

## Source monitoring biases and auditory hallucinations

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*Introduction.* Previous source monitoring studies on schizophrenia reported an association between external source misattribution and hallucinations, but this is often not replicated. This inconsistency may be attributable to a failure in accounting for guessing parameters when computing source monitoring biases.

*Methods.* Fifty-one patients and 20 healthy controls were required to recall the source of items originating from external (computer and experimenter) or internal (the subject) sources. When statistically determined criteria were met, the appropriate counts of false positives were entered as covariates in the statistical analyses (analysis of covariance; ANCOVA) to exclude guessing from source monitoring bias measures.

*Results.* When comparing patients to controls, impairments on item recognition and source discrimination were observed. When comparing patient groups split on hallucinations, a bias towards attributing self-generated items to an external source was observed. A group difference on the externalisation bias was absent when the sample was split on delusions.

*Conclusions.* A bias towards attributing self-generated items to an external source was associated with hallucinations. This ANCOVA methodology is recommended for source monitoring studies investigating group differences, and suggests that previously reported null results may be attributable to a failure in separating guessing and source monitoring measures.

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Fundamental to an understanding of hallucinations in schizophrenia is the study of inner/outer confusions (Fowler, 2000). This relationship is most apparent when considering auditory hallucinations, as these can be conceptualised as the assignment of internally generated mental episodes to an external source. Originally referred to as reality monitoring (Johnson, Hashtroudi, & Lindsay, 1993), the study of inner/outer confusions in memory has recently been employed to investigate the cognitive underpinnings of schizophrenia, typically under the rubric of source monitoring (e.g., Brébion et al., 2000; Keefe, Arnold, Bayen, McEvoy, & Wilson, 2002; Moritz, Woodward, & Ruff, 2003; Moritz, Woodward, Whitman, & Cuttler, 2005; Morrison & Haddock, 1997; Vinogradov et al., 1997).

In source monitoring studies, impairment in source discrimination and item recognition are consistently observed (Bentall, Baker, & Havers, 1991; Keefe et al., 2002; Moritz & Woodward, 2002; Moritz et al., 2003; Moritz et al., 2005; Vinogradov et al., 1997; Woodward, Menon, Hu, & Keefe, 2006). In contrast, an association between hallucinations and a tendency to misremember an internally generated event as originating from an external source is sometimes reported for schizophrenia patients (Bentall et al., 1991; Brébion et al., 2000; Ditman & Kuperberg, 2005) and for healthy subjects who report hearing voices (Larøi, van der Linden, & Marczewski, 2004), but failures to replicate this are also common (Keefe et al., 2002; Keefe, Poe, McEvoy, & Vaughan, 2003; Morrison & Haddock, 1997; Seal, Crowe, & Cheung, 1997; Vinogradov et al., 1997).

We have identified three factors that may have contributed to these negative findings and inconsistencies with respect to the association between hallucination and the “externalisation bias” (for other theoretical and methodological considerations see Larøi & Woodward, 2007). The first factor is related to number of items per source. Except for one of these aforementioned studies, the number of items per source did not exceed 20, and in the one study that utilised more than 20 items per source, the 24 test items were separated into three levels of emotional valence, resulting in only 8 items per source condition. This concern is particularly pronounced when specific error types (e.g., externalisations and internalisations) are the measures of interest, because internalisation errors are relatively rare and may be affected by a floor effect, thereby restricting the range of scores. In the current study we increased the number of items for each source in order to allow an increased rate of internalisation errors.

The second factor is that previous negative results may have been affected by deficiencies in manipulation strength; that is to say, deficiencies in the distinction between internally and externally generated events with respect to cognitive cues. Studies for which cognitive effort was manipulated reported increases in the association between externalisations and hallucinations for high-effort items (Bentall et al., 1991; Larøi et al., 2004), and it is presumably

the generation of cognitive events that contributes to this distinctiveness (Johnson et al., 1993). In the present study we enhanced the distinctiveness of internally and externally generated items by increasing cognitive effort for the internally generated items.

The third factor affecting interpretation of most previous studies is the influence of guessing strategies. One example (for many others see Batchelder & Riefer, 1990) is that when subjects notice that they are recognising too few items from the (less memorable) external source, they tend to compensate by increasing the number of external-source guesses (Batchelder, Hu, & Riefer, 1994). Therefore, in order to accurately measure source parameters, increases in strategic guesses must be excluded. If such confounds are not properly accounted for, inaccurate conclusions can be reached; for example, a hallucinating-group externalisation bias for recognised items may be hidden by a comparison-group increase in external-source guesses for unrecognised items. Multinomial modelling has been used in the past to separate guessing and source discrimination measures (e.g., Keefe et al., 2002, p. 53); however, the flexibility, feasibility and interpretability of multinomial models for this area of study are limited by a number of factors (see Menon & Woodward, 2007; Woodward, Menon, et al., 2006). Instead, for this purpose we used analysis of covariance (ANCOVA), with the appropriate count of false positives entered as covariates when statistically determined criteria were met.

In accordance with the aforementioned literature, we expected to observe impairment in source discrimination and item recognition for people with schizophrenia. In addition, we expected hallucinations to be associated with an externalisation bias, but not an internalisation bias. Two external sources were included as a control, to ensure that inner/outer confusion is not simply due to generalised source confusion. Thus, hallucinations were not expected to be associated with external source confusions for recognised items. In addition, due to previous work that suggests a possible association between delusions and source misattributions (Johns, Gregg, Allen, & McGuire, 2006; Woodward, Menon, et al., 2006), and to ensure the specificity of an externalisation bias to hallucinations and not delusions, we also split the sample on delusions and compared source monitoring biases across these groups in an independent set of analyses.

## METHOD

### Participants

Fifty-one inpatients (37 males, 14 females) diagnosed with schizophrenia or schizoaffective disorder were recruited from Riverview Hospital and the Forensic Psychiatric Services Commission, Coquitlam, Canada. Diagnosis

was determined by ward psychiatrists according to DSM-IV criteria. The patient group was diagnosed as follows: paranoid ( $n = 28$ ), undifferentiated ( $n = 15$ ), and schizoaffective ( $n = 8$ ). Twenty control subjects (9 males, 11 females), consisting of hospital staff, were also recruited for the study, and were matched to the patient groups on age and social status determined using the highest parental occupation and education level (Hollingshead & Redlich, 1958). Table 1 provides a summary of all socio-demographic sample characteristics. Participants were excluded if their IQ was less than 70, if they had a history of primary or acquired brain damage (e.g., stroke, encephalitis) or traumatic head injury (e.g., with a loss of consciousness for more than 10 minutes), or if they tested HIV positive. For all but four of the subjects their first language was English, but all read and spoke English proficiently, and eyesight was 20/50 or better. At the time of testing, all patients were receiving atypical antipsychotic medication (chlorpromazine equivalent dosage in mg:  $M = 663.16$ ,  $SD = 546.89$ ).

The hallucinating group comprised fifteen patients who were experiencing second-person auditory hallucinations (e.g., hallucinations of command). The delusional group comprised 34 patients who were currently acutely delusional. In accordance with the expected co-occurrence of delusions and hallucinations (Liddle, 1987), 14 patients were concurrently experiencing both delusions and hallucinations, so were included in the experimental group for both analysis types (i.e., investigations of hallucinations and delusions). Note that delusional and hallucinating groups were never directly compared within one statistical model, as this violates the assumption of group independence underlying analysis of variance (ANOVA).

## Materials

*Psychopathology.* Psychopathology was assessed using the Signs and Symptoms of Psychotic Illness Rating Scale (SSPI; Liddle, Ngan, Duffield, Kho, & Warren, 2002). The SSPI is a 20-item scale, which can be completed after a 25–30 minute semistructured interview with 15 direct questions about symptoms. The severity of each item is rated on a scale ranging from 0 to 4. The SSPI is criterion referenced, providing specific examples of behaviour which correspond to severity levels for each item. Item 7 from the SSPI was used to quantify the presence/absence of delusions. A rating of 3 (definite delusions, but the delusional beliefs do not have a pervasive influence on thinking or behaviour) or 4 (definite delusions, which have pervasive influence on thinking and/or influence observable behaviour) warranted classification into the delusional group. Item 8.2 from the SSPI (second-person auditory hallucinations) was used to quantify hallucinations. Due to decreased frequency, and the tendency of patients to underreport hallucinations, any rating on this item led to classification into the

TABLE 1  
Sociodemographic characteristics of the samples: means, with standard deviations in parentheses

	Healthy controls (n = 20)	All schizophrenia patients (n = 51)	Hallucinating patients (n = 16)	Nonhallucinating patients (n = 35)	Delusional patients (n = 34)	Nondelusional patients (n = 17)
Age	40.00 (11.46)	37.24 (9.46)	35.94 (10.25)	37.83 (9.18)	37.26 (9.56)	37.18 (9.55)
Social status	40.75 (15.26)	36.07 (14.95)	33.33 (16.50)	37.48 (14.19)	34.93 (16.25)	38.50 (11.90)
Education	15.05 (2.96)	11.73 (2.21)***	12.88 (2.90)	11.20 (1.61)†	11.91 (2.54)	11.35 (1.32)
IQ estimate	110.22 (8.84)	98.25 (8.62)***	98.81 (8.51)	98.00 (8.78)	98.03 (8.31)	98.71 (9.45)
Sex	11 F; 9 M	14 F; 37 M*	3 F; 13 M	11 F; 24 M	8 F; 26 M	6 F; 11 M
Length of illness	n/a	16.24 (8.37)	14.70 (8.86)	17.15 (8.87)	16.23 (8.45)	16.27 (8.54)
Delusions	n/a	2.49 (1.52)	3.31 (1.01)	2.11 (1.57)††	3.47 (0.51)	0.53 (0.72)§§§
Hallucinations	n/a	1.27 (1.69)	3.31 (0.87)	0.34 (1.00)†††	1.79 (1.81)	0.24 (0.66)§§
Thought disorder	n/a	1.04 (1.11)	1.50 (1.21)	0.83 (1.01)†	1.32 (1.17)	0.47 (0.72)
Flat affect	n/a	0.88 (1.16)	0.75 (1.13)	0.94 (1.19)	0.71 (1.00)	1.24 (1.39)
Poverty of speech	n/a	0.43 (1.01)	0.25 (0.77)	0.51 (1.09)	0.24 (0.74)	0.82 (1.33)
Underactivity	n/a	0.63 (0.96)	0.56 (0.96)	0.66 (0.97)	0.50 (0.96)	0.88 (0.93)

\* $p < .05$ , schizophrenia patients vs. controls; \*\*\* $p < .001$ , schizophrenia patients vs. controls; † $p < .05$ , hallucinating patients vs. nonhallucinating patients; †† $p < .01$ , hallucinating patients vs. nonhallucinating patients; ††† $p < .001$ , hallucinating patients vs. nonhallucinating patients; § $p < .01$ , delusional patients vs. nondelusional patients; §§ $p < .001$ , delusional patients vs. nondelusional patients.

hallucinating group; this included a rating of 1 (vague descriptions of hallucinations), or higher.

*Source monitoring task.* The basic stimuli consisted of 100 common English nouns. For 75 of the nouns, a clue was generated that hinted at its meaning. For example, for the noun ‘dog’, an appropriate clue would be ‘a four-legged animal that barks’. Each clue cued only one of the nouns in the experiment, and did not include any of the words used as critical nouns. For the 75 nouns for which a clue was generated, 25 were assigned to each of the self-, computer- or experimenter-generated conditions. For the 25 self-generated nouns, in order to increase the cognitive effort associated with these items, the letters were jumbled by randomly selecting any one of the letters, and displacing it to any location in the word (with the exception of the location in which it originated). The 25 remaining nouns were reserved to be presented as new stimuli in the recognition condition. The nouns were matched across experimenter-, computer-, self-generated, and new conditions on word length (ranged from 4 to 10 letters, mode: 6 letters) and Hyperspace Analogue to Language (HAL) frequency (Balota et al., 2002). The order of the trials was randomly determined, and the same random order was used for all subjects.

## Procedure

Stimuli were presented on a 14 inch monitor, using Superlab version 2.01 (Cedrus, 1999) for Windows. All stimuli were presented against a white background in Times New Roman 30 point bold font. At the onset of every trial, a clue was presented. For self-generated trials, the clue remained on the screen until the subject had finished reading the clue aloud. Following this, the experimenter pressed a key to display the “jumbled word” puzzle, which the subject was required to solve based on the provided clue that remained on the screen. The subject was required to vocalise the solution. The experimenter pressed the “C” key if the subject correctly solved the puzzle, and the “X” key if they did not. The experimenter simply repeated the clue displayed on the computer screen when subjects experienced difficulty solving the word puzzles.

For the experimenter- and computer-generated trials, in order to match the self-generated trials, the clue, but not the answer, was presented visually. In addition, both the clue and the answer were presented in the auditory modality for these trials. Subjects were instructed to attend carefully to the solutions provided by the experimenter or computer, and to say “OK” when they were ready to move on to the next trial. For experimenter-generated trials, the experimenter read the clue, and said the solution, immediately on

presentation of the visual display, and subsequently advanced the screen to the next trial by pressing the “C” key when signalled by the subject to do so. For computer-generated trials, the clue and answer were provided by a digital sound file of a recorded voice immediately on presentation of the visual display, and the experimenter advanced the screen to the next trial by pressing the “C” key when signalled by the subject to do so. This parallel presentation of clue and solution for experimenter- and computer-generated items was designed to minimise self-generation of the response on these trials. Although subjects were instructed not to repeat the answer provided by the computer or the experimenter, if this occurred, the experimenter instead advanced the screen display using the “X” key.

The experimenter’s “C” or “X” keypress was followed by a 250 ms blank screen. All “X” coded items were excluded from analyses of the recall trials (controls: < .5% of trials for all sources; patients: 1.9%, 2.3%, and .6% of trials for patients for the experimenter, subject, and computer sources, respectively). A practice experiment with nine trials, three from each condition, preceded the main experiment, and was repeated as necessary to ensure a thorough understanding of the procedure.

The source memory test followed the puzzle solving session after approximately 20 minutes of carrying out an unrelated nonverbal task. Subjects were asked to respond “old” or “new” to each word (presented verbally by the experimenter), and to indicate for old words whether it was a word that they, the computer or the experimenter said.

*Strategy of data analysis.* The goal of our data analysis methodology was to factor guessing strategies out of source-discrimination processes when comparing source biases across groups, while still measuring source-specific externalisation and internalisation biases. This goal was achieved using analysis of covariance (ANCOVA) when appropriate. Otherwise, analysis of variance (ANOVA) was used.

The covariates used were estimates of guessing rates that may have contributed to the class of responses of interest. The guessing rates were estimated by the number of trials on which the subject produced the responses of interest for new items (false positive rate). Thus, for measures of recognition, the total count of false positive responses (i.e., experimenter, computer, and self responses to new items) was used as a covariate, because these guessing rates can be assumed to contribute to the recognition measures, just as they contributed to the false positives. False positive errors are typically *negatively* correlated with source discrimination scores, and this was replicated in the current data. This indicates that high scores on source discrimination are not the result of a high guessing rate; therefore, a downward adjustment is not appropriate, rendering the ANCOVAs invalid.

Thus, ANOVAs were used as opposed to ANCOVAs for analyses of source discrimination.

For measures of the externalisation bias, the count of experimenter and computer responses to new items was used as a covariate. For measures of the internalisation bias, the count of self responses to new items was used as a covariate. Finally, for measures of confusion of external sources, the count of experimenter and computer responses to new item was used as a covariate. If a covariate did not reach significance, it was not included in the model, and this is reported. Significance of covariates were tested at the level of  $p < .05$ , one tailed. A one-tailed test was used to test the significance of covariates due to the requirement for a positive relationship as mentioned above. All other statistical tests were two-tailed. All reported means are reported as covariate adjusted if the covariate was significant; otherwise, the unadjusted means are reported.

All analyses were carried out using SPSS 14.0 for Windows as mixed ANCOVAs, with group as a between-subjects factor, source as a within-subjects factor, and the appropriate count of false positives as a covariate. Each analysis included assessment of the group  $\times$  source interaction. If significant, the effect of interest could be studied separately for each source, and the appropriate covariate for that specific source. However, the group  $\times$  source interaction was not significant for any of the analyses, greatly reducing the complexity of the reported results, allowing group comparisons to be reported collapsed over source. The sphericity assumption was checked, and was not met for one analysis (i.e., the source discrimination analysis for hallucinations). However, using the adjusted degrees of freedom for this analysis did not impact the conclusions, so the unadjusted degrees of freedom are reported for all analyses.

### Homogeneity of regression coefficients

A fundamental assumption underlying the application of ANCOVA is homogeneity of regression coefficients (Pedhazur, 1982, p. 497). We tested this assumption for all reported analyses, using the method described by Pedhazur (1982, pp. 516–517), and found that the homogeneity of regression coefficients assumption was met for all analyses.

*Dependent variables.* In the following we list how the dependent variables were computed for each analysis type.

- Item recognition: the number old items correctly labelled old (i.e., the number old items falling into the attribution class old).



- Source discrimination: the number of old items attributed to the correct source (i.e., the number of experimenter-, computer-, and self-generated items falling into those respective attribution classes).
- Internalisation bias: the number of old items originating from the experimenter or computer, but attributed to the self (i.e., the number of experimenter and computer generated items falling into the self attribution class).
- Externalisation bias: the number of old items originating from the self, but attributed to either the experimenter or computer (i.e., the number of self-generated items falling into the experimenter or computer attribution class).
- Confusion of external sources: the number of old items originating from the experimenter or computer, but attributed to the wrong experimenter or computer source (i.e., the number of experimenter and computer generated items falling into the computer and experimenter attribution classes, respectively).

## RESULTS

The covariate-adjusted means (or unadjusted means if the covariate was not significant) are presented in Figure 1 and Figure 2, as a function of group, collapsed over source.

### Item recognition

Comparing patients to healthy controls, a significant group effect was observed, with patients' performance ( $M = 17.23$ ) being inferior to that of controls ( $M = 19.34$ ),  $F(1, 68) = 4.66$ ,  $p < .05$ , and a significant source effect was observed,  $F(2, 136) = 93.20$ ,  $p < .001$ , reflecting superior performance on self-generated items ( $M = 22.34$ ) relative to experimenter- and computer-generated items ( $M = 16.35$ ,  $M = 16.16$ , respectively). Comparing hallucinating to nonhallucinating patients, the group effect was not significant,  $F(1, 48) = 0.06$ ,  $p = .80$ , but a significant source effect was observed,  $F(2, 96) = 63.96$ ,  $p < .001$ , reflecting superior performance on self-generated items ( $M = 21.46$ ) relative to experimenter- and computer-generated items ( $M = 15.44$ ,  $M = 15.41$ , respectively). Comparing delusional to nondelusional patients, the group effect was not significant,  $F(1, 48) = 0.11$ ,  $p = .74$ , but a significant source effect was observed,  $F(2, 96) = 70.37$ ,  $p < .001$ , reflecting superior performance on self-generated items ( $M = 21.54$ ) relative to experimenter- and computer-generated items ( $M = 15.25$ ,  $M = 15.18$ , respectively).

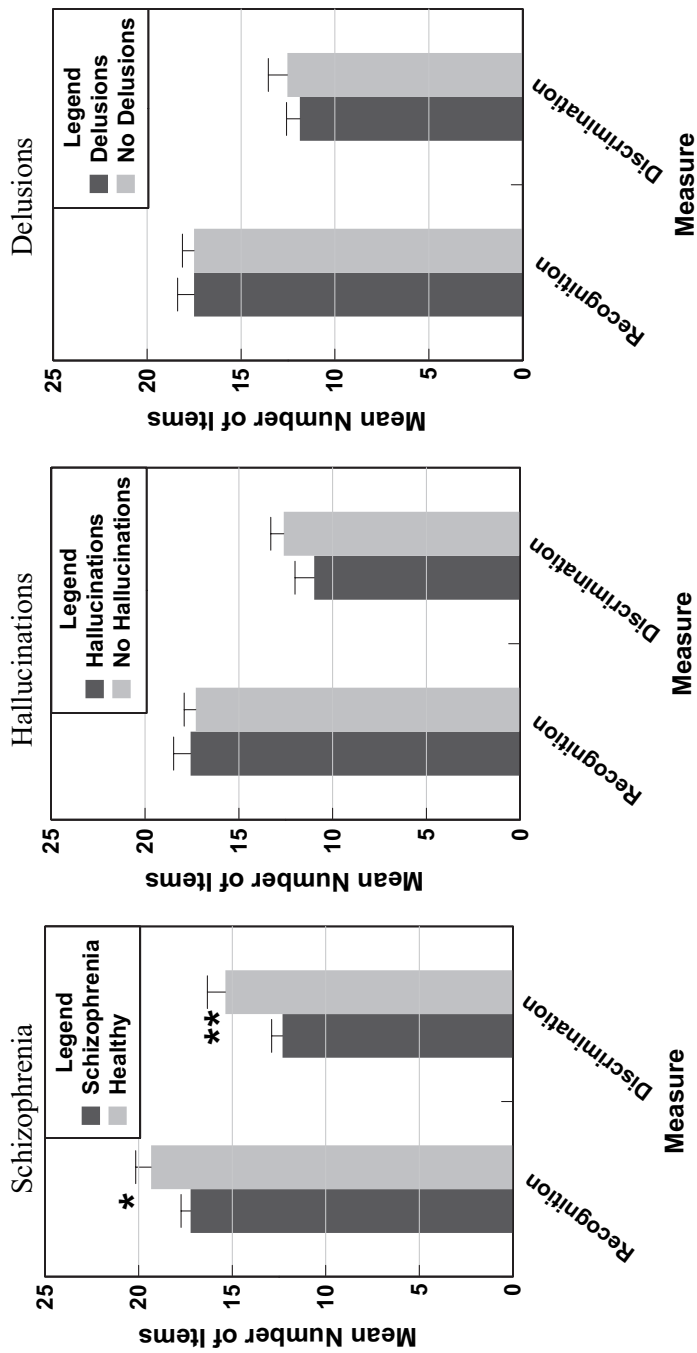
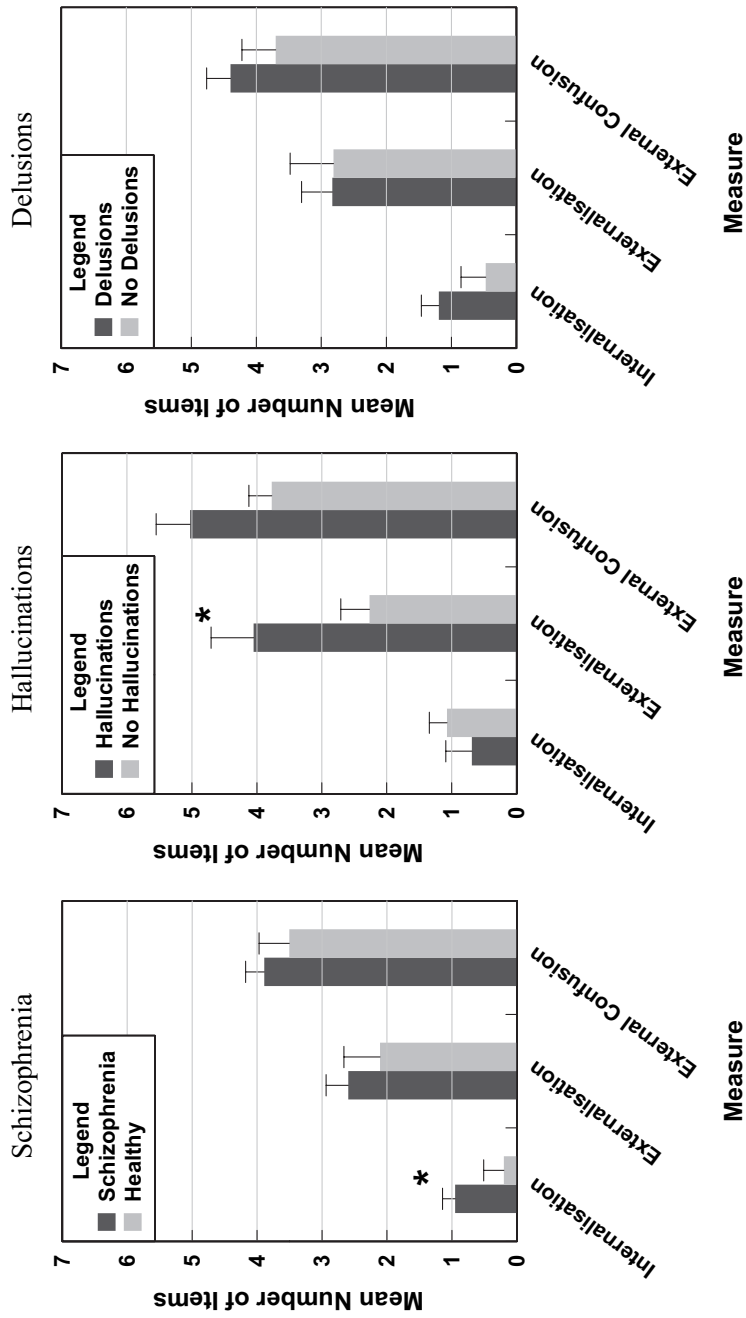


Figure 1. Covariate-adjusted (adjusted only when appropriate, see Results section) mean number of items recognised and discriminated, plotted as a function of group, collapsed over source. Error bars are standard errors. \*\* $p < .01$ , \* $p < .05$ .



**Figure 2.** Covariate-adjusted (adjusted only when appropriate, see Results section) mean number of items resulting from biases, plotted as a function of group, collapsed over source. Error bars are standard errors. \* $p < .05$ .

### Source discrimination

Comparing patients to healthy controls, a significant group effect was observed, with patient performance ( $M = 12.09$ ) being inferior to that of controls ( $M = 15.92$ ),  $F(1, 69) = 11.02$ ,  $p < .01$ , and a significant source effect was observed,  $F(2, 138) = 33.86$ ,  $p < .001$ , reflecting superior performance on self-generated items ( $M = 17.97$ ) relative to experimenter- and computer-generated items ( $M = 12.45$ ,  $M = 11.59$ , respectively). Comparing hallucinating to nonhallucinating patients, the group effect was not significant,  $F(1, 49) = 1.34$ ,  $p = .25$ , but a significant source effect was observed,  $F(2, 98) = 18.73$ ,  $p < .001$ . For this analysis, significant differences were observed between all sources, with highest performance on self-generated items (15.17), lowest performance on computer-generated items (9.00), with performance on experimenter-generated items falling between the two (11.27). Comparing delusional to nondelusional patients, the group effect was not significant,  $F(1, 49) = 0.28$ ,  $p = .60$ , but a significant source effect was observed,  $F(2, 98) = 21.07$ ,  $p < .001$ , with significant differences observed between all sources, with highest performance on self-generated items (15.93), lowest performance on computer-generated items (9.63), with performance on experimenter-generated items falling between the two (11.06).

### Internalisation bias

Comparing patients to healthy controls, the group effect was significant; that is to say, this bias was more pronounced in patients ( $M = 0.95$ ) than controls ( $M = 0.20$ ),  $F(1, 69) = 4.23$ ,  $p < .05$ , and the source effect was not significant,  $F(1, 69) = 1.55$ ,  $p = .22$ . Comparing hallucinating to nonhallucinating patients, the group and source effects were not significant,  $F(1, 49) = 0.63$ ,  $p = .43$ ,  $F(1, 49) = 1.35$ ,  $p = .25$ , respectively. Comparing delusional to nondelusional patients, the group and source effects were not significant,  $F(1, 49) = 2.37$ ,  $p = .13$ ,  $F(1, 49) = 1.73$ ,  $p = .20$ , respectively. The covariate was not significant for any internalisation bias analyses.

### Externalisation bias

Comparing patients to healthy controls, the group and source effects were not significant,  $F(1, 68) = 0.55$ ,  $p = .46$ ,  $F(1, 68) = 3.23$ ,  $p = .07$ , respectively. Comparing hallucinating to nonhallucinating patients, the group effect was significant,  $F(1, 48) = 5.09$ ,  $p < .05$ , reflecting an increased externalisation bias for hallucinating patients ( $M = 4.05$ ) compared to nonhallucinating patients ( $M = 2.26$ ), and the source effect was not significant,  $F(1, 48) = 2.53$ ,

$p = .12$ . Comparing delusional to nondelusional patients, the group and source effects were not significant,  $F(1, 48) = 0.00$ ,  $p = .98$ ,  $F(1, 48) = 3.27$ ,  $p = .08$ , respectively.

### Confusion of external sources

Comparing patients to healthy controls, the group and source effects were not significant,  $F(1, 69) = 0.48$ ,  $p = .49$ ,  $F(1, 68) = 0.47$ ,  $p = .50$ , respectively. Comparing hallucinating to nonhallucinating patients, although they bordered on significance, the group and source effects did not reach significance,  $F(1, 48) = 4.00$ ,  $p = .051$ ,  $F(1, 48) = 4.00$ ,  $p = .051$ . Comparing delusional to nondelusional patients, the group and source effects were not significant,  $F(1, 48) = 1.19$ ,  $p = .28$ ,  $F(1, 48) = 1.08$ ,  $p = .30$ , respectively.

## DISCUSSION

Fundamental to an understanding of hallucinations in schizophrenia is the study of inner/outer confusions in cognition. In the present source monitoring study, subjects were required to distinguish, in memory, between items originating from one of two external sources (computer sound file and experimenter) or an internal source (the subject). When comparing patients to controls, impairments on item recognition and source discrimination were observed. When comparing patient groups split on hallucinations, impairments on recognition and source discrimination were absent, but a bias towards attributing self-generated items to an external source (i.e., an externalisation bias) was observed. In contrast, when comparing patient groups split on delusions, no externalisation bias was observed. This set of results supports the contention that the cognitive systems involved in source monitoring are important for understanding hallucinations in schizophrenia.

The ANCOVA method of accounting for guessing strategies played an important role in interpretation of these results. For example, the group difference in recognition was not significant unless guessing rate was accounted for using ANCOVA. This is because patients commit more false positives than controls ( $M = 4.41$  vs.  $M = 1.20$ , respectively), thereby masking their recognition impairment. Similarly, the externalisation bias observed for hallucinating patients was not significant until the "external" guessing rate was accounted for. However, in contrast to the recognition results, this effect was due to a reduction in the standard error of the group means caused by accounting for variance in the dependent variables with the covariate. In fact, the "external" guessing rate did not differ between hallucinators ( $M = 4.1$ ) and nonhallucinators ( $M = 3.8$ ),  $t(49) = 0.15$ ,

$p = .82$ . This suggests that correcting for guessing using the ANCOVA technique introduced here has the potential to increase sensitivity to group differences by way of two mechanisms.

Despite the apparent subtlety of the effect, the externalisation bias has been confirmed in two reviews of the literature (Ditman & Kuperberg, 2005; Seal, Aleman, & McGuire, 2004). One cognitive explanation of this finding is a degraded “generating-thoughts” signal (Frith, Rees, & Friston, 1998; Frith, 1996; Keefe et al., 2002; Keefe, Arnold, Bayen, & Harvey, 1999). A different possible cause is the addition of vivid auditory sensations to internal events, an interpretation that is congruent with the findings of a number of different approaches to studying hallucinations (Aleman, Bocker, Hijman, de Haan, & Kahn, 2003; Bentall et al., 1991; Bentall & Slade, 1985; Bocker, Hijman, Kahn, & de Haan, 2000; Brébion et al., 2000; Ensum & Morrison, 2003; Franck et al., 2000; Johns et al., 2006; Mintz & Alpert, 1972). These interpretations of the association between hallucinations and externalisations would suggest decreased source discrimination for hallucinations on self items as well as the observed externalisation bias. However, this finding did not emerge in the current set of results, due to the nonsignificant group  $\times$  source interaction for the hallucinations/source discrimination analysis. However, direct inspection of this effect suggests that impaired source discrimination for self-generated items for hallucinating patients ( $M = 13.38$ ) compared to nonhallucinating patients ( $M = 16.97$ ) may emerge with increased power (in current data,  $p = .09$  for this means comparison). Such a “two-hit” model (i.e., an externalisation bias and a deficit in source discrimination for self-generated items) for the cognitive underpinnings of hallucinations has been proposed by others (Seal et al., 2004).

Confusion of the two external sources served as a control condition, to ensure that inner/outer confusion was distinct from general source confusion, as indexed by computer/experimenter source confusion. Although this control measure did not differ significantly between groups for any analysis, a trend towards significance was present when patients were split on hallucinations. Concern that general confusion may underlie the externalisation/hallucination association is mitigated by repeating the analysis when adding external confusion as a covariate: the covariate was not significant ( $p = .60$ ), so did not change the significance of the group difference. However, future source monitoring studies should include two external sources to assess the reliability of this trend (for expansion on this point and other recommendations for future source monitoring studies, see Larøi & Woodward, 2006). If this is a reliable trend, this would suggest that source confusion for hallucinations is not limited to externalisations; however, if reliable this generalised source confusion may extend to delusions as well as hallucinations (see Figure 2).

An unexpected result was that an internalisation bias was increased in all patients compared to controls. However, it may be important to note that inspection of Figure 2 suggests that hallucinating patients' scores *decreased* the internalisation bias patient group mean, whereas delusional patients' scores *increased* it. In fact, in an exploratory analysis of the internalisation bias involving splitting the delusional sample into hallucinators and nonhallucinators, the nonhallucinating delusional patients ( $M = 1.58$ ) differed significantly from the nondelusional patients ( $M = 0.47$ ,  $p < .05$ ), whereas the hallucinating delusional patients ( $M = 0.70$ ) did not ( $p = .68$ ). One interpretation of the results is that hallucinations may suppress a delusion-associated internalisation bias. The association between internalisations and delusions is conceptually supported in the literature, as delusions are likely to be enhanced by a self-focused cognitive style (Freeman, Garety, & Phillips, 2000), and it has been acknowledged that the "content of delusional thinking is often self-referential" (Birchwood, 1999, p. 316). Empirical support has also been reported in a reanalysis of other published data (Woodward, Menon, et al., 2006).

This study was subject to various limitations. First, the patient-healthy control differences must be interpreted with caution, because these groups differed with respect to their general cognitive skills, and also possibly with respect to the cognitive effort exerted when solving puzzles. Although impaired cognition and reduced attentional resources are acknowledged to be an important aspect of schizophrenia, it remains unclear from these results how much this impacted the recognition deficits observed in the schizophrenia group. However, these concerns may be relevant only to differences between *the entire patient sample* and controls. The symptom-split patient groups did not differ on measures of intelligence (see Table 1) or memory (see Figure 1), so this potential limitation may not apply to these comparisons, unless the groups differ on cognitive domains not captured by the IQ estimate used here. A second limitation is that the patient groups differed on symptomatology other than the target symptoms (see Table 1), raising the possibility that symptoms other than hallucinations or delusions may have contributed to symptom group differences. Third, although presentation of the clue and answer were simultaneous, the nonself conditions (experimenter generated and computer generated) may have involved self-generation if the participants generated the associated word themselves as the answer was being provided, potentially reducing the internal/external manipulation strength. Finally, assignment of items to conditions was not counterbalanced, and this could potentially distort results if one item group is more memorable than another, particularly if the effect interacts with participant group.

An interpretational concern with these results is that the causal relationship between cognition and symptomatology may be the reverse of what we have

assumed in this study. Specifically, it is possible that hallucinating patients may become conditioned to believe that internally generated cognitive events are not self generated. If this were the case, the current set of results would also support this interpretation, such that externalisations may be a conditioned response to having hallucinations rather than their cause.

This study supports the contention that the cognitive systems involved in source monitoring are important for understanding hallucinations in schizophrenia. Future work may focus on longitudinal studies to assess whether changes in these biases are associated with, or even precede, changes in symptoms. If they do, it has been suggested that objective measures of psychosis, such as source monitoring biases combined with other cognitive bias measures (Frith, 1994; Garety, Hemsley, & Wessely, 1991; Woodward, Moritz, Cuttler, & Whitman, 2006), could eventually (once refined and combined) prove more sensitive than the self-report measures of psychosis typically used to indicate treatment responsiveness in schizophrenia patients (Keefe et al., 2003, p. 385). Thus, cognitive measures that underlie the symptoms of psychosis may play an important role in optimising psychosocial and pharmacological treatment of schizophrenia spectrum disorders, particularly with respect to early intervention.

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