

Are Anomalous Perceptual Experiences Necessary For Delusions?

Reference as:

Bell, V., Halligan, P.W. & Ellis, H.D. (2008) Are anomalous perceptual experiences necessary for delusions? *Journal of Nervous and Mental Disease*, 196 (1), 3-8.

Authors and affiliations:

Vaughan Bell¹

Peter W. Halligan¹

Hadyn D. Ellis¹

¹ School of Psychology, Cardiff University, UK

Grant support:

None. This research was funded by Cardiff University.

Acknowledgements:

We would like to thank Emmanuelle Peters and Nicola Smedley for collecting the data on the hallucinating and non-hallucinating delusional patients, and for their helpful comments during the study. We would also like to thank the participants and patients who kindly volunteered to take part in the study.

Please Note:

The original version of this article contains an error in the first paragraph of the 'Discussion' which was submitted as a proof correction but was inexplicably not included by the publishers. This manuscript contains the correct version.

Abstract

Several theories of delusions suggest that anomalous perceptual experience is necessary for delusion formation. This study evaluated the levels of anomalous perceptual experience in a large group of non-clinical participants from the general population (N = 337), a group of psychotic inpatients (N = 20), and two groups of hallucinating (N = 24) and non-hallucinating (N = 24) deluded patients. The aims of the study were to evaluate the hypothesis that pathological levels of anomalous perceptual experience were necessarily associated with delusions. Using the Cardiff Anomalous Perceptions Scale (CAPS) the main finding was that as a group, non-hallucinating deluded patients were not significantly different from non-clinical participants on any of the anomalous perceptual experience indices. We conclude that anomalous perceptual experience, as measured by the CAPS, is not necessary for the presence of delusions.

Keywords: hallucination, delusion, anomalous, perception, schizophrenia, psychosis

Introduction

The idea that delusions arise from a primary disturbance of perception was originally put forward by philosopher John Locke (Locke, 1689 / 2004; Porter, 1987). This ‘empiricist’ approach to delusions (Campbell, 2001) proposes that delusions are the result of anomalous experience. In keeping with this approach, Maher (1974; 1988; 1999) consistently argued for an account of delusion formation, where delusions were the product of otherwise normal reasoning processes applied to anomalous perceptual experience. According to Maher’s influential account, anomalous perceptual experience was both necessary *and* sufficient to account for the formation of delusions. This account has subsequently been challenged (Bell et al., 2006a). Indeed, Maher’s assertion that anomalous perceptual experience is necessary for delusion formation has formed an underlying premise for a number of major neuropsychological theories of delusion formation (Davies et al., 2001; Ellis et al., 1997; Langdon and Coltheart, 2000). Moreover, while other existing cognitive theories of delusion formation do not explicitly suggest that anomalous perceptual experience is necessary, they often draw inspiration from Maher’s model in stressing its importance (Bentall et al., 2001; Freeman and Garety, 2004; Garety and Hemsley, 1994).

A point often overlooked in the research literature is that Maher’s definition of an ‘anomalous perceptual experience’ and how it relates to delusions has changed over time, as he increasingly included what seem to be post-perceptual processes into his explanation of ‘perceptual disturbance’. In Maher’s early work (e.g. Maher, 1974) the explanation for delusion formation is relatively clear-cut and distinctly focused on disturbed perception: “a delusion is a hypothesis designed to explain unusual perceptual phenomena and developed through the operation of normal cognitive processes”, which he claims are “indistinguishable from [those] employed by non-patients, by scientists, and by people generally”.

In his later work, however, Maher (1988) introduces mechanisms to account for why a person might need to seek an explanation for an anomalous experience, and to explain how perceptions are experienced as anomalous at all, both of which, despite his assertions, encroach on areas traditionally thought to be outside the first ‘perceptual’ stage of delusion formation.

In later work still, Maher (1999) further explains the origins of anomalous experience as including:

... a broad range of neuropsychological anomalies. These include, but are not confined to, (a) endogenous neural activation of the feeling of significance normally triggered by pre-conscious recognition of changes in a familiar environment; (b) unrecognized defects in the sensory system, such as undiagnosed hearing loss, or the endogenous activation or inhibition of the central neural representations of sensory input; (c) temporary alterations in the intensity and vividness of sensory input, as in some forms of drug intoxication; (d) neurologically based difficulties in the focusing of attention with consequent difficulty in discriminating between situationally relevant and irrelevant elements of the environment; (e) experienced discrepancies between the willed intent of a response and the actual form of a response; (f) impairment in the monitoring and calculation of recurring sequential probabilities in environmental events

Most notably, (f) seems unequivocally to describe inferential reasoning, whereas (a) and (e) suggest a post-perceptual breakdown in metacognition: (a) has been proposed as a metacognitive factor in dual-process models of memory involved in the control of memory retrieval (Koriat, 2000); and (e) in terms of the intention-monitoring system proposed by Blakemore et al. (2002). Similarly, (d) could equally describe any number of disorders to the high-level contention scheduling / supervisory attention model (Shallice and Burgess, 1998), which is not thought to have a direct role in perception. Although anomalous experience is not consistently defined in the literature by Maher

or others, we define it as an internally generated perception or experience that is rare or statistically aberrant in the population and that may or may not involve a modality specific hallucination.

Anomalous perceptual experiences have been linked to delusions in a number of correlational studies of psychotic patients (Bilder et al., 1985; Peralta and Cuesta, 1999; Peralta et al., 1992) and have also been shown to be associated both with paranormal beliefs (Thalbourne, 1994) and delusional ideation in non-clinical populations (Bell et al., 2006b; Verdoux et al., 1998). Few studies have formally empirically evaluated Maher's claim that anomalous experiences are necessary for delusions. Chapman and Chapman (1988) conducted an (admittedly poorly-controlled) study and interviewed a number of students with high levels of schizotypy about their perceptual experiences and subsequently argued that bizarre beliefs could arise without the necessity of anomalous perceptual experience. The study findings were preliminary, however, as the study relied on interview and did not use standardised measures. On a related note, Lawrence and Peters (2004) tested believers in the paranormal and reported that reasoning biases were related to paranormal beliefs, but not paranormal experiences.

To the best of our knowledge, no one has directly tested Maher's theory in delusional patients. One difficulty in testing such an hypothesis, is that most if not all existing psychometric measures of perceptual distortion derive their content and language from mainstream clinical psychiatry and tend to focus on a restricted set of anomalous experiences, usually of a hallucinatory nature. This potentially confounds any inferences about whether delusions are linked to anomalous experience, owing to the narrow definition of anomalous experience, typically drawn from the clinical presentation of psychosis. The Cardiff Anomalous Perceptions Scale (CAPS; Bell et al., 2006b) was devised to address these issues and has proved to be a valid and reliable measure for a wide range of anomalous experiences drawn from a principled approach to understanding the phenomena.

The CAPS examines the presence of frank hallucinations and illusions in a number of modalities (including auditory verbal hallucinations), perceptual flooding and confusion, changes in sensory intensity, distortions of body image and proprioception, feelings of being uplifted, changes in time perception, thought broadcast and echo, and the experience of a sensed presence (Bell et al., 2006b) while specifically excluding experiences that may have occurred under the influence of drugs. Notably, the CAPS does not enquire about thought interference (blocking, insertion, withdrawal) and dissociative experiences

Therefore, the aim of the current study was to test the critical hypothesis that patients with delusions show pathological levels of anomalous perceptual experience. Three groups of psychotic patients were included, one group with, and one group without concurrent hallucinations, and a group of patients with current psychosis not distinguished by their hallucination status. While we predicted that hallucinating delusional patients and the 'unselected' psychotic patients would score highly on a measure of anomalous perceptual experience over a group of patients without hallucinations, a Maherian account of delusions would predict that non-hallucinating delusional patients should also score higher than a non-clinical sample.

Method

Participants and Procedure

Four groups of participants were involved in this study. Two have been previously reported by Bell et al. (2006b), the non-clinical and ‘unselected psychosis’ group, and two groups have not been reported before: the ‘deluded inpatients with hallucinations’ (D+H) and the ‘deluded patients without hallucinations’ (D-H). All groups are used in a comparison of levels of anomalous perceptual experience, and the non-clinical group and the ‘unselected psychosis’ group are also used for new analyses which have not been previously reported.

Non-clinical

The non-clinical sample has been fully described in Bell et al. (2006b) and is briefly described below. The non-clinical group consisted of 337 participants (mean age = 21.6, $SD = 5.4$, range 18 – 54). Participants were largely drawn from undergraduate students ($N = 305$) including 111 males and 176 females (not disclosed = 18) with a mean age 19.9 ($SD = 2.6$; range 18 – 44; not disclosed = 13), who took part as part of their induction programme (the academic introduction during the first week of university) or who responded to requests for participants. The remaining 32 participants were drawn from an anonymous postal survey, having responded to advertisements posted on general-purpose internet discussion groups. This sample consisted of 17 females and 14 males (not disclosed = 1) with a mean age of 32.4 ($SD = 10.2$; range 18 – 54; not disclosed = 1). All participants completed the CAPS (Bell et al., 2006b) and the 21-item Peters et al. Delusions Inventory (PDI; Peters et al., 2004).

Clinical

In addition to the non-clinical sample, three clinical samples completed the CAPS, an ‘unselected psychosis’ sample, previously reported in Bell et al. (2006b), and a new sample of 48 psychotic inpatients, categorised into hallucinating and non-hallucinating patients:

- i) unselected psychosis (UP), $N = 20$
diagnoses of: schizophrenia = 9, bipolar disorder = 6, psychotic depression = 2, delusional disorder = 1, schizoaffective disorder = 1, and unspecified psychosis = 1.
- ii) deluded inpatients *with* hallucinations (D+H), $N = 24$
diagnoses of: schizophrenia = 19; schizoaffective disorder = 2; bipolar disorder = 2; unspecified psychosis = 1.
- iii) deluded patients *without* hallucinations (D-H), $N = 24$
diagnoses of: schizophrenia = 14; schizoaffective disorder = 5; bipolar disorder = 2; psychotic depression = 2; unspecified psychosis = 1.

Data from the ‘unselected psychosis’ group were recruited from 4 acute psychiatric admission wards in the Cardiff area. The patients were selected on the basis of having been diagnosed with a current psychotic episode by the responsible clinician. Patients in this sample were screened with the Psychosis Screening Questionnaire (PSQ; Bebbington and Nayani, 1995) to confirm the clinical classification. The group consisted of 13 females and 7 males and had mean age 40.68 ($SD = 10.6$, range 25 – 64, not disclosed = 1).

CAPS data from the deluded psychiatric inpatients were collected by Nicola Smedley and Emmanuelle Peters (Department of Psychology, Institute of Psychiatry, King’s College London) from inpatients in acute wards ($N = 19$), inpatients in rehabilitation wards ($N = 20$) and service users in residential rehabilitation housing in the South London and Maudsley NHS Trust area ($N = 9$). Of these 48 inpatients, 24 were diagnosed as being currently deluded with hallucinations (D+H) and 24 were diagnosed as being currently deluded without hallucinations (D-H). Diagnoses were originally made by the responsible clinician, and confirmed by administration of the Scale for the Assessment

of Positive Symptoms (SAPS; Andreasen, 1984). Participants in the group of deluded patients with hallucinations consisted of 13 males and 11 females, had mean age of 38.13 ($SD = 9.78$, range 18 – 65, not disclosed = 2), a mean hallucinations subscale score of 1.98 ($SD = .88$, range 2 – 5), and a mean delusions subscale score of 1.82 ($SD = .71$, range 2 – 5). Participants in the group of deluded patients without hallucinations consisted of 15 males and 9 females, had a mean age of 40.64 ($SD = 11.24$, range 21 – 63), a hallucinations subscale score of .1 ($SD = .19$, range 0 – 1) and a mean delusions subscale score of 1.22 ($SD = .66$, range 2 – 5).

When tested with a chi-square test, there was no significant difference in the distribution of gender between the clinical groups ($\chi^2 = 3.411$; Fisher's exact $p = .216$). When the clinical groups were compared with a one-way between subjects ANOVA there was no effect of group on age ($F_{(2,62)} = .440$, $p = .646$) and no significant differences were found when groups were compared individually using a Tukey-HSD post-hoc comparison. As expected, the deluded group with hallucinations were rated as having significantly greater levels of hallucinations than the deluded group without hallucinations when SAPS delusions score was compared using an independent samples t-test ($t_{(46)} = -10.183$, $p < .0001$) with the deluded group without hallucinations scoring close to zero (.1). Although both deluded groups were rated as being clinically deluded on the SAPS, it is notable that when compared using an independent samples t-test, the difference between the deluded group with hallucinations (D+H) and the deluded group without hallucinations (D-H) was significant ($t_{(46)} = -3.023$, $p < .005$), although with only a .6 difference in mean rating between groups.

None of the participants in the clinical samples had a history of either brain injury, or substance or alcohol abuse, and, except for one patient, were all on a medication regime at the time of testing. This was confirmed by review of the clinical notes. The CAPS was completed with the experimenter present to assist with any difficulties in reading or comprehension.

The study was ethically reviewed and approved by all the participating institutions and informed consent was obtained from participants (both clinical and non-clinical) before participation.

Results

Comparison of levels of anomalous perceptual experience

The means for the CAPS total score and subscale scores are illustrated in Figure 1. The non-clinical controls, and the hallucinating (D+H) and non-hallucinating deluded (D-H) patients were compared on all subscales of the CAPS using a one-way independent samples ANOVA. There was a significant effect of group on all CAPS subscales: CAPS total score (non-clinical mean = 7.33, $SD = 5.8$; UP mean = 15.85, $SD = 8.54$; D-H mean = 6.0, $SD = 5.24$; D+H mean = 11.42, $SD = 7.25$; ANOVA $F_{(2,382)} = 6.251, p < .005$), CAPS distress subscale (non-clinical mean = 15.47, $SD = 14.47$; UP mean = 46.95, $SD = 36.77$; D-H mean = 19.08, $SD = 18.49$; D+H mean = 32.08, $SD = 27.54$; ANOVA $F_{(2,382)} = 12.626, p < .0001$), CAPS intrusiveness subscale (non-clinical mean = 17.99, $SD = 16.95$; UP mean = 50.2, $SD = 35.82$; D-H mean = 17.17, $SD = 17.0$; D+H mean = 32.08, $SD = 27.53$; ANOVA $F_{(2,382)} = 7.156, p < .005$) and CAPS frequency subscale (non-clinical mean = 14.6, $SD = 14.2$; UP mean = 50.2, $SD = 40.08$; D-H mean = 17.42, $SD = 14.98$; D+H mean = 40.17, $SD = 31.87$; ANOVA $F_{(2,382)} = 29.082, p < .0001$).

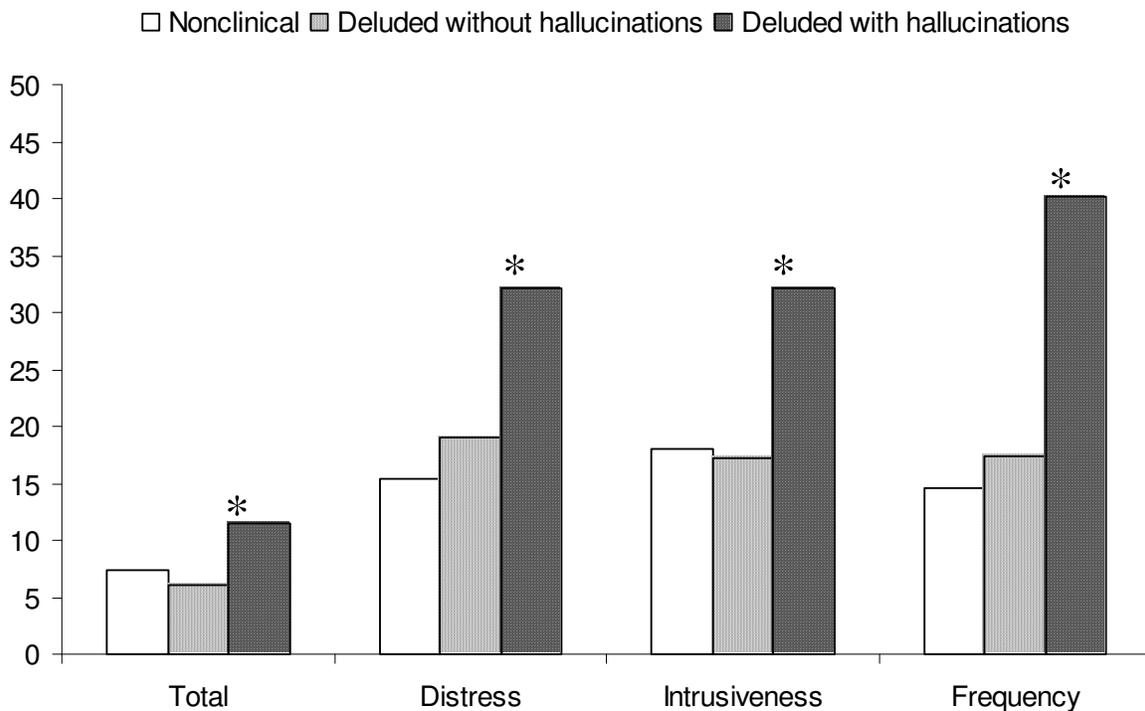


Figure 1. CAPS scores for non-clinical, hallucinating and non-hallucinating groups.

The asterisk denotes scores significantly different from non-clinical group at least $p < 0.005$

Tukey-HSD post-hoc tests were performed to compare the differences between individual groups on individual subscales, the results of which are displayed in Table 1. As can be seen, the hallucinating group differed from the non-clinical group on all CAPS scores by at least $p < .005$, and from the non-hallucinating group on all CAPS scores by at least $p < .05$. Notably, no significant differences were observed between the non-clinical and non-hallucinating groups on any of the CAPS scales.

CAPS subscale comparison	<i>p</i> value	95% CIs
<i>CAPS Total</i>		
Nonclinical vs D-H	.535	-1.60 to 4.25
Nonclinical vs D+H	.003	-7.01 to 1.17
D+H vs D-H	.004	-9.41 to -1.42
<i>CAPS Distress</i>		
Nonclinical vs D-H	.527	-11.47 to 4.25
Nonclinical vs D+H	<.0001	-24.48 to -8.75
D+H vs D-H	0.013	2.25 to 23.75
<i>CAPS Intrusiveness</i>		
Nonclinical vs D-H	.974	-8.00 to 9.66
Nonclinical vs D+H	.001	-22.92 to -5.26
D+H vs D-H	0.011	2.85 to 26.98
<i>CAPS Frequency</i>		
Nonclinical vs D-H	.679	-10.7 to 5.08
Nonclinical vs D+H	<.0001	-33.46 to -17.67
D+H vs D-H	<.0001	11.96 to 33.54

Table 1. Post-hoc comparison between clinical groups on CAPS subscales.
 CIs = confidence intervals for mean difference; D-H = deluded group without hallucinations;
 D+H = deluded group with hallucinations.

Howell (2005) and Honeig and Heisey (2001) note that the use of retrospective power calculations to aid the interpretation of null results is inappropriate, and, instead, confidence intervals should be used to examine the likely extent of any difference between means. For the comparisons in our study, the confidence intervals for all the differences between the non-clinical and non-hallucinating groups span zero, suggesting the possibility of no true difference between the groups. The question of how confidently we can draw these conclusions remains, however. To quote Honeig and Heisey (2001), “if the nonrefuted states are clustered tightly about a specific null value, one has confidence that nature is near the null value”. For our critical comparison, the confidence intervals for the differences on CAPS total score between the non-clinical and non-hallucinating group range between -1.60 to 4.25, suggesting a high degree of confidence that the difference between the group means is genuinely zero.

These differences suggest that the non-hallucinating deluded group (D-H) did *not* show pathological levels of anomalous perceptual experience despite reporting currently active delusions and allows us to conclude that pathological levels of anomalous perceptual experience are not necessary to account for the presence of all delusions.

Comparison of age and sex in clinical and non-clinical groups

As can be seen from the descriptions of the participant samples, there was a small imbalance in the numbers of male and female participants between the clinical and non-clinical samples, although the distribution was not significantly different from chance when tested with a chi-square test (clinical [male = 35, female = 33], non-clinical [male = 125, female = 193]; $\chi^2 = 3.415, p = .078$). There was, however, a significant difference in age between the samples when tested with an independent samples t-test (non-clinical mean = 21.13; *SD* = 5.45; clinical mean = 39.71; *SD* =

10.42; $t = -20.89$, $p < .0005$) with the non-clinical sample comprising significantly younger participants than the clinical sample. Although the age differences between the samples are likely to have affected the results, they do not significantly affect the main conclusions drawn from this study, given that age is inversely associated with psychotic symptoms in adults, in both clinical and non-clinical populations (Gonzalez-Pinto et al., 2004; van Os et al., 2000). This suggests that a comparison between age-matched groups is likely to show a greater effect in agreement with the study's main hypothesis than the potentially age-attenuated results reported here.

Discussion

Previous studies have shown that hallucinations and delusions commonly co-occur in clinical samples (Bilder et al., 1985; Peralta and Cuesta, 1999; Peralta et al., 1992) and this association has, in turn, been used to support theoretical models of delusions that suggest that anomalous perceptual experience is necessary for delusion formation. However, previous studies have not differentiated delusional patients into hallucinating and non-hallucinating groups, and, hence, the original assumption regarding the co-presence (and assumed necessity) may reflect a population average rather than a causal prerequisite. This study evaluated the levels of anomalous perceptual experience in a group of non-clinical participants from the general population and groups of hallucinating (D+H) and non-hallucinating (D-H) deluded patients. The main finding was that non-hallucinating deluded patients (D-H) were not significantly different from non-clinical participants on any of the anomalous perceptual experience indices. This provides evidence against current theories that emphasise that pathological levels of anomalous perceptual experience are necessary for the presence of delusions.

Several researchers have previously argued that anomalous experiences are either necessary, or necessary and sufficient, for delusions (Ellis, 1998; Ellis and Young, 1990; Ellis et al., 1997; Langdon and Coltheart, 2000; Maher, 1974, 1988, 1999). Other theorists, such as Bentall et al. (2001) and Freeman and Garety (2004), are non-committal as to whether such experiences are necessary for delusions, although both accounts cite anomalous experience as a key part of the model. If it is indeed the case that there are subsets of delusional patients that do not show significantly elevated levels of anomalous perceptual experience, as our results suggest, then the possibility arises that processes involved in perception and reasoning could be differentially affected, and that delusions could arise from pathologies to both or either. Indeed, this is consistent with speculations in recent models of persecutory delusions (Bentall et al., 2001; Freeman and Garety, 2004) and more general models by McKay et al. (2005) and Davies et al. (2001).

It is important to note that this study focused on anomalous perceptual experiences, and not on other potential sources of experience distortion, such, as anomalous affective experience. The results provide evidence against Maher's influential (1974) theory which clearly stated that "a delusion is a hypothesis designed to explain unusual *perceptual* phenomena and developed through the operation of normal cognitive processes" [our emphasis]. However, in Ellis and Young's model of Capgras delusion (Ellis, 1998; Ellis and Young, 1990; Ellis et al., 1997), it is argued that the anomalous experience is the product of a deficit in the covert emotional response to familiar faces which in turn produces an anomalous recognition experience. Although the perceptual effect of this anomalous experience may be captured by the CAPS, the proposed affective anomaly may not. These sorts of distinctions pose further questions as to whether perceptual or affective distortions can or should be considered discrete given the significant evidence for the reciprocal influence of cognition, affect and perception (Bruce et al., 2003). It is useful to suggest at this stage that there is probably a spectrum of influences on conscious perceptual experience. Consequently, the current study can be seen as providing evidence against the 'strong form' of the anomalous experience hypothesis that emphasises the necessity of perceptual distortions, rather than the necessity of all 'anomalous experiences', although it is perhaps worth sounding a note of caution about any theory that uses such an undefined concept as 'anomalous experience' because it is likely to be too wide to be empirically useful, and operationally unfalsifiable.

As illustrated by the explanation of Capgras delusion, anomalous *affective* experiences may still play a key role in delusion formation. Indeed, affect is an increasingly likely candidate for mediating belief pathology, given its central role in defence and motivational accounts of delusions (e.g. Bentall et al., 2001), and in theories which cite emotion as have a more direct effect on delusion formation (e.g. Freeman and Garety, 2004).

There is also growing evidence that the emotional appraisal of anomalous experiences, whether strictly perceptual or otherwise, might be a key mediator of the distress and disability which is likely to determine whether an unusual belief becomes clinically relevant. Indeed, Maher (1988) proposed that anomalous experiences may cause anxiety, and that delusions result from 'explanation seeking' in an attempt to help resolve this tension (in a mechanism similar to cognitive dissonance). There is now a more recent body of research suggesting that some explanations, i.e. negative appraisals, are linked to increased distress themselves (Freeman et al., 2001; Freeman and Garety, 2003, 2004; Gauntlett-Gilbert and Kuipers, 2005) and experimental evidence has recently been provided by the longitudinal studies of Hanssen et al. (2005) and Krabbendam et al. (2005) who have reported that participants experiencing distress, in combination with an hallucinatory episode at the first assessment, have an increased chance of presenting with a delusion by the second. Further work has suggested that the risk may be even greater for those with abnormally-reactive dopamine systems (Myin-Germeys et al., 2005). This would suggest that the emotional impact of an anomalous perceptual experience may be the critical factor in determining whether, in the long-term, it leads to a delusion.

Limitations of the current study include the fact that, as a cross-sectional study we cannot draw firm conclusions as to the potential temporal or causal role of anomalous perceptual experience. For example, one possible hypothesis is that currently non-hallucinating patients had their delusions 'activated' by anomalous perceptions which long since abated but are nevertheless left with an ongoing delusion. This scenario is not considered by Maher's theory, which argues that delusions arise from the normal interpretation of ongoing anomalous experience. Although rarely tackled directly in theories based primarily on patients with monothematic delusions (Davies et al., 2001; Ellis, 1998; Ellis and Young, 1990; Ellis et al., 1997; Langdon and Coltheart, 2000), the implication is that ongoing anomalous experience is necessary for the maintenance of delusions, particularly in the case of the Capgras delusion, from which many of these theories mostly draw their inferences. Although the largely unexplored phenomenon of delusional memory (David and Howard, 1994; Howard and Burns, 1992) may account for cases where anomalous experience was initially involved in the formation but not maintenance of delusions, this issue remains unresolved. Although studies have looked at the longitudinal influence of hallucination and distress on later delusion formation (Hanssen et al., 2005; Myin-Germneys et al., 2005), the ongoing influence of perceptual distortions has yet to be studied.

Furthermore, it could be argued that only a very circumscribed or specific anomalous experience would be necessary for a delusion to form. As the CAPS score is based upon a summation of experiences, this situation might not be adequately captured by the scale. It is important to note, however, that this hypothesis is ultimately unfalsifiable, as defenders of this position can always argue that the failure to find evidence of anomalous experience does not refute the hypothesis (as there might always be an ever more circumscribed or fleeting anomalous experience that has yet to be uncovered). We admit that this is a possibility, but would argue that as the first direct test of Maher's theory, our findings suggest that there are some delusional patients which do not have the gross perceptual distortions often cited as necessary.

Conclusions

In summary, the present study presents for the first time, evidence that pathological levels of anomalous perceptual experience, as measured by the Cardiff Anomalous Perceptions Scale, are not necessary to account for the presence of all delusions, given that the non-hallucinating group of delusional patients did not score differently from healthy controls. Analysis of the confidence intervals for this difference suggests that the result can be accepted with a high level of confidence.

References

- Andreasen NC (1984) *Scale for the Assessment of Positive Symptoms (SAPS)*. Iowa City: Department of Psychiatry, University of Iowa College of Medicine.
- Bebbington PE, Nayani T (1995) The psychosis screening questionnaire. *International Journal of Methods in Psychiatric Research*, 5: 11-19.
- Bell V, Halligan PW, Ellis HD (2006a) Explaining delusions: a cognitive perspective. *Trends Cogn Sci*, 10: 219-26.
- Bell V, Halligan PW, Ellis HD (2006b) The Cardiff Anomalous Perceptions Scale (CAPS): A new validated measure of anomalous perceptual experience. *Schizophr Bull*, 32: 366-77.
- Bentall RP, Corcoran R, Howard R, Blackwood N, Kinderman P (2001) Persecutory delusions: a review and theoretical integration. *Clin Psychol Rev*, 21: 1143-92.
- Bilder RM, Mukherjee S, Rieder RO, Pandurangi AK (1985) Symptomatic and neuropsychological components of defect states. *Schizophr Bull*, 11: 409-419.
- Blakemore, S. J., Wolpert, D. M., Frith, C. D. (2002) Abnormalities in the awareness of action. *Trends Cogn Sci*, 6: 237-242.
- Bruce V, Green PR, Georgeson MA (2003) *Visual Perception: Physiology, Psychology and Ecology*. Hove: Psychology Press.
- Campbell J (2001) Rationality, meaning, and the analysis of delusion. *Philosophy, Psychiatry, and Psychology*, 8: 89-100.
- Chapman LJ, Chapman JP (1988) The genesis of delusions. In Oltmanns TF, Maher BA (Eds) *Delusional beliefs* (pp 167-183). Chichester: Wiley.
- Davies M, Coltheart M, Langdon R, Breen N. (2001) Monothematic delusions: Towards a two-factor account. *Philosophy, Psychiatry, and Psychology*, 8: 133-158.
- David AS, Howard, R. (1994) An experimental phenomenological approach to delusional memory in schizophrenia and late paraphrenia. *Psychol Med*, 24: 515-24.
- Ellis HD (1998) Cognitive neuropsychiatry and delusional misidentification syndromes: An exemplary vindication of the new discipline. *Cognitive Neuropsychiatry*, 3: 81-90.
- Ellis HD, Young AW (1990) Accounting for delusional misidentifications. *Br J Psychiatry*, 157: 239-48.
- Ellis HD, Young AW, Quayle AH, De Pauw KW (1997) Reduced autonomic responses to faces in Capgras delusion. *Proc R Soc Lond B Biol Sci*, 264: 1085-92.
- Freeman D, Garety PA, Kuipers, E. (2001) Persecutory delusions: developing the understanding of belief maintenance and emotional distress. *Psychol Med*, 31:1293-306.

Freeman D, Garety PA. (2003) Connecting neurosis and psychosis: the direct influence of emotion on delusions and hallucinations. *Behav Res Ther*, 41: 923-47.

Freeman D, Garety P (2004) *Paranoia: The Psychology of Persecutory Delusions*. Hove: Psychology Press.

Garety PA, Hemsley DR (1994) *Delusions: Investigations into the Psychology of Delusional Reasoning*. Hove: Psychology Press.

Gauntlett-Gilbert J, Kuipers E (2005) Visual hallucinations in psychiatric conditions: Appraisals and their relationship to distress. *British Journal of Clinical Psychology*, 44: 77-87.

Gonzalez-Pinto A, van Os J, Peralta V, Perez de Heredia JL, Mosquera F, Aldama A, Gonzalez C, Gutierrez M, Mico JA. (2004) The role of age in the development of Schneiderian symptoms in patients with a first psychotic episode. *Acta Psychiatr Scand*, 109: 264-8.

Hanssen M, Krabbendam L, de Graaf R, Vollebergh W, van Os, J. (2005) Role of distress in delusion formation. *Br J Psychiatry Suppl*, 48: s55-8.

Hoening JM, Hesley DM (2001) The abuse of power: The pervasive fallacy of power calculations for data analysis. *Am Stat*, 55: 1-6.

Howard R, Burns A (1992) Delusional memory in schizophrenia. *Br J Psychiatry*, 160: 129.

Howell DC (2005) Power. In Everitt BS, Howell DC, (Eds) *Encyclopedia of Statistics in Behavioral Science* (pp 1558-1564) London: John Wiley and Sons Ltd.

Koriat A (2000) The feeling of knowing: some metatheoretical implications for consciousness and control. *Conscious Cogn*, 9: 149-71.

Krabbendam L, Myin-Germeys I, Bak M, van Os J (2005) Explaining transitions over the hypothesized psychosis continuum. *Aust N Z J Psychiatry*, 39: 180-6.

Langdon R, Coltheart M (2000) The cognitive neuropsychology of delusions. *Mind and Language*, 15: 183-216.

Lawrence E, Peters E (2004) Reasoning in believers in the paranormal. *J Nerv Ment Dis*, 192: 727-33.

Lock, J. (1689) *An essay concerned human understanding*.

Lock, J. (2004) *An essay concerned human understanding*. London: Penguin Books.

Maher BA. (1974) Delusional thinking and perceptual disorder. *Journal of Individual Psychology*, 30: 98-113.

- Maher BA. (1988) Anomalous experience and delusional thinking: The logic of explanations. In Oltmanns TF, Maher BA (Eds). *Delusional beliefs* (pp 15-33). Chichester: Wiley.
- Maher BA. (1999) Anomalous experience in everyday life: Its significance for psychopathology. *The Monist*, 82: 547-570.
- McKay R, Langdon R, Coltheart, M. (2005) "Sleights of mind": Delusions, defences and self-deception. *Cognitive Neuropsychiatry*, 10: 205-326.
- Myin-Germeys I, Marcelis M, Krabbendam L, Delespaul P, van Os J. (2005) Subtle fluctuations in psychotic phenomena as functional states of abnormal dopamine reactivity in individuals at risk. *Biol Psychiatry*, 58: 105-10.
- Peralta V, de Leon J, Cuesta MJ. (1992) Are there more than two syndromes in schizophrenia? A critique of the positive-negative dichotomy. *Br J Psychiatry*; 161: 335-43.
- Peralta V, Cuesta MJ. (1999) Dimensional structure of psychotic symptoms: an item-level analysis of SAPS and SANS symptoms in psychotic disorders. *Schizophr Res*, 38: 13-26.
- Peters E, Joseph S, Day S, Garety, P. (2004) Measuring delusional ideation: the 21-item Peters et al. Delusions Inventory (PDI). *Schizophr Bull*, 30: 1005-22.
- Porter, R. (1987) *Mind-forg'd manacles: A history of madness in England from restoration to the regency*. London: Penguin Books.
- Shallice T, Burgess PW (1998) The domain of supervisory processes and temporal organization of behaviour. In Roberts AC, Robbins TW, Weiskrantz L. (Eds) *The prefrontal cortex: Executive and cognitive functions*. Oxford: Oxford University Press.
- Thalbourne MA. (1994) Belief in the paranormal and its relationship to schizophrenia-relevant measures: a confirmatory study. *Br J Clin Psychol*, 33: 78-80.
- van Os J, Hanssen M, Bijl RV, Ravelli A. (2000) Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophr Res*, 45: 11-20.
- Verdoux H, van Os J, Maurice-Tison S, Gay B, Salamon R, Bourgeois, M. (1998) Is early adulthood a critical developmental stage for psychosis proneness? A survey of delusional ideation in normal subjects. *Schizophr Res*, 29: 247-54.