FEP and Control Groups

	FEP (<i>n</i> = 108)	Control (<i>n</i> = 101)	Statistic
	Mean (SD)		<i>t</i> (<i>df</i>), <i>P</i>
Age (in years) at consent (range: 18–65)	30.0 (9.0)	30.9 (12.7)	0.6 (207), <i>P</i> = .5
General intelligence (IQ) (range: 70–155)	92.5 (15.8)	107.4 (16.3)	7.1 (207), <i>P</i> < .001
Working memory ^a (range: 1–18)	9.2 (3.07)	10.88 (2.9)	4.0 (198), <i>P</i> < .001
	<i>n</i> (%)		χ^2 (<i>df</i>), <i>P</i>
Gender			
Male/Female	71/37 (66/34)	47/54 (46/53)	7.8 (1), <i>P</i> = .006
Ethnicity			
White	45 (42)	62 (61)	8.1 (2), <i>P</i> = .017
Black	46 (43)	29 (29)	
Other	17 (16)	10 (10)	

Note: FEP, first episode psychosis; IQ, intelligence quotient.

^aControl = 91; FEP = 100.

Table 2. Binary Logistic Regression Analysis of the Association of the JTC Bias With Clinical Status Controlling for Demographic Variables Across Tasks

Task	Clinical Status OR	95% CI	<i>P</i> Value
JTC 85:15	2.5	1.4 to 4.5	.003
	2.4 ^a	1.3 to 4.4	.005
	2.2 ^b	1.2 to 4.0	.01
	2.1 ^c	1.1 to 4.0	.02
JTC 60:40	3.8	1.8 to 7.9	.001
	3.9 ^a	1.8 to 8.4	<.001
	3.5 ^b	1.6 to 7.5	.001
	3.7 ^c	1.7 to 8.0	.001

Note: JTC, jumping to conclusions.

^aControlling for gender.

^bControlling for ethnicity.

^cControlling for gender and ethnicity.

Half of the FEP participants (49%) demonstrated the JTC bias on at least 1 task, compared with only a quarter of controls (26%). Rates were higher on the 85:15 task (FEP 44% JTC; Control 24% JTC) than the 60:40 task (FEP 31% JTC; Control 11% JTC). Mean draws to decision was 4.6 (SD 5.1) on the 85:15 task and 6.3 (SD 5.5) on the 60:40 task. Logistic regression analyses revealed clinical status to be a significant predictor of the tendency to JTC, across tasks, and even after controlling for gender and ethnicity (table 2).

Hypothesis 2: JTC will be associated with severity of delusions/delusional ideation

Delusion severity was significantly associated with the tendency to JTC on the 85:15 task (OR = 1.3, *P* = .03, 95% CI 1.0 to 1.7; JTC mean PANSS delusions 3.2 SD 1.5; no JTC mean 2.5 SD 1.6) but did not reach significance on the 60:40 task (OR = 1.1, *P* = .40, 95% CI 0.9 to 1.5). Nonclinical delusional ideation and total PSQ were not associated with JTC (0.9 < ORs < 1.1, *P* values > .70).

Hypothesis 3: JTC will be associated with neuropsychological functioning, irrespective of clinical status

Both general intelligence and WM scores were lower in the JTC group compared with the no JTC group, in both FEP and control groups, and the 2 groups combined, and the pattern was identical across both task variants (table 3). Logistic regression showed both components of neuropsychological functioning to be significantly associated with JTC, irrespective of clinical status and task, with each point decrease in IQ increasing the likelihood of JTC by around 4%, and each point decrease in WM score increasing the likelihood of JTC by around 20%. When both variables were entered together, for the whole sample, the change in the odds of JTC associated with IQ remained stable (85:15 task OR 0.97, 95% CI 0.9 to 1.0, *P* = .03; 60:40 task OR 0.9, 95% CI 0.9 to 1.0, *P* = .002), while the change in odds associated with WM score was halved, and became nonsignificant (85:15 task OR 0.9 95% CI 0.8 to 1.0, *P* = .03; 60:40 task OR 0.9, 95% CI 0.8 to 1.0, *P* = .09). Clinical status was no longer a significant predictor when IQ and WM were entered as predictors together (OR 1.3 and 1.6, for the 85:15 and 60:40 tasks, respectively).

Exploratory Analysis: Are Neuropsychological Profile and Delusion Severity Independently Associated With JTC?

To examine the independent associations of delusion severity, IQ, and WM with JTC, all 3 variables were entered into a backward regression model. As only clinical delusions were associated with JTC and only on the 85:15 task variant, analysis was restricted accordingly. The model resolved in 2 steps; both delusion severity (OR 1.4, 95% CI 1.0 to 1.9, *P* = .03) and IQ (OR 0.9, 95% CI

Table 3. Neuropsychological Functioning Differences According to the Tendency to JTC

		Mean (SD), <i>n</i>		OR, 95% CI, <i>P</i>
		JTC	No JTC	
85:15 Task				
All	IQ	92.4 (16.2), 71	103.4 (15.9), 138	0.96, 0.9 to 1.0, <i>P</i> = .002 ^a
	WM	8.7 (3.2), 66	10.6 (2.8), 125	0.8, 0.7 to 0.9, <i>P</i> = .001 ^a
FEP	IQ	88.4 (13.0), 47	95.6 (13.7), 61	0.96, 0.9 to 1.0, <i>P</i> = .009
	WM	8.19 (3.0), 43	9.9 (3.0), 57	0.8, 0.7 to 0.9, <i>P</i> = .008
Control	IQ	100.1 (19.0), 24	109.6 (14.7), 77	0.96, 0.9 to 1.0, <i>P</i> = .01
	WM	9.7 (3.3), 23	11.3 (2.6), 68	0.8, 0.7 to 1.0, <i>P</i> = .03
60:40 Task				
All	IQ	88.13 (14.9), 45	102.8 (15.9), 164	0.9, 0.9 to 1.0, <i>P</i> < .001 ^a
	WM	8.27 (3.0), 41	10.4 (2.9), 150	0.8, 0.7 to 0.9, <i>P</i> = .002 ^a
FEP	IQ	86.2 (13.5), 34	95.3 (13.1), 74	0.9, 0.9 to 1.0, <i>P</i> = .002
	WM	8.2 (3.0), 30	9.6 (3.0), 70	0.8, 0.7 to 1.0, <i>P</i> = .04
Control	IQ	94.0 (18.1), 11	109.0 (15.4), 90	0.9, 0.9 to 1.0, <i>P</i> = .006
	WM	8.6 (3.2), 11	11.2 (2.7), 80	0.7, 0.6 to 0.9, <i>P</i> = .007

Note: Abbreviations are explained in the first footnote to tables 1 and 2. WM, working memory.

^aControlling for clinical status, gender, and ethnicity.

0.9 to 1.0, *P* = .002) were independently associated with JTC. WM was not included in the final model.

Discussion

We set out to investigate the associations of the JTC reasoning bias with delusions and neuropsychological functioning in a large FEP group, compared with a non-psychotic control group, employing standardized and stringently administered assessments. Our purpose was to provide a convincing test of whether the associations of JTC with both delusion severity and neuropsychological functioning, demonstrated in established psychosis, were replicated in a FEP group. Previous research on FEP participants has been inconclusive, with variation in the methods used to assess delusions and small samples, limiting the number of participants with the JTC bias and with delusions.

We found a prevalence of JTC of almost half in the FEP group, who were more than twice as likely as the control group to JTC. We also found clear associations of JTC with delusion severity, but not subclinical delusional ideation, and with neuropsychological functioning, both general intelligence and WM, irrespective of clinical status. Delusion severity and general intelligence were independently associated with JTC.

The findings indicate that the JTC bias is as prominent in FEP as in more established psychosis, consistent with the report of Dudley and colleagues.⁴³ In common with the majority of the literature, but in contrast to Dudley and colleagues,⁴³ JTC was associated with delusions, as assessed by a standardized rating of severity (in contrast to the rating of distress employed by Dudley and colleagues). The association of JTC with current delusions

remained consistent irrespective of controlling for neuropsychological functioning, in contrast to some of the previous literature in established psychosis, which has argued for a stronger relationship of JTC with neuropsychological functioning.^{32,34,36} The disparities in the existing literature may be attributable to methodological differences. For example, other studies variously recruited participants with a limited range of delusion severity; used atypical tasks to assess JTC; or reported low base rates of JTC, all of which may contribute to a weaker association between JTC and delusions. It is also noteworthy that the association of JTC with delusions in our study was found only on the 85:15 version of the beads task and not on the 60:40 version. Restricting investigation to tasks with beads in more difficult ratios may therefore also contribute to inconsistency between studies. The current study was not designed to investigate differences between JTC tasks, but this should be addressed in future studies, employing an appropriate design (eg, counterbalancing order of administration and colors used).

A consistent and robust relationship was found between JTC and neuropsychological functioning, both general intelligence and WM, across FEP and control participants. JTC was associated with lower scores on the neuropsychological tasks. Effect sizes were small but highly significant. Controlling for demographic and clinical variables did not alter the magnitude of the effect size. The association of JTC with clinical status was rendered nonsignificant by the inclusion of neuropsychological functioning in the model. In contrast, the association of neuropsychological functioning with JTC remained consistent irrespective of controlling for clinical status. Furthermore, the magnitude of the association was consistent across both FEP and control groups.

Findings are consistent with neuropsychological functioning having both a state and a trait influence on JTC. As a trait vulnerability factor, worse cognitive functioning is shown to be linked with the presence of JTC across clinical and control populations; as a state phenomenon, the worsening of cognitive functioning associated with schizophrenia may increase the tendency to JTC, with a consequent increase in vulnerability to delusional thinking.

JTC in the control group was solely predicted by levels of IQ and WM, and no association with subclinical psychotic-like experiences (including delusional ideation) was found. Others have reported no association between JTC and delusion proneness,⁵⁵ but studies using more comprehensive and detailed measures of delusional ideation than in the present study have found associations with JTC.^{56,57} The PSQ⁴⁹ is designed as a screen, which identifies psychotic-like experiences in the general population. It measures delusional ideation by a single item, which refers to suspiciousness; thus, this constitutes a limitation of the present work. As previous studies suggest that it may be conviction in delusional ideation, rather than simply endorsement of an unusual idea, that is most associated with JTC,^{6,11,24,29,57} our failure to find an association between JTC and severity of delusional ideation in the control group may be a limitation of measurement, rather than necessarily going against a continuum model.⁵⁸ Nevertheless, the strong association of JTC with clinical status does suggest that whatever factors distinguish delusions or delusion-like ideas—be it conviction or other variables—may have distinct cognitive underpinnings.

Limitations

The study employed a cross-sectional design and therefore the hypothesized causal role of JTC in the onset and maintenance of psychosis cannot be demonstrated. We did not formally assess comprehension of the JTC task, which may have influenced performance.^{59,60} The causal role of neuropsychological variables in the occurrence of JTC is also untested. The PSQ is a crude instrument for the assessment of psychotic-like symptomatology and delusional ideation in a control group, and our finding of no association of JTC with delusional ideation may be attributable to limitations of measurement. Furthermore, the PSQ was not suitable for administration to both clinical and control participants and therefore restricted the investigation of the associations between delusions and JTC across all participants. The neuropsychological battery, while more extensive than those usually employed to assess the association between JTC and functioning, did not constitute a full neuropsychological assessment, and WM in particular was assessed by a single test. However, each index of the full-scale IQ was represented, and evidence suggests that in psychiatric settings, short forms

show little meaningful deviation from full-scale scores.⁶¹ Although the beads task involves visually presented material, it is possible that participants employ verbal strategies in their decision making, so a more comprehensive WM assessment would have been desirable.

Implications

Our findings, together with previous research, indicate that JTC has both clinical and neuropsychological correlates. The bias appears to arise in the context of a specific neuropsychological profile and in the presence of delusions. The link between JTC and neuropsychological functioning in controls is consistent with the bias operating as a *trait* vulnerability factor, which may contribute to the formation of psychotic symptoms. In terms of mechanism, specific cognitive difficulties with processing contextual information have been hypothesized to underlie both JTC and the neuropsychological profile characteristic of psychosis and psychosis vulnerability^{62,63} and may therefore be a candidate common process. The relationship with severity of clinical delusions identifies the bias as having a *state* component, which can be hypothesized to maintain current psychotic symptomatology, by limiting the information considered in decision making and thereby both increasing the likelihood of, and perpetuating, incorrect conclusions.

Our findings support the practice of targeting reasoning biases with psychological interventions to reduce the severity of delusions^{15–23} and suggest that these interventions could be adopted at an early stage to attempt to prevent transition to psychosis or reduce psychosis risk. The findings imply that reasoning interventions could be usefully supplemented by strategies to improve neuropsychological functioning and highlight candidate mechanisms by which cognitive behavioral and cognitive remediation intervention strategies could work synergistically. The conceptualization of JTC as both a *trait* and a *state* phenomenon delineates routes for future longitudinal research to test the impact of naturalistic or induced changes in JTC and neuropsychological functioning on the likelihood of developing psychosis and on current delusional severity.

Conclusions

The JTC reasoning bias is elevated in FEP and is specifically associated with delusion severity. The bias is also associated with neuropsychological functioning, irrespective of clinical status and delusion severity. The results are consistent with JTC operating (1) as part of a neuropsychological vulnerability to psychosis and (2) as a maintaining influence on current delusions. Changes in reasoning may therefore impact on both transition to psychosis and symptom severity and are suitable targets for intervention.

Funding

This work was supported by NIHR Biomedical Research Centre for Mental Health at the South London and Maudsley NHS Foundation Trust and Institute of Psychiatry, King's College London; the Institute of Psychiatry at King's College London; the Psychiatry Research Trust. D.F. is supported by a Medical Research Council Senior Clinical Fellowship (G0902308).

Acknowledgments

We are grateful for the time and effort of all participants recruited for this study. We thank all the GAP researchers and Principle Investigators for their contributions to this study. The authors have declared that there are no conflicts of interest in relation to the subject of this study.

References

- Freeman D. Suspicious minds: the psychology of persecutory delusions. *Clin Psychol Rev.* 2007;27:425–457.
- Garety PA, Freeman D. Cognitive approaches to delusions: a critical review of theories and evidence. *Br J Clin Psychol.* 1999;38(pt 2):113–154.
- Garety PA, Freeman D. The past and future of delusions research: from the inexplicable to the treatable. *Br J Psychiatry.* 2013;203:327–333.
- So SH, Garety PA, Peters ER, Kapur S. Do antipsychotics improve reasoning biases? A review. *Psychosom Med.* 2010;72:681–693.
- Colbert SM, Peters ER. Need for closure and jumping-to-conclusions in delusion-prone individuals. *J Nerv Ment Dis.* 2002;190:27–31.
- McKay R, Langdon R, Coltheart M. Need for closure, jumping to conclusions, and decisiveness in delusion-prone individuals. *J Nerv Ment Dis.* 2006;194:422–426.
- McKay R, Langdon R, Coltheart M. Jumping to delusions? Paranoia, probabilistic reasoning, and need for closure. *Cogn Neuropsychiatry.* 2007;12:362–376.
- Merrin J, Kinderman P, Bentall RP. Jumping to conclusions and attributional style in persecutory delusions. *Cogn Ther Res.* 2007;31:741–758.
- White LO, Mansell W. Failing to ponder? Delusion-prone individuals rush to conclusions. *Clin Psychol Psychother.* 2009;16:111–124.
- Moritz S, Woodward TS. Plausibility judgment in schizophrenic patients: evidence for a liberal acceptance bias. *Germ J Psychiatry.* 2004;7:6674.
- Moritz S, Woodward TS. Jumping to conclusions in delusional and non-delusional schizophrenic patients. *Br J Clin Psychol.* 2005;44:193–207.
- Moritz S, Woodward TS, Hausmann D. Incautious reasoning as a pathogenetic factor for the development of psychotic symptoms in schizophrenia. *Schizophr Bull.* 2006;32:327–331.
- Moritz S, Woodward TS, Jelinek L, Klinge R. Memory and metamemory in schizophrenia: a liberal acceptance account of psychosis. *Psychol Med.* 2008;38:825–832.
- Moritz S, Veckenstedt R, Hottenrott B, Woodward TS, Randjbar S, Lincoln TM. Different sides of the same coin? Intercorrelations of cognitive biases in schizophrenia. *Cogn Neuropsychiatry.* 2010;15:406–421.
- Garety PA, Freeman D, Jolley S, Waller H, Dunn G. Jumping to conclusions: the psychology of delusional reasoning. *Adv Psych Treat.* 2011;17:332–339.
- Warman DM, Martin JM, Lysaker P. Jumping to conclusions and delusions: the impact of discussion of the bias on the bias. *Schizophr Res.* 2013;150:575–579.
- Ross K, Freeman D, Dunn G, Garety P. A randomized experimental investigation of reasoning training for people with delusions. *Schizophr Bull.* 2011;37:324–333.
- Waller H, Freeman D, Jolley S, Dunn G, Garety P. Targeting reasoning biases in delusions: a pilot study of the Maudsley Review Training Programme for individuals with persistent, high conviction delusions. *J Behav Ther Exp Psychiatry.* 2011;42:414–421.
- Sanford N, Woodward TS, Lecomte T, Leclerc C, Wykes T. Change in jumping to conclusions linked to change in delusions in early psychosis. *Schizophr Res.* 2013;147:207–208.
- Moritz S, Kerstan A, Veckenstedt R, et al. Further evidence for the efficacy of a metacognitive group training in schizophrenia. *Behav Res Ther.* 2011;49:151–157.
- Moritz S, Veckenstedt R, Randjbar S, Vitzthum F, Woodward TS. Antipsychotic treatment beyond antipsychotics: metacognitive intervention for schizophrenia patients improves delusional symptoms. *Psychol Med.* 2011;41:1823–1832.
- Moritz S, Veckenstedt R, Bohn F, et al. Complementary group metacognitive training (MCT) reduces delusional ideation in schizophrenia. *Schizophr Res.* 2013;151:61–69.
- Woodward TS, Munz M, LeClerc C, Lecomte T. Change in delusions is associated with change in “jumping to conclusions”. *Psychiat Res.* 2009;170:124–127.
- Garety PA, Freeman D, Jolley S, et al. Reasoning, emotions, and delusional conviction in psychosis. *J Abnorm Psychol.* 2005;114:373–384.
- So SH, Freeman D, Dunn G, et al. Jumping to conclusions, a lack of belief flexibility and delusional conviction in psychosis. *J Abnorm Psychol.* 2012;121:129–139.
- Broome MR, Johns LC, Valli I, et al. Delusion formation and reasoning biases in those at clinical high risk for psychosis. *Br J Psychiatry Suppl.* 2007;51:s38–s42.
- Peters E, Garety P. Cognitive functioning in delusions: a longitudinal analysis. *Behav Res Ther.* 2006;44:481–514.
- Bensi L, Giuberti F, Nori R, Gambetti E. Individual differences and reasoning: a study on personality traits. *Br J Psychol.* 2010;101:545–562.
- Freeman D, Pugh K, Garety P. Jumping to conclusions and paranoid ideation in the general population. *Schizophr Res.* 2008;102:254–260.
- Warman DM, Lysaker PH, Martin JM, Davis L, Haudenschild SL. Jumping to conclusions and the continuum of delusional beliefs. *Behav Res Ther.* 2007;45:1255–1269.
- Garety PA, Hemsley DR, Wessely S. Reasoning in deluded schizophrenic and paranoid patients. Biases in performance on a probabilistic inference task. *J Nerv Ment Dis.* 1991;179:194–201.
- Lincoln TM, Lange J, Buro J, Exner C, Moritz S. The effect of state anxiety on paranoid ideation and jumping to conclusions. An experimental investigation. *Schizophr Bull.* 2010;36:1140–1148.
- Corcoran R, Rowse G, Moore R, et al. A transdiagnostic investigation of ‘theory of mind’ and ‘jumping to

- conclusions' in patients with persecutory delusions. *Psychol Med*. 2008;38:1577–1583.
34. Bentall RP, Rowse G, Shryane N, et al. The cognitive and affective structure of paranoid delusions: a transdiagnostic investigation of patients with schizophrenia spectrum disorders and depression. *Arch Gen Psychiatry*. 2009;66:236–247.
 35. Woodward TS, Mizrahi R, Menon M, Christensen BK. Correspondences between theory of mind, jumping to conclusions, neuropsychological measures and the symptoms of schizophrenia. *Psychiatry Res*. 2009;170:119–123.
 36. Van Dael F, Versmissen D, Janssen I, Myin-Germeys I, van Os J, Krabbendam L. Data gathering: biased in psychosis? *Schizophr Bull*. 2006;32:341–351.
 37. Garety P, Joyce E, Jolley S, et al. Neuropsychological functioning and jumping to conclusions in delusions. *Schizophr Res*. 2013;150:570–574.
 38. Lunt L, Bramham J, Morris RG, et al. Prefrontal cortex dysfunction and jumping to conclusions: bias or deficit? *J Neuropsychol*. 2012;6:65–78.
 39. Rubio JL, Ruiz-Veguilla M, Hernández L, et al. Jumping to conclusions in psychosis: a faulty appraisal. *Schizophr Res*. 2011;133:199–204.
 40. van Hooren S, Versmissen D, Janssen I, et al. Social cognition and neurocognition as independent domains in psychosis. *Schizophr Res*. 2008;103:257–265.
 41. Menon M, Mizrahi R, Kapur S. Jumping to conclusions and delusions in psychosis: relationship and response to treatment. *Schizophr Res*. 2008;98:225–231.
 42. So SH, Freeman D, Garety P. Impact of state anxiety on the jumping to conclusions delusion bias. *Aust N Z J Psychiatry*. 2008;42:879–886.
 43. Dudley REJ, Shaftoe D, Cavanagh K, et al. Jumping to conclusions in first-episode psychosis. *Early Interv Psychiatry*. 2011;5:50–56.
 44. Dudley R, Daley K, Nicholson M, et al. 'Jumping to conclusions' in first-episode psychosis: a longitudinal study. *Br J Clin Psychol*. 2013;52:380–393.
 45. Ormrod J, Shaftoe D, Cavanagh K, et al. A pilot study exploring the contribution of working memory to "jumping to conclusions" in people with first episode psychosis. *Cogn Neuropsychiatry*. 2012;17:97–114.
 46. Di Forti M, Iyegbe C, Sallis H, et al. Confirmation that the AKT1 (rs2494732) genotype influences the risk of psychosis in cannabis users. *Biol Psychiatry*. 2012;72:811–816.
 47. O'Connor JA, Wiffen BD, Reichenberg A, et al. Is deterioration of IQ a feature of first episode psychosis and how can we measure it? *Schizophr Res*. 2012;137:104–109.
 48. Stilo SA, Di Forti M, Mondelli V, et al. Social disadvantage: cause or consequence of impending psychosis? *Schizophr Bull*. 2013;39:1288–1295.
 49. Bebbington P, Nayani T. The Psychosis Screening Questionnaire. *Int J Methods Psychiatr Res*. 1995;5:11–19.
 50. Wechsler D. *Wechsler Adult Intelligence Scale - Third Edition*. San Antonio, TX: Harcourt Assessment; 1997a.
 51. Wechsler D. *Wechsler Memory Scale - Third Edition: Administration and Scoring Manual*. San Antonio, TX: The Psychological Corporation; 1997b.
 52. Kay SR, Fiszbein A, Opler LA. The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophr Bull*. 1987;13:261–276.
 53. McGuffin P, Farmer A, Harvey I. A polydiagnostic application of operational criteria in studies of psychotic illness. Development and reliability of the OPCRIT system. *Arch Gen Psychiatry*. 1991;48:764–770.
 54. American Psychiatric Association. *Task Force on DSM-IV. Diagnostic and Statistical Manual of Mental Disorders DSM-IV*. Washington, DC: American Psychiatric Association; 1994.
 55. Moritz S, Van Quaquebeke N, Lincoln TM. Jumping to conclusions is associated with paranoia but not general suspiciousness: a comparison of two versions of the probabilistic reasoning paradigm. *Schizophr Res Treatment*. 2012;2012:384039.
 56. Peters ER, Joseph SA, Garety PA. Measurement of delusional ideation in the normal population: introducing the PDI (Peters et al. Delusions Inventory). *Schizophr Bull*. 1999;25:553–576.
 57. Ben-Zeev D, Morris S, Swendsen J, Granholm E. Predicting the occurrence, conviction, distress, and disruption of different delusional experiences in the daily life of people with schizophrenia. *Schizophr Bull*. 2012;38:826–837.
 58. David AS. Why we need more debate on whether psychotic symptoms lie on a continuum with normality. *Psychol Med*. 2010;40:1935–1942.
 59. Balzan R, Delfabbro P, Galletly C. Delusion-proneness or miscomprehension? A re-examination of the jumping-to-conclusions bias. *Aust J Psychol*. 2012;64:100–107.
 60. Balzan RP, Delfabbro PH, Galletly CA, Woodward TS. Over-adjustment or miscomprehension? A re-examination of the jumping to conclusions bias. *Aust N Z J Psychiatry*. 2012;46:532–540.
 61. Lange RT, Iverson GL, Viljoen H, Brink J. Clinical validation of Canadian WAIS-III Index short forms in inpatient neuropsychiatry and forensic psychiatry. *Clin Neuropsychol*. 2007;21:434–441.
 62. Hemsley DR. The development of a cognitive model of schizophrenia: placing it in context. *Neurosci Biobehav Rev*. 2005;29:977–988.
 63. Hemsley DR. The schizophrenic experience: taken out of context? *Schizophr Bull*. 2005;31:43–53.