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SPECIAL ISSUE

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Psychological pathways to depression in schizophrenia

Studies in acute psychosis, post psychotic depression and auditory hallucinations

Introduction

Emotional dysfunction and schizophrenia have long been uncomfortable bedfellows. It was Bleuler who first argued that problems of affect lie at the heart of schizophrenia and that the symptoms we all focus on, the hallucinations and delusions, are merely 'accessory' and common to many forms of disorder. This view gave way to the now familiar distinction between affective and non-affective psychosis and to Jaspers' hierarchical approach to diagnosis wherein affective symptoms are 'trumped' by the presence of schizophrenia in terms of diagnosis and treatment. Yet emotional dysfunction, depression in particular, is pervasive in non-affective psychosis!

It is now understood that the 'prodromal' and early stages of psychosis 'set the scene' for the development of the long-term illness trajectories and disabilities, embodied in the concept of schizophrenia (Birchwood et al. 1998; Harrison et al. 2001; Jones et al. 1993, Wiersma et al. 1998, 2000); emotional dysfunction and depression in particular, forms a key part of these early symptoms (Häfner et al. 1998 and this volume).

In acute schizophrenia, the second generation of factor analytic studies of schizophrenia symptoms has yielded a further dimension of depressive symptoms (McGorry et al. 1998; van Os et al. 1999) in addition to the familiar dimensions of positive and negative symptoms. When orthogonality is not imposed on these factors, the dimensions co-vary in-patient (Peralta and Cuesta 1999) and community samples (Stefanis et al. 2002); indeed, studies of depression in acute schizophrenia suggest that up to 50 % experience depressive symptoms (Birchwood et al. 2000).

Generally, the prevalence of depression in schizophrenia ('post psychotic depression' -PPD) ranges from 22 % to 75 % depending upon the criteria used and the chronicity of the sample. This diversity is most commonly attributed to the numerous diagnostic criteria available to assess symptoms, the frequency of follow-up and the variations in patient environment. Furthermore, and contrary to both DSM-IV (APA 1994) and ICD-10 (WHO 1993) diagnostic criteria, Bressan Chaves, Pilowsky et al. (2002) have suggested that the requirement of a temporal link between postpsychotic depression and the immediately preceding psychotic episode is not born out by the evidence.

Individual psychotic symptoms have also been linked with a raised risk of depression. Studies of the phenomenology of auditory hallucinations have revealed that between two-thirds and three-quarters of voice hearers will be at least moderately depressed and distressed by the experience (Chadwick and Birchwood 1994; Birchwood and Chadwick 1997; Trower et al. 2004).

It is tempting to characterize depression as integral to psychosis and part and parcel of the accompanying loss of functioning, and it is perhaps for this reason that the pathogenesis of these emotional co-morbidities is not understood and few effective treatments are available. In order to improve our understanding of emotional dysfunction and to develop new treatments, we have argued (Birchwood 2003) that we need to make a clear distinction between three core, but not mutually exclusive pathways: depression that is intrinsic to the psychosis diathesis, a psychological reaction to it, and the product of disturbed developmental pathways resulting from developmental trauma and the childhood antecedents of psychosis.

In this paper we will focus on the second pathway and present data from three studies which examine this in relation to depression in: PPD, auditory hallucinations and in acute, first episode schizophrenia. Here our em-

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phasis is on psychosis the life event rather than on psychosis the illness. In the following studies we draw strength from the cognitive school of psychiatry (Clark and Fairburn 1998) with its emphasis on beliefs and attributions people make about their circumstances; and also the literature linking life events and unipolar depression (Paykel 2003) which has shown that it is the personal and negative appraisal patients make about these events rather than the events themselves, which trigger depression.

Post psychotic depression

The application of "ranking theory" to the role of life-events in triggering unipolar depression (Brown, Harris and Hepworth 1995; Farmer and McGuffin 2003) provides a framework to help understand the link between a putative life-event and depression, where the appraisal of the life-event is of primary importance. Recent ideas based on social ranking theory and power from evolutionary psychology (Gilbert 1992), argue that certain life situations are likely to be depressogenic, particularly if they are appraised as leading to loss, humiliation and entrapment (or defeat). We have argued that placing the individual at the centre of our thinking locates psychosis as a major life-event whose appraisal may involve all of these elements; in particular, the ability of psychosis to limit activity in the interpersonal and achievement domains, thus leading to a loss of future aspired roles or goals, and to the individual being unable to assert an identity (Rooke and Birchwood 1998).

In the following study we show that PPD can be predicted by how patients appraise the personal threat of this potentially shattering life event: in particular where the individual appraises his/her psychosis as leading to loss of social goals, roles and status; as a source of social shame; and as a diagnosis from which escape is thwarted, i. e., entrapment by a supposed malignant disorder.

Study 1: Cognitive appraisals of psychosis and post psychotic depression

In this study (Birchwood et al. 2000; Iqbal et al. 2000), a sample of 105 patients were followed up after an acute episode and monitored every 3 months for PPD and measures taken of the cognitive appraisals following recovery using the Personal Beliefs about Illness Questionnaire (PBIQ: Birchwood et al. 1993). Psychotic symptoms were assessed at each time point using the SANS and SAPS (Andreason 1982, 1986) as was insight (Birchwood et al. 1994). The first aim was to establish the course patterns of PPD and their link with positive/negative symptoms.

The definition of post psychotic depression

The ICD-10 definition of post psychotic depression (F20.4) requires that, along with general criteria for schizophrenia during the previous 12 months, the patient must still exhibit either persistent hallucinations, thought disorder or negative symptoms not due to depression or neuroleptic medication. These criteria make many of the assumptions embodied in the theories outlined in the introduction whereas in this study we are making no such assumptions and indeed we are attempting to discern empirical support for them. Hence our definition revolves around depressed mood per se.

Given that we are concerned with depressed mood and not necessarily formal major depression, the definition of caseness is arbitrary; however for reasons outlined in the introduction we used a definition of "at least moderate depression" (BDI greater than or equal to fifteen). The correlation between the self-report BDI and the interview based CDS (Calgary Depression Scale for Schizophrenia: Addington et al. 1993) approached unity ($r = 0.91$) and the thresholds proposed by Addington et al. (1993) conformed almost exactly to subgroups defined below using the BDI.

Using this definition, 70 % of the sample was depressed at onset ($n = 55$); and of this group 48 (87 %) dipped below this threshold for at least one post-recovery follow-up observation, 26 (54 %) of whom became depressed once again with the remaining 22 (46 %) staying below threshold. 5 (9 %) were depressed at all follow-up points, whilst 2 (4 %) were depressed at all but the final follow-up point. 23 were not depressed at onset; 13 (56 %) exceeded the threshold on at least one occasion (post recovery) while 9 (39 %) remained below threshold. 1 (4 %) subject became depressed at the recovery observation but remained below threshold at all future follow-up points.

Course types

Using these criteria all but 8 patients were assigned to the following groups:

Post-psychotic depression (PPD, N = 39):

I Depressed at onset, improvement and return of depression (N = 26)
II No depression at onset; depressed at one or more follow-up points (N = 13)

No post-psychotic depression (no PPD, N = 31):

III Depressed at onset; no depression throughout the follow-up period (N = 22)
IV No depression at onset or throughout the follow up period (N = 9)

Five patients did not recover from their acute illness throughout this follow-up period and a further three patients could not be assigned to the above groups. Of the

three patients unable to be assigned two were above threshold for all but the final follow-up point, and the third was depressed at the recovery observation.

For those patients with PPD, we also define the follow-up point immediately prior to its appearance as 'pre-PPD'; in other words the point in the follow-up period where the patient manifested no depression prior to the onset of PPD. The individual subject variation in the timing of PPD, and therefore pre-PPD points, would make any attempt at analysing the data virtually impossible using the original temporal sequence. We therefore examined data at the pre-PPD and PPD stages by realigning individual subject data at the appropriate follow-up points. As the average number of days from the discharge interview to the pre-PPD observation was 167.5 (SD = 124.9), the default "pre-PPD" and PPD observations for those subjects who did not develop postpsychotic depression (n = 31) were those at 4 and 8 months after recovery respectively.

PPD and psychotic symptoms

Table 1 shows the evolution of PPD (BDI score) in each of the two groups juxtaposed with that for positive symptoms (SAPS total score) and negative symptoms (SANS total score).

SAPS scores remained unchanged during the PPD episode in both group I (pre-PPD vs. PPD: $t = 1.84$, $P = 0.08$) and group II ($t = 1.34$, ns). SANS scores were also stable in group I ($t < 1$, ns) and group II ($t < 1$, ns).

While the BDI score in group I (PPD) reverted to that observed in the acute episode [25], the SAPS score rose only slightly (but none significantly). In the second PPD group (II), BDI rose to 18.2, an unprecedented rise in this particular group, and without a concomitant increase in SAPS. It should be noted that the mean level of depression during PPD was 'moderate' for the latter group and 'severe' for the former, using conventional criteria (see Williams 1992 for a review).

Overall then, in groups I and III depression and positive symptoms 'follow the same course' (combined n = 48) during the acute episode, whereas in groups II and IV (n = 22) depression and positive symptoms are desynchronous at the acute episode. However, comparing patients with and without depression at onset (BDI ≥ 15) there was no significant difference in SAPS score at onset confirming that, in general terms, any link between positive symptoms and depression is confined to a sub-group.

Table 1 Mean subgroup values for the course of depression, positive and negative symptoms across the four follow-up points

	Onset	Recovery	Pre-PPD	PPD
Group 1 (n = 26)				
BDI	25.9	12.5	9.5	24.8
SAPS	7.8	3.3	2.6	4.1
SANS	13.1	8.3	8.6	9.0
Group 2 (n = 13)				
BDI	8.7	7.1	7.8	18.2
SAPS	7.6	4.8	2.6	1.8
SANS	6.8	6.4	6.2	6.2
Group 3 (n = 22)				
BDI	26.4	14.7	7.4	8.1
SAPS	7.8	1.6	1.5	2.7
SANS	9.2	7.0	7.5	8.7
Group 4 (n = 9)				
BDI	6.0	7.6	6.3	6.2
SAPS	8.7	4.0	2.5	2.3
SANS	12.9	8.1	6.5	6.6

Who develops PPD?

Since dysphoria is a known precursor of psychotic relapse (Birchwood, Smith, MacMillan et al. 1989), eleven patients who had displayed a rise in depression during the PPD phase were found at follow-up to have suffered a further psychotic episode. These patients were rejected from the PPD group, and of the remaining 28 patients developing PPD, 68 % were also depressed at onset. 22/48 (46 %) with depression at onset did not develop PPD and, taking into account the relapse subgroup (n = 11), conversely 9/28 (32 %) with PPD were not depressed at onset. Overall the presence of depression at onset did not raise the risk of PPD ($\chi^2 = 0.63$, ns). There were no differences between the groups with (n = 28) and without PPD (n = 31) in: age at onset, duration of illness or total number of admissions; nor in gender, marital status or ethnicity; or whether under a section of the Mental Health Act.

Beliefs about psychosis and PPD

Hypotheses

1. Patients will, immediately prior to and during PPD onset, show more negative appraisals of psychosis, cognitive vulnerability and more insight, in comparison to non-PPD patients.
2. Relapsing patients will not differ in beliefs about psychosis, insight or cognitive vulnerability in comparison to patients without relapse or without PPD.
3. Patients developing PPD will show a greater propensity to describe their possible future selves in terms of "low" status roles than "high" status roles than those not developing PPD.

Table 2 shows the course of the five beliefs about psychosis measured in the PBIQ in the PPD and non-PPD groups. The factors measure patients' beliefs about their psychosis and its implications for the self as described in Rooke and Birchwood (1998): *entrapment* (control over illness); *shame*; *loss* of autonomy and valued social role; *humiliation* and loss of rank, arising from a belief in the social segregation of the mentally ill, and *attribution*

Table 2 Mean PPD (n = 28) vs. Non-PPD (n = 31) group values for cognitive appraisal of psychosis (PBIQ) across the four follow-up points

Appraisal	Subgroup	Follow-up point			
		Onset	Recovery	Pre-PPD	PPD
Humiliation (social)	PPD	4.36	3.96	3.86	3.78
	Non-PPD	3.87	4.03	3.32	3.10
Entrapment	PPD	10.37	9.21	8.86	10.07
	Non-PPD	10.06	9.71	7.52	6.90
Loss	PPD	6.71	6.75	6.93	7.39
	Non-PPD	6.90	6.84	5.61	5.29
Self vs. illness	PPD	9.04	8.68	8.25	8.21
	Non-PPD	8.90	8.45	7.22	6.71
Shame	PPD	7.25	6.64	5.61	6.32
	Non-PPD	6.87	6.35	5.48	4.77

(self versus illness as responsible for behaviour/experience).

The first hypothesis was tested using a MANOVA model with factors of group (PPD vs. Non-PPD), time (pre-PPD and PPD) and their interaction. The dependent variables were entrapment, shame, social humiliation, loss and attribution of illness to the self. A significant group effect ($F = 3.4$, $df = 5,53$, $p < 0.01$) and group x time interaction ($F = 2.5$, $df = 5,53$, $p < 0.05$) emerged.

The main test of the hypothesis is the difference between groups at the pre-PPD stage. At this stage (i. e. when neither group was depressed), the PPD group was significantly more likely than non-PPD participants to attribute the cause of psychosis to the self rather than an "externalised" illness ($F = 4.3$, $p < 0.05$); to perceive greater loss of autonomy and valued role ($F = 4.9$, $p < 0.05$); and to perceive themselves to be humiliated ($F = 4.0$, $p = 0.05$), and entrapped by their illness ($F = 3.5$, $p = 0.07$). No difference was observed between the two groups for shame ($F < 1$, ns).

At the PPD stage (i. e. while depressed) all five PBIQ appraisals were significantly more negative in those participants with PPD (entrapment: $F = 27.5$, $p < 0.001$; shame: $F = 10.3$, $p < 0.01$; social humiliation: $F = 8.0$, $p < 0.01$; illness attributed to self: $F = 9.4$, $p < 0.01$ and loss of autonomy/role: $F = 12.8$, $p < 0.01$).

Insight

The course of insight is depicted as a total insight score for both PPD and non-PPD groups in Table 3. The MANOVA model revealed a significant group effect ($F = 7.0$, $df = 3,54$, $p < 0.01$) and group × time interaction ($F = 3.0$, $df = 3,54$, $p < 0.05$). The main test of the first hypothesis is the difference between groups at the pre-PPD stage, where no difference was apparent between the PPD and non-PPD groups on any insight variables: awareness ($F = 2.8$, ns); symptom relabelling ($F = 2.6$, ns); treatment need ($F = 0.5$, ns) and total insight ($F = 0.9$, ns).

When patients were depressed however, they reported greater insight, including awareness of illness ($F = 16.2$, $p < 0.001$), relabelling of symptoms ($F = 7.3$, $p < 0.01$), need for treatment ($F = 4.2$, $p < 0.05$) and total insight score ($F = 10.1$, $p < 0.01$).

Table 3 Mean insight score for PPD (n = 28) and Non-PPD (n = 31) groups across the four follow-up points

Subgroup	Onset	Recovery	Pre-PPD	PPD
PPD	7.89	7.84	7.66	9.73
Non-PPD	7.95	7.42	6.65	6.60

PPD and the severity of psychosis

The PPD group did not experience more relapse, use of the mental health act or longer duration of illness. Indeed patients with a first episode of psychosis were more prone to PPD than those with multiple episodes (50 % vs 32 %: $p < 0.01$). Patients with PPD were no more likely to be unemployed than the non PPD group.

Comment

In accordance with the first hypothesis, those who went on to develop PPD appraised greater loss, humiliation and entrapment arising from their psychosis. Insight did not differentiate the two groups when the participants were not depressed. During PPD, patients reported greater insight into their illness, further lowering of self-esteem and hardening of the appraisals of loss, humiliation, entrapment, shame and self-blame; this is a replication of similar comparisons of depressed vs. non-depressed participants in our previous studies (Rooke and Birchwood 1998; Birchwood et al. 1993, 1994).

These results show that it may be possible prospectively to determine who develops PPD by reference to these vulnerability (appraisal) variables. The issue arises: is this vulnerability cause or effect? There are two possibilities. First the vulnerabilities we have identified may simply be markers or epiphenomena of past depressive episodes with no causal value. The high level of PPD in first episodes seen in this and other studies (Addington et al. 1998) suggests on the contrary that they can be primary. It is highly likely however that repeated depressive episodes will leave their mark on self esteem when well, who later develop PPD and that this largely revolves around how they perceive. What is remarkable is how patients' awareness of their illness ('insight') increased during PPD, supporting our belief that psychosis is indeed what patients are depressed about (Birchwood and Iqbal 1998).

The second possibility is that the vulnerabilities we have identified are a direct result of particularly adverse experiences of psychosis. In fact no difference was found between the PPD groups in duration of psychosis, num-

ber of episodes etc.; indeed patients with a first episode of psychosis were more likely to develop PPD. Thus although we are arguing that a degree of 'depressive realism' about psychosis underpins the appraisals, they do seem to go beyond patients' direct experience and engage the individual's personal interpretation or appraisal. What was not measured however was individuals' premorbid aspirations and functioning and thus the limits the illness may have 'objectively' placed on functioning.

Depression and the experience of auditory hallucinations

Auditory hallucinations are a core feature of the diagnosis of schizophrenia (Jablensky et al. 1994). They can also be found in other diagnoses (Altman et al. 1997) and in community samples who do not present to services (Poulton et al. 2000; van Os et al. 2001). The presence of auditory hallucinations in non-help seeking community samples emphasises that the experience per se is not a 'problem' but rather the distress they can give rise to, often is. In previous work, we reported that over 60 % of voice hearers were 'severely depressed' as measured by the Beck Depression Inventory and over 75 % reported that they were 'highly distressed' by the experience (Birchwood et al. 2000). Our cognitive model of voices that we focus on here attempts to understand the factors which maintain the distress and depression attached to voice hearing.

Recent research on psychosis applying this integrated cognitive and social mentalities approach, with a focus on the social rank mentality (for recognising and organising dominate-subordinate interactions) and social power (Birchwood et al. 2000) has found that it is voice hearers' appraisal of the power and omnipotence of voices (and their own subordination to them), which determines their response, irrespective of the content. Thus, voices perceived as powerful and malevolent were at first resisted, but ultimately submitted to or appeased; 'benevolent' voices on the other hand, were courted and usually complied with (Beck-Sander, Birchwood and Chadwick 1997; Birchwood and Chadwick 1997). In a study of 70 voice hearers (Birchwood and Chadwick 1997), it was found that: (a) beliefs rather than content governed the response, and (b) the high rate of depression in this sample (60 %) was directly attributable to the belief in the power of voices, and not voice frequency, loudness or indeed content. This work has been independently replicated (Close and Garety 1998, Sayer et al. 2000; van der Gaag et al. 2003) and in a subsequent study, it was found that these appraisals largely governed individuals' response to commanding voices, rather than the command itself (Beck-Sander et al. 1997; Trower et al. in press).

A key question arises: what guides the patient's automatic tendency to feel subordinate to a voice and to perceive it to be a dominant-omnipotent entity? Our first insight was to appreciate that the patient personifies his voices and develops an interpersonal relationship with them. We have argued that this interpersonal relationship a voice hearer has with his voice is partly shaped via recruitment of a specialised social processing system ('social mentalities') that act as guides for social roles and scripts (e. g. from early attachment). Birchwood et al. (2000) and Gilbert et al. (2001) found that there was a significant relationship between how voice hearers experienced relationships with others in their everyday lives (e. g. as relatively powerless, inferior and subordinated) and how subordinated and powerless they felt with their voices.

There are a number of ways these key processes (e. g. voice-self relationships and depression) may operate (Fig. 1). First, it is possible that the depression evident in this group is primary and 'drives' the linkage between hostile voice and subordinate self, i. e. the more depressed one is, the more inferior one feels to voices and others and the more distress voices will create. In support of this, recent factor analytic studies of psychotic symptoms have identified depression as a distinct dimension of psychosis, alongside the 'positive' and other symptoms, with its own regulators (Stefanis et al. 2002). Second, it could be that a (delusional) belief in voice power elicits a subordinate self-perception and this, in the context of more severe (e. g. frequent) hallucinations, leads to depression and general distress. The second model suggests that the greater the frequency and loudness of the voices, the greater the resulting distress and depression. In other words, the more severe the voices and power beliefs, the greater the distress. The third (hypothesised) model predicts that feeling inferior acts as the social mentality and psychobiological template to others in general, and this sets the stage for the appraisal of voices as dominant, powerful and threatening, and the self as a subordinate, relatively powerless to resist or defend against the insults and shaming attacks of a voice. It is this trapped, shamed, beaten-down and threatened social mentality which we hypothesise gives rise to distress and depression attached to voices.

With the development of new statistical techniques it is now possible to test these models using covariance structural equation modelling (SEM). We used SEM because it enables each of the 'paths' postulated by each model to be tested (Ullman 2000). SEM cannot 'prove' that a particular model is true, but it can reject competing models; this is analogous to the scientific method (Shadish et al. 2001).

Study 2: Depression and auditory hallucinations: the role of social appraisal

In this study (Birchwood et al. 2004), we tested the hypothesis that patients who perceive themselves to possess low social power and social status ('rank'), provides a template leading them also to perceive themselves to be subordinated to their dominant voice, i. e. the pa-

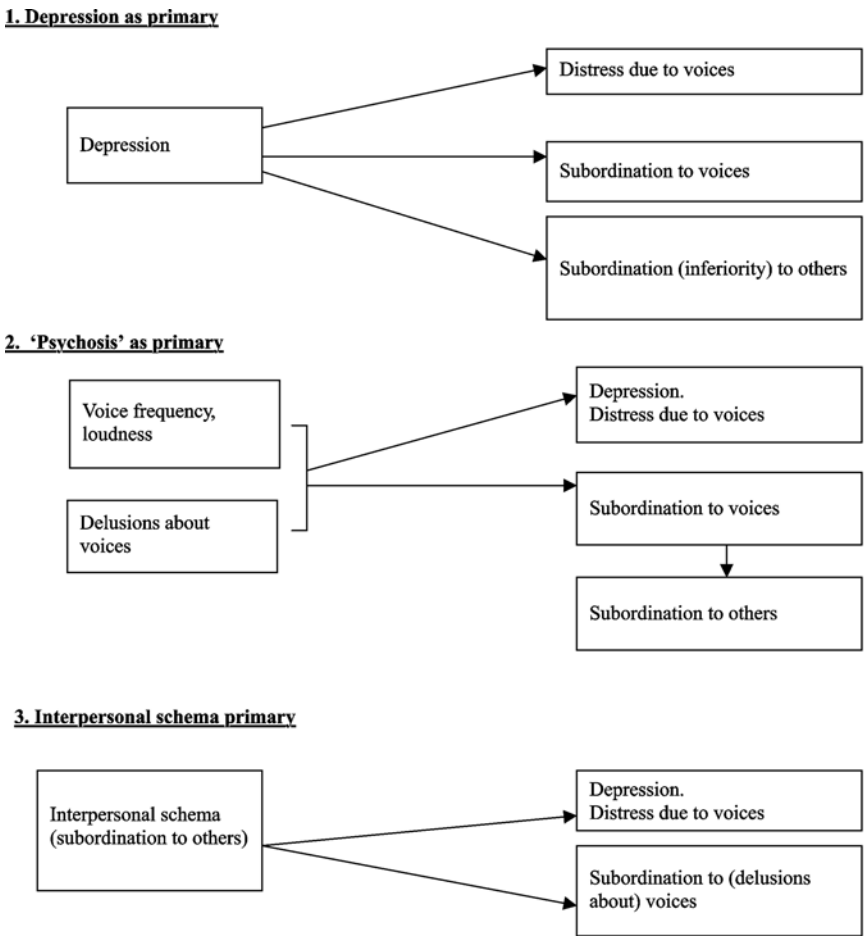

Fig. 1 Three models of distress arising from voices

tient's voice, and his social peers, be appraised as more powerful and with higher social status than himself. If confirmed, we then proceed to test the three causal models outlined in Fig. 2.

In this study then, measures of social status and power were applied to two kinds of social relationship: with the dominant voice and with significant others.

Participants

A total of 125 people consented to participate in the study and completed the protocol. The mean age was 33.7 yrs (SD: 9.3). All patients had heard voices for at least one year and had a clinical diagnosis of schizophrenia and symptoms recorded at the previous acute episode conformed to ICD-10 criteria for schizophrenia (n = 81), schizophreniform disorder (n = 15) or paranoid psychosis (n = 29).The measures used are indicated below: for a full account, the reader is referred to Birchwood et al. (2000, 2004).

Interpersonal schema and the relationship with the dominant voice

(i) Social Power (SPS: Birchwood et al. 2000)

Consistent with our previous research, patients who perceived themselves to have lower social power also perceived themselves to be more subordinated (powerless) relative to their voice ($p < 0.01$) (see Table 4).

(ii) Social rank (SCS: Gilbert and Allen 1992)

Table 4 also shows that where individuals perceived themselves to be of low social status, this was mirrored in their relationship with their dominant voice.

(iii) Distress attributed to hearing voices (Hustig and Häfner 1990)

Those who appraised the voice with higher power and status than themselves were significantly more distressed by their voices (Table 4).

Model testing

Covariance structural equation modelling (SEM) was used to test goodness of fit with each of the following models, in addition to the null model (see Fig. 2).

1. Depression is primary and leads to the appraisal of social rank, voice power and distress.
2. Greater voice activity (frequency, audibility), and the presence of delusions about voices leads to depression and the appraisal of voices' power.
3. Social rank and social power leads to the appraisal of

Table 4 Comparison of patients appraising their voices with 'high' vs 'low' rank

		Social Power[2] (S. P.D)	Social Rank[3] (S. C.S)	Depression (BDI)	Distress (Hustig and Hafner)
Voice Social Rank (VRS)					
High[1] (n = 69)	Mean (SD)	23.1 (4.3)	34.2 (7.7)	27.4 (16.6)	4.3 (0.9)
Low (n = 57)	Mean (SD)	26.5 (4.2)	25.4 (9.1)	16.6 (9.8)	3.3 (1.4)
	p	< 0.01	< 0.01	< 0.001	< 0.01
Voice Power[1] (V.P.D.)					
High (n = 65)	Mean (SD)	23.2 (3.6)	30.2 (7.7)	27.4 (10.8)	4.3 (1.1)
Low (n = 60)	Mean (SD)	26.9 (4.1)	22.9 (9.9)	15.6 (10.7)	3.3 (1.5)
	P	< 0.001	< 0.01	< 0.001	< 0.01

[1] NB. Where the perceived power of the voice is *high*, the patient feels himself to be relatively powerless
[2] *Lower* scores indicate lower social power
[3] Higher scores indicate lower social rank

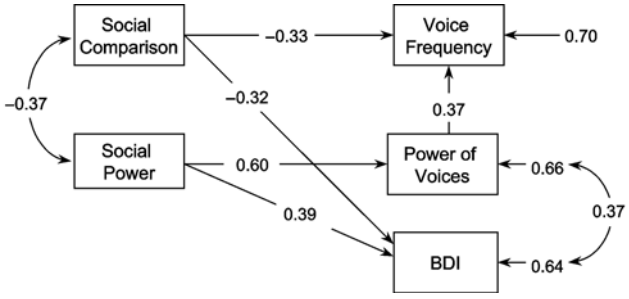

Fig. 2 S.E.M results-path diagram. Model 2 – Social Rank and power primary ($\chi^2 = 1.2$, df = 3, p = 0.75, standardised estimates shown: all parameter estimates statistically significant at p < 0.005.)

voice power, distress and depression (the hypothesised model).

Maximum likelihood estimation of the models was carried out, based on the covariance matrices using LISREL 8.51 (Jöreskog and Sörbom 1999). Previous research findings indicate that a correlation between social power and social comparison would be expected (Birchwood et al. 2000). In addition, a correlation between depression and power of voices was expected (Birchwood and Chadwick 1997; Birchwood et al. 2000; Gilbert et al. 2001). The perception of the frequency of voices has been shown to be influenced by their perceived power; for example, the perceived power of the voice increases vigilance, thus giving rise to a greater awareness of voice activity (Birchwood et al. 2000). These three parameters were therefore added to all models (where appropriate).

Table 5 shows the results for each model. These show that the null model (no relationship between variables), model 1 (depression primary) and model 2 (psychosis primary) were each rejected. The hypothesized model 3 (social rank and power primary) failed to be rejected and provided the best fit to the data.

Comment

The direction and magnitude of the path coefficients show that the appraisal of social power and status had a major influence, in statistical terms, on the voice hearer's appraisal of the power and influence of his voices. The SEM results also revealed that the high rate of depression in this sample flowed from these social status focused, social and interpersonal beliefs (SCS, SPD). It should be noted that in this, as in our previous research, depression was strongly correlated with voice power; the SEM results suggest that the key variables underlying this are the broader social and interpersonal schema held by the voice hearer.

In general then, these data support our contention that the distress occasioned by persisting symptoms operates through a 'psychological filter' and is not directly attributable to the topography of those symptoms themselves.

Study 3: Depression in acute schizophrenia: the role of the appraisal of threat

In this preliminary study we investigate the hypothesis that depression in acute schizophrenia may arise from similar psychological processes to those identified in PPD and in 'voices' (studies 1 and 2 above). We argue that the presumed threat from persecutors to the individual's well being is sufficient to trigger depression and fear, particularly if the patient feels defenceless (i. e. without protective 'safety behaviours'). Freeman et al. (2001) have looked at the relationship between patients and their persecutors and found higher depression associ-

Table 5 Results of the covariance structure model

Model	χ^2 (df)	P	RMSEA	NFI	CFI
Null	171 (9)	< 0.001	0.45	0.00	0
Model 1: Depression Primary	37 (4)	< 0.001	0.26	0.76	0.77
Model 2: Psychosis primary	60 (6)	< 0.001	0.28	0.74	0.76
Model 3: Social Rank and power primary	1.2 (3)	0.75	0.00	0.99	1.00

ated with a perception that persecutors are more powerful and omnipotent and unable to defend against supposed threats.

Second, we have shown that during acute schizophrenia, insight is not completely lost (Birchwood et al. 2000) and this available awareness of illness may be sufficient to trigger a dysphoric response during the acute episode if the individual appraises psychosis with the cognitions identified in study 1.

Hypotheses:

1. Patients attributing greater power and omnipotence to persecutors and who have fewer 'safety behaviours' will be more depressed, independent of positive symptoms.
2. Patients appraising their psychosis with greater 'loss', 'shame' and 'entrapment', will report greater depression.

This study examines these hypotheses in acute, first episode psychosis conforming to ICD-10 criteria for schizophrenia. Sequential referrals to the Early Intervention Service in Birmingham, with a first episode of psychosis were invited to participate in the study. Assessment included: SCAN (WHO 1998), PANNS (Kay 1987) interviews, Calgary Depression Scale for Schizophrenia (Addington and Addington 1993), Details of Threat Questionnaire and Safety Behaviours Questionnaire (Freeman 2001) together with voice appraisals and appraisal of psychosis.

To date, 37 patients have been identified: 26 patients completed the protocol; 5 refused to participate, 4 were not first episode and 2 had no psychotic symptoms. The mean PANNS positive score was 19.24.

Initial results show 66 % of patients were significantly depressed, scoring 7 or more on the CDSS (CDSS scores of 7 and above will have an 82 % specificity had 85 % sensitivity for predicting a moderate depressive episode: Addington and Addington 1993).

Hypothesis 1

Significant correlations were found between depression and the perception of the degree of threat attributed to the persecutors ($r = 0.71$; $p \leq 0.01$) and the perceived ability to escape and control the threat ($r = 0.49$; $p < 0.05$), independent of the level of PANSS positive symptoms.

These preliminary results suggest that how one appraises the threat from persecutors is complex and very relevant to the development of depression in the acute episode. If threat is perceived to be high, the likelihood of depression is higher; however this appears to be mitigated by different reactions to threat: particularly the perceived opportunities for avoidance and escape from the persecutor.

Hypothesis 2

Patients appraising their psychosis as embodying greater 'loss', 'shame' and 'entrapment' were at significantly greater risk of depression ($r = 0.66$, 0.58 and 0.59 respectively; all $p < 0.001$), independent of PANNS symptoms.

Comment

These preliminary findings offer an original perspective on the depression dimension in acute psychosis and are in line with the general theme of this paper: namely that it is the way the patient appraises the meaning and significance of the psychotic experience, and of the presumed diagnosis, for his present and future well-being, that underlies the development of depression and distress; and that this is the case even during the acute phases of illness where insight is not totally lost.

Implications for treatment

A recent systematic review of the efficacy of antidepressants for treatment of depression in schizophrenia (Whitehead et al. 2003) concluded that the evidence was weak and affected by publication bias. The data presented here suggest that cognitive therapy may be well suited to this task by focusing on patients' appraisals (beliefs) of the threat to well being posed by the diagnosis, by supposed persecutors and by perceived social shame. We believe that therapy aimed at promoting adaptation to psychosis must focus exclusively on the resolution of appraisals of shame and entrapment by psychosis, in a coherent and theoretically driven way. 'Scattershot' approaches involving many components, for example psychoeducation, supportive psychotherapy, relapse prevention etc., do, we believe, risk at best losing the focus on these crucial core appraisals and at worst simply confusing the client. In relation to depression arising from individual symptoms, these findings point strongly to a focus on the perceived power of persecutors, including voices, to harm or shame the individual. This is the approach we recently took in a trial attempting to reduce compliance with commanding voices: this showed that, in addition to reducing compliance, the cbt prevented the escalation of depression and reduced distress (Trower, Birchwood, Meaden et al. 2004). The efficacy of cbt with psychotic symptoms needs now to be applied to depression and these studies have hopefully clarified the specific cognitions that need to be the focus of therapy.

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