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Symptom-related attributional biases in schizophrenia and bipolar disorder

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ABSTRACT

Introduction: Biases in causal attributions and evidence integration have been implicated in delusions, but have not been investigated simultaneously to examine additive or multiplicative effects. It was hypothesised that paranoid delusions would correlate with self-serving and personalising biases ("defence" model of paranoia), particularly when these biases were disconfirmed.

Methods: Constrained principal component analysis was used to investigate differences between schizophrenia patients (paranoid vs. non-paranoid), bipolar disorder patients, and healthy controls, as well as to examine the extent to which psychotic symptoms could predict patterns of responding on a novel attributional bias task (Attributional Style BADE, or ASB) that requires integrating contextual information.

Results: Although no group differences were found, disorganisation and manic symptoms correlated with situation attributions and self-blame when such attributions were unsupported by the available evidence, and depression and anxiety correlated with other-person and self attributions (not situation attributions) when confirmed by the available evidence, regardless of diagnosis.

Conclusions: While group differences accounted for little variance in responses on the ASB task, a transdiagnostic association between symptoms of psychosis and the ASB task was observed. This highlights the importance of considering symptom profiles rather than diagnostic groupings when investigating cognitive biases and related non-pharmacological treatments.

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Introduction

Psychotic illness presents an intriguing area for cognitive research because cognitive biases are thought to underlie symptoms. Delusions have been of particular interest in cognitive neuropsychiatry due to their associations with measurable cognitive biases and amenability to non-pharmacological treatments such as metacognitive training (MCT; Moritz, Veckenstedt, Randjbar, Vitzthum, & Woodward, 2011). For example, the "jumping to conclusions" bias observed on probabilistic reasoning tasks is thought to underlie the hasty belief formation that produces delusions (Sanford, Lecomte, Leclerc, Wykes, & Woodward, 2013; Speechley, Whitman, & Woodward, 2010; Woodward, Munz, Leclerc,

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& Lecomte, 2009). Related to this is the bias against disconfirmatory evidence (BADE) suggested to underlie the fixedness of delusions (Riccaboni et al., 2012; Sanford, Veckenstedt, Moritz, Balzan, & Woodward, 2014; Speechley, Ngan, Moritz, & Woodward, 2012; Woodward, Moritz, Cuttler, & Whitman, 2006). Attributional biases, a domain of social cognition, have been implicated in psychosis as well (Combs & Penn, 2008), and have been framed as a special case of jumping to conclusions (Merrin, Kinderman, & Bentall, 2007).

Social cognition is impaired in schizophrenia (Mehta et al., 2013; Savla, Vella, Armstrong, Penn, & Twamley, 2013) and uniquely predicts functional outcome (Couture, Penn, & Roberts, 2006; Fett et al., 2011). Attributional bias is particularly relevant to delusions, as misattributions of events and others' actions may play a role in maintaining a delusional belief. Moreover, attributional biases are targeted in delusion treatment programs such as MCT (Moritz, Woodward, & Burlon, 2005) and social functioning treatments such as Social Cognition and Interaction Training (SCIT; Roberts et al., 2014).

Although attributional biases exist in healthy individuals, extreme/dysfunctional attributional biases may be linked to certain psychopathological conditions. For example, a self-serving bias (i.e., blaming external causes for negative events and/or assigning self-credit for positive events) is a normal cognitive bias thought to help maintain high self-esteem (Mezulis, Abramson, Hyde, & Hankin, 2004). It has been proposed that paranoia arises from an extreme self-serving bias, with a tendency to blame other people rather than the situation for negative events (i.e., a personalising bias; Bentall, Kinderman, & Kaney, 1994). By contrast, a depressive attributional bias is characterised as a high degree of self-blame for negative events and/or external attributions for positive events (Kinderman & Bentall, 1997). Some research suggests that an extreme attributional style (whether positive or negative) in bipolar disorder may predict relapse of manic/hypomanic episodes and longer time to recover during depressive episodes (Stange, Sylvia, da Silva Magalhães, Frank, et al., 2013; Stange, Sylvia, da Silva Magalhães, Miklowitz, et al., 2013).

Bentall et al. (1994) first proposed a "defence" account of paranoid delusions, suggesting that such patients possess extreme self-serving biases to defend against threats to self-esteem, which gives rise to persecutory beliefs. Subsequent studies supported the notion that patients with paranoid delusions have an extreme self-serving bias with a particular tendency for personalising blame (Kinderman & Bentall, 1996, 1997). Although more recent evidence supports a defensive attributional style underlying paranoia (e.g., Diez-Alegría, Vázquez, Nieto-Moreno, Valiente, & Fuentenebro, 2006), it may not be specific to acutely paranoid patients (Martin & Penn, 2002). Some researchers demonstrated a more specific association with a personalising bias than with a general self-serving bias (Combs et al., 2009; Diez-Alegría et al., 2006; Langdon, Ward, & Coltheart, 2010), although others suggested that personalising may be related to general severity of delusions rather than paranoia in particular (Martin & Penn, 2002). Randjbar, Veckenstedt, Vitzthum, Hottenrott, and Moritz (2011) proposed that attributional bias in paranoid patients may be better explained by a non-specific reduced sense of self-causation.

Although a substantial body of research has addressed attributional style in psychosis, previous studies have been highly hypothesis-driven, focusing almost exclusively on paranoid delusions. Given the heterogeneity of psychosis, a more data-driven approach may be more informative. Further, research in attributional bias has yet to examine the fixedness of attributions when faced with conflicting information; that is, there has been extensive

investigation of cognitive constructs of belief *formation* in the context of attributional reasoning, but not those of belief *maintenance*.

A BADE in schizophrenia has been demonstrated on delusion-neutral material, illustrating a cognitive process proposed to underlie the fixedness of delusions (Sanford et al., 2014; Speechley et al., 2012; Woodward, Moritz, & Chen, 2006; Woodward, Moritz, Cuttler, & Whitman, 2006). A BADE is typically assessed by a task in which a participant rates the plausibility of several interpretations of a given scenario, and may adjust these ratings based on accumulating evidence. For example, the statement, "Jenny can't fall asleep", may be interpreted in several ways (e.g., "Jenny is worried about her exam the next day" or "Jenny is excited about Christmas morning"); as more statements are presented (e.g., "Jenny can't wait until it is finally morning"), it becomes apparent as to which interpretation is true. An unwillingness to down-rate initially plausible interpretations (lures) that are subsequently revealed to be implausible is particularly enhanced in delusional patients (Riccaboni et al., 2012; Sanford et al., 2014; Speechley et al., 2012; Woodward, Moritz, & Chen, 2006; Woodward, Moritz, Cuttler, & Whitman, 2006).

Extreme attributional biases and an enhanced BADE have been independently observed in schizophrenia patients, but have not been examined simultaneously to check for additive/multiplicative effects on associations with delusions. Given the considerable interest in attributional biases underlying paranoia, as well as the role of a general BADE in the fixedness of delusions, it follows that this area of research should be extended to investigate the BADE in the context of attributional reasoning.

To assess attributional beliefs and their amenability, we developed the Attributional Style BADE (ASB) task, a variation of the BADE task which incorporates a standard attributional bias measure, the Internal, Personal and Situational Attributions Questionnaire (IPSAQ; Kinderman & Bentall, 1996). The present research aimed to examine (1) the cognitive operations underlying the ASB task, (2) attributional biases differing between healthy individuals, schizophrenia patients (as a whole as well as divided into paranoid and non-paranoid subgroups), and bipolar disorder patients, and (3) the components of the ASB task that are predictable from symptoms of psychotic illness. Consistent with the "defence" account of paranoia, we hypothesised that paranoid delusions would be associated with self-serving biases when such attributions were unsupported by evidence.

Methods

Participants

All analyses were drawn from a dataset of 48 schizophrenia patients, 50 bipolar disorder patients, and 58 healthy controls (total $n = 156$). Patients with bipolar disorder served as a psychiatric comparison group because of their similarity to schizophrenia patients in cognitive function, genetic and environmental susceptibility factors, stigma, and medication (Barrett, Mulholland, Cooper, & Rushe, 2009). Participants were recruited by advertisements or referred to the study by their mental health care providers. All participants provided written informed consent after a complete description of the study. Inclusion criteria were: (1) 19–55 years of age, (2) fluent in English, and (3) estimated IQ $\geq$ 70 based on the Kaufmann Brief Intelligence Test composite score (KBIT-2; Kaufman & Kaufman, 2004).

For between-groups analyses, participants were also excluded if they had a known history of a neurological condition (e.g., stroke, epilepsy, encephalitis, etc.), head injury resulting in serious concussion or loss of consciousness, or met diagnostic criteria for substance dependence. In addition to these exclusion criteria, *patients* did not qualify if they (1) did not have a diagnosis of schizophrenia, psychosis, schizoaffective disorder, schizophreniform disorder, or bipolar disorder, (2) had psychosis that was a direct consequence of substance abuse, or (3) had a concurrent psychiatric diagnosis (e.g., major depression). As confounding variables are less of a concern in correlational analyses, these exclusion criteria were somewhat relaxed for the analysis of correlations between attributional biases and symptoms. Healthy controls were excluded if they had a history of any psychiatric condition. All screening was performed by trained research staff using the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998).

Materials

Symptom ratings

Symptoms in patients were assessed with the Signs and Symptoms of Psychotic Illness scale (SSPI; Liddle, Ngan, Duffield, & Warren, 2002). The SSPI is a semi-structured interview consisting of 20 symptoms, each rated on a 5-point scale (0–4, with 0 = absent, 4 = severe), with additional subscales for delusions (guilt/worthlessness, grandiosity, paranoia, and Schneiderian themes) and hallucinations (second person auditory and Schneiderian). A score $\geq$3 on the delusions scales indicates the presence of clinically significant delusions, and therefore patients with a paranoid delusions score of 0–2 were assigned to the non-paranoid subgroup and patients scoring 3 or 4 were assigned to the paranoid subgroup where applicable.

ASB task

The ASB task is computerised (Figure 1(a,b)), with a total of 20 scenarios (10 positive, 10 negative). Like the IPSAQ, a positive or negative scenario is presented, and the participant evaluates the degree to which themselves, other people, and the situation are responsible. The ASB task features three independent rating scales with which the participant indicates his/her endorsement of each attributional locus. The participant may adjust these ratings following additional information that implies a particular cause. For example, an individual may be presented with the scenario, "Your team won an important game"; first, the individual rates on three separate scales: the degree of responsibility of themselves, other people, and the situation. The individual is then presented with information about a possible cause (e.g., "Now imagine that the other team played poorly") and may adjust his/her ratings accordingly. Therefore, three general categories of attributions emerge: (1) initial attributions (i.e., attributions rated before contextual information is provided), (2) confirmed attributions (i.e., attributions rated after contextual information is provided that matches the respective attributional locus), and (3) disconfirmed attributions (i.e., attributions rated after contextual information is provided that disconfirms the respective attributional locus). The order of scenarios and the attribution implied in the second statement were randomised for each participant; for example, the statement "your team won an important game" could be followed up by either "now imagine you played really well", "now imagine that the other team played poorly", or "now imagine

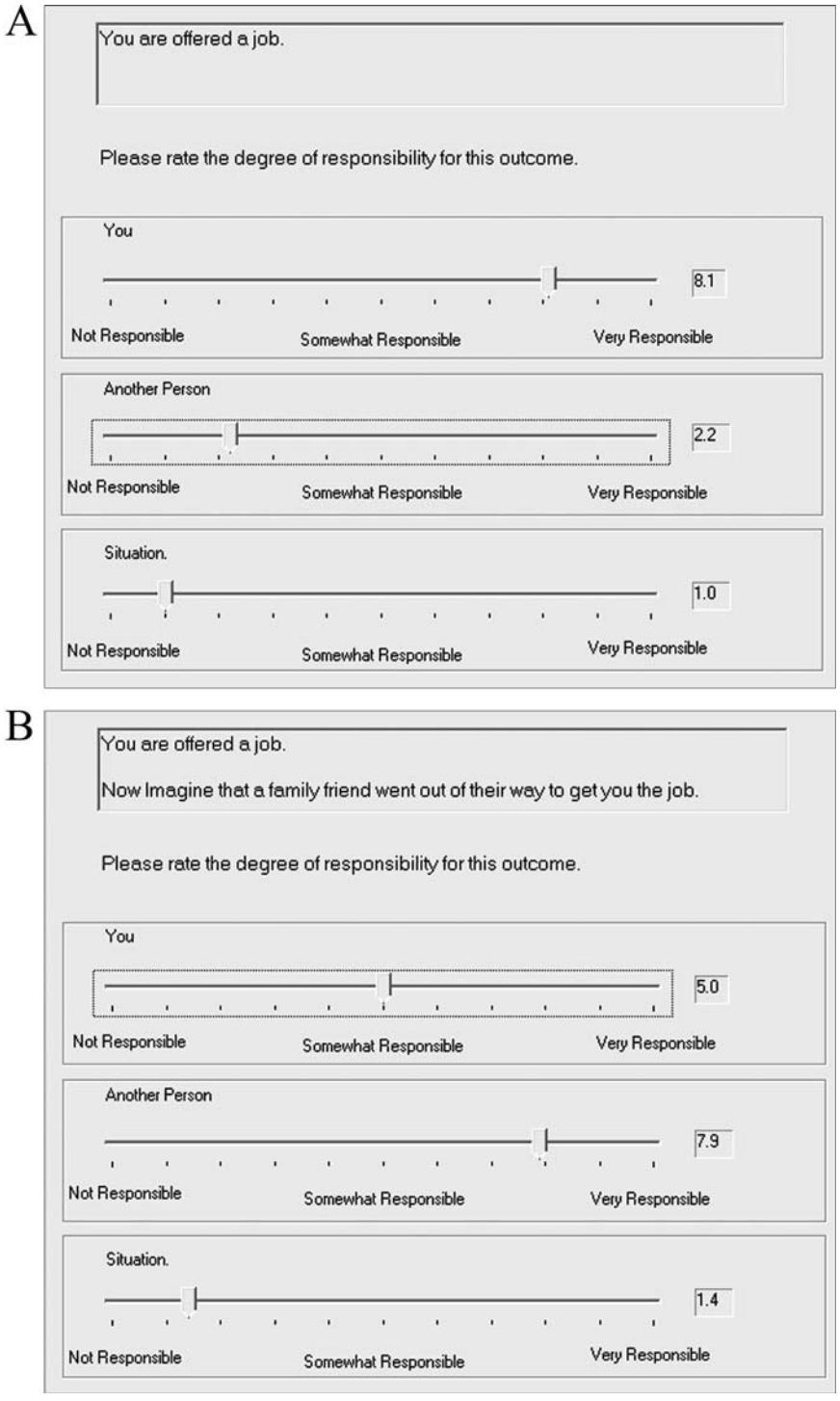

Figure 1. Screenshots of an ASB task trial. (a) A positive scenario is presented with no information about the cause of the event, and the participant uses the scales provided to rate the degree of responsibility of each attributional locus. (b) Additional information is provided that suggests a particular cause of the event, and the participant uses the scales to revise their ratings.

that the sun was in the other players' eyes the whole match". The rating for each possible attribution was made on a scale of 0 ("not responsible") to 10 ("very responsible") in increments of 0.1, with a small box to the right of each scale showing the current rating. Participants were instructed to rate the three attributions independently of each other, and were informed that they could change their ratings as little or as much as they would like after receiving additional information. Participants completed practice trials to familiarise themselves with the task before starting the experiment.

Analysis

Initial attribution ratings

To verify that the ASB task could demonstrate self-serving and personalising biases consistent with existing literature (Mezulis et al., 2004), an analysis of participants' initial attribution ratings was performed, as this part of the ASB paradigm is analogous to the IPSAQ. This was accomplished using a 2 (positive/negative scenario) × 3 (self/other/situation attribution) repeated measures analysis of variance (ANOVA). The Greenhouse–Geisser correction for degrees of freedom was applied in cases where the assumption of sphericity was violated and the correction changed statistical results.

Component structure of the ASB task

Principal component analysis (PCA) was used to investigate cognitive processes engaged in the ASB task. The number of components to retain was determined by examining the plotted eigenvalues (i.e., the scree plot), which illustrates the extent to which each component contributes a meaningful portion of variance (Cattell, 1966; Cattell & Vogelmann, 1977). Components were rotated using varimax rotation with Kaiser normalisation.

Constrained PCA

Constrained PCA (CPCA; Hunter & Takane, 2002), a statistical method that combines multivariate multiple regression with PCA, was employed to determine whether any cognitive operations engaged in the ASB task would arise from participant group differences or symptoms of psychotic illness.

The first step in CPCA (i.e., the external analysis) involves partitioning the total variability in the dependent variable (Z) into variance that can be explained by the predictor (G) and variance that cannot be explained by the predictor, producing a matrix of predicted scores (GC) and a matrix of residuals (E). This is illustrated in the regression equation:

$$Z = GC + E$$

where Z = the matrix of dependent variables, G = the matrix of independent variables, C = the weights applied to G to produce the predicted scores (GC), and E = the residuals.

The next step in CPCA (i.e., the internal analysis) is to apply PCA to GC to determine which components of Z are predictable from G. Each component extracted from GC is then correlated with the independent variables in G, producing a matrix of G loadings (the correlation coefficients) that indicate the degree to which each variable in G is related to the components extracted from GC.

In the present research, attribution ratings from the ASB task were entered into Z. In the between-groups analyses, G was comprised of indicator variables coding the

participant groups. In the symptom-based analysis, *G* was comprised of SSPI scores. Only the component structure of the respective *GC* matrices is reported, although a complete set of PCA results is available by request from the corresponding author.

Results

Cognitive operations of the ASB task

Initial attribution ratings

A 2 (positive/negative scenario) × 3 (self/other/situation attribution) repeated measures ANOVA was performed on initial attribution ratings, from the whole sample of participants ($n = 156$). Within-subject effects were observed for scenario, $F(1, 155) = 31.44$, $p < .001$, and attribution, $F(2, 310) = 12.99$, $p < .001$, as well as a scenario × attribution interaction, $F(2, 310) = 295.90$, $p < .001$.

To examine these effects, paired *t*-tests were performed to compare the ratings between positive and negative scenarios for each attribution. Self attribution ratings for positive scenarios were significantly higher than those for negative scenarios (mean positive = 6.71, SD = 1.92; mean negative = 3.67, SD = 1.80; $t(155) = 19.25$, $p < .001$). Other-person attribution ratings for positive scenarios were significantly lower than those for negative scenarios (mean positive = 4.00, SD = 1.82; mean negative = 5.98, SD = 2.00; $t(155) = 14.61$, $p < .001$). Situation attribution ratings did not differ between positive and negative scenarios (mean positive = 4.53, SD = 2.32; mean negative = 4.44, SD = 2.18; $t(155) = 0.71$, $p = .477$). These relationships are presented in Figure 2.

Component structure

The component structure of the ASB task was investigated in the full sample of participants (mean attribution ratings listed in Table 1). PCA revealed a four-component solution, accounting for 73.43% of the total variance. These components were dominated

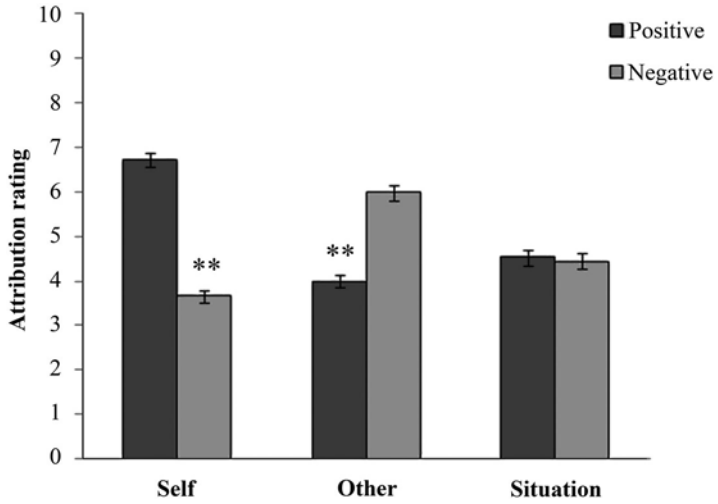

Figure 2. Mean initial attribution ratings for positive and negative scenarios (whole sample, $n = 156$). **$p < .001$.

by: (1) confirmed attributions (22.99% of the total variance), (2) initial and disconfirmed situation attributions (22.43%), (3) initial and disconfirmed other-person attributions (15.47%), and (4) initial and disconfirmed self attributions (12.61%). The loadings for each component are listed in Table 2.

Note that initial and disconfirmed attributions for each attributional locus loaded together on one component (components 2, 3, and 4), distinct from confirmed attributions (component 1). Initial and disconfirmed attributions can both be considered unsupported by the available evidence, and so are hereafter labelled "unsupported" attributions. Therefore, it appears that the 18 ASB task variables may be reduced to four dimensions, dominated by: (1) confirmed attributions, (2) unsupported situation attributions, (3) unsupported other-person attributions, and (4) unsupported self attributions.

Constrained principal component analysis

Group comparisons

Thirteen participants were excluded from the following analysis due to failure to meet inclusion criteria ($n = 8$) or missing demographic/IQ scores ($n = 5$). This resulted in a sample of 143 participants (58 healthy controls, 41 schizophrenia patients, and 44 bipolar disorder patients). Two bipolar disorder patients in this sample were experiencing paranoid delusions. The groups differed in gender distribution, $\chi^2(2) = 6.29$, $p < .05$, IQ, $F(2, 140) = 3.77$, $p = .025$, years of education, $F(2, 140) = 14.48$, $p < .001$, age, $F(2, 140) = 7.43$, $p = .001$, and socio-economic status factor score (SES), $F(2, 140) = 5.62$, $p = .004$. Post hoc comparisons using Tukey's honest significant difference revealed a significant difference in IQ between the healthy control group ($M = 106.16$, SD = 9.52) and the bipolar disorder patients ($M = 100.41$, SD = 10.69; $p = .028$), but no differences between either group and the schizophrenia patients ($M = 101.80$, SD = 13.36; ps > .10). For years of education, there was a significant difference between the healthy controls ($M = 15.92$, SD = 1.84) and both the bipolar group ($M = 14.34$, SD = 1.94; $p = .001$) and schizophrenia

Table 1. Mean attribution ratings (whole sample, $n = 156$).

	Scenario type	
Attribution	Positive	Negative
Initial ratings		
Self	6.71 (1.92)	3.67 (1.80)
Other	4.00 (1.82)	5.98 (2.00)
Situation	4.53 (2.32)	4.44 (2.18)
Confirmed		
Self	8.58 (1.70)	7.91 (2.37)
Other	7.90 (2.03)	8.06 (2.41)
Situation	7.36 (2.30)	7.37 (2.44)
Disconfirmed		
Self	5.34 (2.05)	2.74 (2.00)
Other	4.59 (2.05)	4.12 (1.96)
Situation	5.16 (2.49)	4.42 (2.49)

Notes: Initial ratings are attribution ratings provided following the first statement presented; confirmed attributions are the final ratings provided when the respective attributional locus was supported by the preceding evidence; disconfirmed attributions are the final ratings provided when the respective attributional locus was *not* supported by the evidence.
Standard deviations in parentheses.

Table 2. Component loadings for the ASB task, with four components extracted.

Attribution	Confirmed (All)	Unsupported situation	Unsupported other	Unsupported self
Other, confirmed (neg)	**0.85**	−0.11	0.27	0.03
Other, confirmed (pos)	**0.78**	−0.02	0.36	0.02
Self, confirmed (neg)	**0.75**	0.01	−0.11	0.11
Self, confirmed (pos)	**0.73**	0.01	0.10	**0.48**
Situation, confirmed (neg)	**0.71**	0.36	−0.02	−0.15
Situation, confirmed (pos)	**0.69**	**0.48**	−0.01	−0.12
Situation, disconfirmed (pos)	0.10	**0.87**	0.12	0.13
Situation, initial (pos)	0.07	**0.85**	0.18	0.19
Situation, disconfirmed (neg)	−0.02	**0.84**	0.23	0.11
Situation, initial (neg)	0.21	**0.83**	0.25	0.18
Other, disconfirmed (pos)	0.10	0.31	**0.79**	0.10
Other, initial (pos)	0.17	0.28	**0.76**	0.15
Other, disconfirmed (neg)	−0.07	0.19	**0.74**	0.23
Other, initial (neg)	**0.54**	0.01	**0.69**	0.21
Self, disconfirmed (pos)	−0.03	0.19	0.21	**0.81**
Self, initial (pos)	**0.46**	0.11	0.25	**0.69**
Self, initial (neg)	0.05	**0.50**	0.17	**0.62**
Self, disconfirmed (neg)	−0.32	**0.54**	0.20	**0.49**

Notes: pos, positive scenario; neg, negative scenario; initial, ratings provided following the first statement presented; confirmed, final ratings provided when the respective attributional locus was supported by the preceding evidence; disconfirmed, final ratings provided when the respective attributional locus was *not* supported by the evidence. The "Unsupported" Situation, Other, and Self components were specifically dominated by unsupported (i.e., initial and disconfirmed) attributions for each respective attributional locus, whereas the "Confirmed" component consisted of all confirmed attributions for all attributional loci. Component loadings ≥|.40| are displayed in bold font. Variables are sorted by absolute loadings (descending). Components are ordered by decreasing magnitude of total variance explained.

group ($M = 13.70$, SD = 2.70; $p < .001$). For years of age, healthy controls were significantly younger ($M = 29.81$, SD = 8.85) than both the bipolar group ($M = 36.55$, SD = 10.87; $p = .002$) and schizophrenia group ($M = 35.63$, SD = 9.33; $p = .010$). Finally, for SES, there was a significant difference between the schizophrenia patients ($M = 75.44$, SD = 27.80) and both the healthy controls ($M = 61.43$, SD = 17.14; $p = .004$) and bipolar disorder patients ($M = 63.91$, SD = 18.84; $p = .035$). SSPI symptom scores for the two patient groups are provided in Table 3.

To control for group differences related to demographics, attribution ratings were regressed onto the potential confounds that were found earlier. IQ, years of education, age, SES, and gender combined accounted for 4.56% of the total variance in attribution ratings. With these variables partialled out of *Z*, differences between schizophrenia patients, bipolar disorder patients, and healthy individuals accounted for only 1.39% of the remaining variance. When schizophrenia patients were divided into paranoid/non-paranoid subgroups ($n = 10$ paranoid, $n = 31$ non-paranoid), differences between the four groups accounted for only 2.38% of the variance. Therefore, no further between-group analyses were performed.

Symptom-based analysis

As group differences in attribution ratings were negligible, schizophrenia and bipolar disorder patients were combined to form a more heterogeneous sample of patients likely to have experienced symptoms measured on the SSPI scale. Some exclusion criteria (i.e., substance use, history of head injury, comorbid psychiatric diagnosis) were relaxed and eight patients were added. In addition, five patients who had been excluded in the previous analysis due to missing demographic/IQ scores were added. A total of six bipolar disorder

Table 3. Mean SSPI symptom scores for patients included in group comparisons analysis (standard deviations in parentheses).

Symptom	Schizophrenia ($n = 41$)	Bipolar disorder ($n = 44$)
Anxiety	1.27 (0.98)	1.30 (1.11)
Depression	0.98 (1.23)	1.02 (1.19)
Anhedonia	1.05 (1.20)	0.98 (1.09)
Elated mood	0.41 (0.81)	0.73 (1.09)
Insomnia	0.90 (1.22)	1.27 (1.17)
Somatic complaints	0.17 (0.59)	0.09 (0.29)
Delusions—general	2.07 (1.33)	0.86 (1.19)
Paranoid delusions	1.39 (1.48)	0.55 (1.00)
Guilt/worthlessness	0.46 (0.90)	0.16 (0.57)
Grandiose delusions	0.90 (1.22)	0.36 (0.92)
Schneiderian delusions	0.93 (1.29)	0.25 (0.75)
Hallucinations—general	1.54 (1.82)	0.32 (0.96)
Second person auditory hallucinations	1.00 (1.70)	0.25 (0.87)
Schneiderian hallucinations	0.90 (1.62)	0.23 (0.86)
Attentional impairment	1.34 (0.99)	1.27 (0.90)
Disorientation	0.07 (0.26)	0.00 (0.00)
Overactivity	0.83 (1.05)	1.07 (1.00)
Underactivity	1.15 (1.17)	0.86 (0.93)
Flattened affect	1.29 (1.01)	0.59 (0.90)
Inappropriate affect	0.12 (0.51)	0.07 (0.33)
Pressured speech	0.32 (0.72)	0.34 (0.64)
Poverty of speech	0.41 (0.74)	0.14 (0.46)
Disordered thought	0.34 (0.82)	0.07 (0.33)
Peculiar behaviour	0.20 (0.60)	0.05 (0.21)
Irritability/hostility	0.41 (0.71)	0.30 (0.51)
Impaired insight	1.29 (1.19)	0.77 (1.03)

patients and seven schizophrenia patients were added back into the sample, resulting in a group of 98 patients (50 bipolar patients, 48 schizophrenia patients). *G* was comprised of all general symptom scores and subscales for delusions and hallucinations (26 independent variables; symptom scores provided in Table 4).

SSPI scores accounted for 28.68% of the total variance in *Z*. PCA of the SSPI-predicted scores revealed a two-component solution accounting for 62.87% of the variance in *GC* (32.65% and 30.22% from components 1 and 2, respectively), dominated by: (1) unsupported self attributions for negative scenarios, as well as unsupported situation attributions for positive and negative scenarios, and (2) initial and confirmed other-person attributions and confirmed self attributions. Component 1 was highly correlated with disordered thought, motor overactivity, inappropriate affect, disorientation, and pressured speech. Component 2 was positively correlated with depression and anxiety. *GC* component loadings and *G* loadings are listed in Table 5.

As paranoia was the primary symptom of interest, comparisons between paranoid ($n = 13$; 2 bipolar disorder and 11 schizophrenia patients) and non-paranoid patients ($n = 85$) on indices of the two components were performed. Applying a component loading threshold of .40, component 1 was indexed as the sum of initial and disconfirmed self attribution ratings (negative scenarios only) and initial and disconfirmed situation attribution ratings (positive and negative scenarios). Similarly, component 2 was indexed as the sum of initial other-person attribution ratings (positive and negative scenarios), confirmed other-person attribution ratings (negative scenarios only), and confirmed self attribution ratings (positive scenarios only). Neither measure differed between paranoid and non-paranoid patients (both $ps > .06$; see Figure 3).

Table 4. Mean SSPI symptom scores for all patients included in symptom-based analysis ($n = 98$; standard deviations in parentheses).

Symptom	Severity
Anxiety	1.30 (1.06)
Depression	1.06 (1.23)
Anhedonia	1.02 (1.12)
Elated mood	0.56 (0.96)
Insomnia	1.10 (1.21)
Somatic complaints	0.14 (0.45)
Delusions—general	1.40 (1.42)
Paranoid delusions	0.89 (1.28)
Guilt/worthlessness	0.27 (0.71)
Grandiose delusions	0.62 (1.11)
Schneiderian delusions	0.60 (1.15)
Hallucinations—general	0.90 (1.56)
Second person auditory hallucinations	0.61 (1.39)
Schneiderian hallucinations	0.54 (1.30)
Attentional impairment	1.33 (0.93)
Disorientation	0.03 (0.17)
Overactivity	0.95 (1.02)
Underactivity	0.98 (1.05)
Flattened affect	0.88 (0.99)
Inappropriate affect	0.12 (0.56)
Pressured speech	0.35 (0.76)
Poverty of speech	0.24 (0.59)
Disordered thought	0.27 (0.74)
Peculiar behaviour	0.12 (0.44)
Irritability/hostility	0.40 (0.68)
Impaired insight	1.06 (1.15)

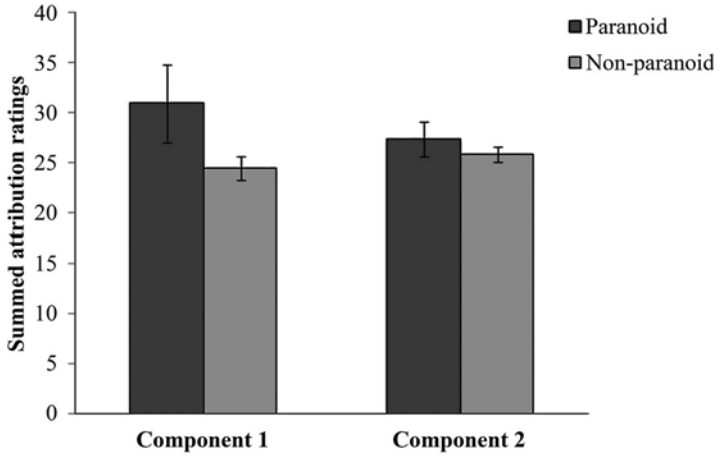

Figure 3. Mean summed scores for components 1 and 2, comparing paranoid with non-paranoid patients (both groups consist of a mix of schizophrenia and bipolar disorder patients). Component 1 score = sum of mean initial and disconfirmed self ratings (negative scenarios only) and initial and disconfirmed situation ratings (positive and negative scenarios); mean score for paranoid group = 30.95; mean score for non-paranoid group = 24.46. Component 2 score = sum of initial other ratings (positive and negative scenarios), confirmed other ratings (negative scenarios only), and confirmed self ratings (positive scenarios only); mean score for paranoid group = 27.43; mean score for non-paranoid group = 25.82. Neither measure differed significantly between groups.

Table 5. *GC* component loadings and *G* loadings from CPCA in patients ($n = 98$; Z = attribution ratings, G = SSPI).

GC			*G*		
Attributions	1	2	Symptoms	1	2
Self, initial (neg)	**0.56**	0.13	Disordered thought	**0.57**	0.01
Situation, disconfirmed (neg)	**0.52**	0.06	Overactivity	**0.46**	0.13
Situation, initial (neg)	**0.50**	0.10	Inappropriate affect	**0.46**	−0.06
Self, disconfirmed (neg)	**0.45**	−0.10	Disorientation	**0.41**	0.07
Situation, initial (pos)	**0.44**	0.02	Pressured speech	**0.40**	0.15
Situation, disconfirmed (pos)	**0.43**	0.10	Depression	−0.18	**0.44**
Other, initial (neg)	0.12	**0.49**	Anxiety	−0.08	**0.41**
Other, confirmed (neg)	−0.05	**0.49**	Peculiar behaviour	0.39	0.13
Other, initial (pos)	0.25	**0.42**	Elated mood	0.34	0.30
Self, confirmed (pos)	0.04	**0.40**	Underactivity	−0.33	−0.25
Self, confirmed (neg)	0.01	0.39	Attentional impairment	0.29	−0.11
Other, confirmed (pos)	−0.06	0.39	Paranoid delusions	0.29	0.09
Other, disconfirmed (pos)	0.21	0.37	Grandiose delusions	0.29	0.16
Situation, confirmed (pos)	0.08	0.34	Delusions—general	0.29	0.05
Situation, confirmed (neg)	0.08	0.27	Schneiderian delusions	0.27	−0.05
Self, initial (pos)	0.20	0.20	Insomnia	−0.26	0.15
Other, disconfirmed (neg)	0.21	0.12	Impaired insight	0.23	−0.02
Self, disconfirmed (pos)	0.20	0.02	Guilt/worthlessness	0.11	0.34
			Poverty of speech	0.23	−0.34
			Hallucinations—general	0.21	0.18
			Anhedonia	−0.19	0.11
			Second person auditory hallucinations	0.15	0.01
			Irritability/hostility	0.09	0.17
			Flattened affect	0.05	−0.16
			Somatic complaints	−0.08	−0.13
			Schneiderian hallucinations	0.04	0.01

Notes: Variables are sorted by absolute loadings (descending). Loadings ≥ |.40| are displayed in bold font. Components are ordered by decreasing magnitude of total variance explained. Pos, positive scenario; neg, negative scenario; initial, ratings provided following the first statement presented; confirmed, final ratings provided when the respective attributional locus was supported by the preceding evidence; disconfirmed, final ratings provided when the respective attributional locus was *not* supported by the evidence. *G* loadings are correlation coefficients representing the relationships between component scores and SSPI symptoms.
The *GC* and *G* tables complement each other and must be interpreted together.

Discussion

Previous research on attributional bias has demonstrated extreme self-serving and personalising biases in patients with paranoid delusions, which has been proposed to reflect a defence mechanism generating persecutory beliefs. Moreover, a BADE is associated with delusions in schizophrenia, and may underlie the fixedness of beliefs. However, existing measures of attributional bias engage only one aspect of this cognitive bias (belief *formation*) and fail to consider another aspect that is important for delusions (belief *maintenance*). The present research attempted to address this shortcoming with use of the ASB task, which combines a standard attributional bias paradigm (IPSAQ) with a test of hypothesis judgement used in delusions research (BADE).

A four-component structure was obtained from the ASB task. Three of these components reflected common variance among unsupported attributions, which was distinct for each of the three attributional loci. The shared variance reflected in each "unsupported" component cannot be fully explained by positive correlations between participants' initial and final ratings, as this overlap was specific to *disconfirmed* attributions; a "confirmed" attributions component, which was dominated by ratings that were made

after receiving evidence that supported the respective attribution, emerged independently. These components may reflect similar constructs observed in analyses of the original BADE task, which exhibits a two-component structure (Sanford et al., 2014; Speechley et al., 2012). One of these is a so-called evidence integration component, which is dominated by disconfirmed interpretations and negatively loaded confirmed interpretations. The second component is dominated by interpretations that are typically rated high ("lure" interpretations and confirmed interpretations), and has been termed "conservatism" because it appeared to be driven by individuals providing low ratings for these interpretations when it was discovered. Thus, the three unsupported attribution components in the ASB task could reflect attribution-specific evidence integration, and the confirmed attributions component in the ASB task may be akin to the conservatism component in the original BADE task.

Participant group differences accounted for a negligible amount of variance in the ASB task; therefore, the component solution observed when symptom scores were entered into *G* can be considered transdiagnostic. The first component in the SSPI-predicted solution was dominated by unsupported self attributions for negative scenarios and situation attributions for positive and negative scenarios, which correlated with some symptoms of disorganisation (disordered thought, inappropriate affect, and disorientation) and mania (motor overactivity and pressured speech). The second component was dominated by initial other-person attributions as well as confirmed other-person and self attributions, and correlated with depression and anxiety. The absence of a strong contribution of delusions to the SSPI-predicted solution was unexpected, given previous research showing delusions being associated with attributional biases and a BADE. It has been suggested that an enhanced BADE in delusional patients depends on the strength of the evidence presented (Balzan, Delfabbro, Galletly, & Woodward, 2012; Balzan, Delfabbro, Galletly, & Woodward, 2013); however, unlike the standard BADE paradigm, the ASB task does not use explicit "lure" interpretations (i.e., interpretations of a scenario that initially seem most plausible, and then are disproved by subsequent evidence), as it was assumed that participants would implicitly consider the self attribution as the most plausible for positive scenarios, and external attributions the most plausible for negative scenarios. Although a self-serving bias was evident in participants' initial attribution ratings, it is possible that the initial presentation of the scenario must imply a specific cause in order for individuals to be led into a firm belief and for a BADE to be detectable.

The attributions dominating component 1 in the SSPI-predicted solution were those that tended to be rated low, though it is not clear why these would be correlated with symptoms of disorganisation (disordered thought, inappropriate affect, and disorientation) and mania (overactivity and pressured speech). The involvement of situational attributions could reflect an inability to connect the socially relevant information provided with the attribution it is meant to imply, consistent with previous research suggesting that the ability to use contextual information to make causal inferences in social cognition tasks is impaired in schizophrenia and bipolar disorder (Baez et al., 2013). The shared variance between situational attributions and self-blame is seemingly contradictory; however, self-blame is a generally dysfunctional cognitive bias which, although normally associated with depression, may also be associated with high general psychopathology (Mizrahi, Addington, Remington, & Kapur, 2008). The high contribution of self-blame is consistent with the notion of this bias being broadly exhibited in severe mental illness, despite the fact

that it was not specifically correlated with guilt or depression. Although it is surprising to find self-blame rather than self-credit being correlated with symptoms of mania, it is worth noting that this subset of manic symptoms did not include inflated self-esteem, as suggested by the low contribution of grandiose delusions.

Component 2 revealed a contrasting pattern to that of component 1: initial other-person attributions dominated the component (as opposed to initial self and initial situation attributions) as well as confirmed—rather than disconfirmed—attributions (excluding confirmed situation attributions). As this component did not exhibit high loadings from situation attributions, it may reflect a tendency to attribute the cause of an event to human actions rather than uncontrollable circumstances. The correlations between this component and depression and anxiety were unexpected, although the population under investigation did not consist of patients with a primary diagnosis of depression. It is possible that the relationship between attributional style and depression in the context of psychotic illness may differ from that in major depression, which is generally associated with self-blame for adverse events. Moreover, symptoms that are typically linked to depressed mood, such as guilt and anhedonia, did not exhibit high loadings on this component, suggesting that this relationship was specific to symptoms of low mood and anxiety rather than depression or negative symptoms as a whole.

Components obtained from unconstrained PCA are not maximally predictable from the independent variables of interest, and as such, the component solution obtained from the SSPI-predicted scores was strikingly different from that of observed ASB scores. This does not necessarily imply that the independent variable is a poor predictor of task performance, but rather that the primary *dimensions* of the dependent variable are not predictable from the independent variable. This is important to note, given that such components often form the subscales of standard behavioural assessments; that is, one might not find a significant correlation between an independent variable of interest and the standard dimensions of a given assessment, even though this variable may have a meaningful relationship with some other aspect of the assessment.

Limitations

The present research may not fully explain the cognitive mechanisms underlying the ASB task. Other social cognitive domains, such as social perception (i.e., ability to identify social roles, societal rules, and social context), may affect participants' ability to use the task content to form conclusions about an event. In order to fully understand the cognitive processes being engaged, future research should investigate relationships between responses on the ASB task and other social cognition tasks.

The clinical samples may have been unusually high-functioning compared with typical schizophrenia and bipolar disorder patients in the general population. Notably, schizophrenia patients' IQ was not significantly different from that of healthy controls, and IQ was lowest in bipolar disorder patients. It is possible that the sample's low severity precluded strong symptom-related cognitive biases; however, it is also worth noting that the BADE has been associated with delusion proneness in non-clinical samples, independent of neurocognitive functions (e.g., Woodward, Buchy, Moritz, & Liotti, 2007).

Results from PCA and regression are sensitive to idiosyncrasies of the sample under investigation, and so replications in larger and varied samples are necessary to assess

the generalisability of our results. Minimum sample size recommendations for good recovery of population factors in PCA and factor analysis vary widely (MacCallum, Widaman, Zhang, & Hong, 1999). Further complicating this is the possibility that typical recommendations for unconstrained PCA may not apply to constrained PCA. For the unconstrained PCA in the present analysis, the *n* (participants) to *p* (variables) ratio was 8.67 (156:18), which is above the minimum recommended by some authors (Cattell, 1978; Gorsuch, 1983) but not others (Everitt, 1975). In multiple regression (performed in the external analysis of CPCA), there is a known risk of an inflated R^2 value with a large number of independent variables; this effect could have arisen in the symptom-based CPCA, where *G* consisted of 26 variables. However, the variance of interest was in the subsequent reduction of the predicted scores (*GC*) into their component structure and its correlations with each of the variables in *G*, not in the total percentage of predictable variance. Nevertheless, our statistical power may have been insufficient for detecting group differences between paranoid ($n = 10–13$) and non-paranoid patients.

Conclusions

Patients with certain symptoms of disorganisation and/or mania may be more likely to attribute the cause of an event to uncontrollable circumstances (but with a tendency to exhibit self-blame as well), whereas patients with low mood and/or anxiety are more likely to attribute the cause to human actions. However, the ASB task may not fully engage the cognitive operations thought to be involved in the maintenance of delusional beliefs.

The present research has shed light on largely overlooked symptoms in attributional bias research. Evidently, attributional bias warrants further study in the context of psychosis, particularly using a multivariate symptom-based approach rather than a diagnosis-based approach. The results also suggest that cognitive therapy that focuses on attributional biases should be tailored to individuals on a case-by-case basis rather than attempting to apply one approach to all patients with a given diagnosis. Future research with the ASB task and other cognitive measures will further our understanding of context-dependent causal inferences and the fixedness of attributional biases.

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