

Pathways to emotional dysfunction in first-episode psychosis

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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 is pervasive in non-affective psychosis. Sometimes (and unhelpfully) referred to as ‘comorbidity’, these disorders include depression, usually accompanied by hopelessness and suicidal thinking; social anxiety, usually accompanied by social avoidance and problems in forming relationships; and traumatic symptoms (post-traumatic stress disorder, PTSD). There is also the distress (fear, anger, shame) attached to the experience of psychotic symptoms. Emotional dysfunction, in common with the core symptoms and disabilities, develops rapidly and aggressively during the prodrome and early phase (Harrison *et al*, 2001). Following the first episode, more than half of patients report ‘post-psychotic depression’ during a period which carries a high risk for suicide (Birchwood *et al*, 2000a); more than a third report traumatic reactions sufficient to qualify for PTSD, particularly unwanted intrusions of images surrounding the first episode and its treatment (McGorry *et al*, 1991); and nearly half display a ‘marked and persistent fear of social interaction’, i.e. social anxiety disorder (Cosoff & Hafner, 1998). Making clear diagnostic distinctions between these emotional disorders is not easy even in people without psychosis, for example, where PTSD and depression overlap.

It is tempting to characterise these emotional problems as simply part and

parcel of psychosis and the loss of functioning that accompanies it, and it is perhaps for this reason that the pathogenesis of these emotional difficulties is not understood and few effective treatments are available. This is particularly important since the presence of these disorders increases the probability of early relapse and their presence during the prodrome may act as a risk factor for transition to psychosis.

How are we to make sense of these disorders of emotion? I believe that in order to improve our understanding and to develop new treatments we need to make a clear distinction between three core (but not mutually exclusive) pathways: emotional disorder that is intrinsic to the psychosis diathesis, a psychological reaction to it, or the product of disturbed developmental pathways.

EMOTIONAL DISORDER AS INTRINSIC TO PSYCHOSIS

The clearest example of an emotional disorder intrinsic to psychosis is depression. The second generation of factor analytical studies of psychotic symptoms yield an additional dimension of depressive symptoms alongside the positive and negative dimensions. When orthogonality is not imposed on these factors, these dimensions co-vary within patient and community samples (Stefanis *et al*, 2002). Depression is nearly always part of the first-episode prodrome that recedes with the positive symptoms (Birchwood *et al*, 2000a). The process underlying this link between positive symptoms, negative symptoms and depression is unknown, but biological and psychological processes are plausible. The therapeutic implications of this pathway to emotional disorder lie in the treatment of core psychotic symptoms.

EMOTIONAL DISORDER AS A PSYCHOLOGICAL REACTION TO PSYCHOSIS

Psychosis and psychotic symptoms can be considered as a challenging or traumatic life event that requires adaptation by individuals and their families. Post-psychotic depression is known to occur some months after recovery from the acute episode and has been shown to be predicted by how patients appraise the personal threat of this shattering life event: the individual’s appraisal of psychosis as leading to loss of social goals, roles and status, as a source of shame, and as a state from which escape is thwarted, i.e. entrapment by a supposedly malignant disorder, predicts the later emergence of post-psychotic depression with hopelessness (Birchwood *et al*, 2000a). First episodes were followed by a higher rate of post-psychotic depression (over 50%), linked to heightened awareness of the diagnosis and its implications. Where symptoms persist, depression has been traced to the perceived power of voices (Birchwood *et al*, 2000b) and of persecutory delusions (Freeman *et al*, 2001), and to the subjective experience of negative symptoms. In general, the distress occasioned by persisting symptoms has been shown to operate through a ‘psychological filter’: patients with more-positive self-schemas seem better able to withstand the threat of voices or other persecutors (Birchwood *et al*, 2000b; Freeman *et al*, 2001).

Traumatic reactions do not appear to be linked with the ‘objective’ trauma of psychosis such as compulsory admission, as would be required for a DSM-IV diagnosis (McGorry *et al*, 1991; American Psychiatric Association, 1994). In non-psychotic PTSD, attention now focuses on the perceived threat of traumatic events and how people cope; in psychosis, patients may perceive themselves to be at risk of injury or death from supposed persecutors (Freeman *et al*, 2001), voices (Birchwood *et al*, 2000b) or from others in a disturbed psychiatric ward, but the impact of this perception on trauma is as yet unknown.

With regard to social anxiety, it is known that patients perceive themselves to be shamed and socially subordinated by others because of their psychosis and patient status (Haghighat, 2001): in non-psychotic social anxiety, patients fear criticism and humiliation in social encounters, which drives social avoidance (Clark,

2001). It has been argued that a similar process, namely social shame and fear of discovery, may also underpin avoidance in psychosis (Birchwood *et al*, 2000b). In a similar vein, the problems of fear and social avoidance in the context of active psychotic symptoms may be traced to the supposed threat posed by others: patients with persecutory delusions often deal with the perceived threat to their well-being through avoidance of high-risk social encounters; in cognitive therapy this is one of a class of 'safety behaviours' which function to reduce threat (Freeman *et al*, 2001). Social disengagement can also be traced to the content of command hallucinations that can directly undermine trust in others.

The therapeutic emphasis in this pathway focuses on patients' appraisals (beliefs, cognitions) of the threat posed by the diagnosis, by voices and other persecutors and by perceived social shame.

DISORDERS OF EMOTION ARISING FROM DEVELOPMENTAL ANOMALY AND TRAUMA

Birth cohort and retrospective studies (e.g. Isohanni *et al*, 1998) reveal that first-episode psychosis is often preceded by social difficulty and emotional disorder as well as by low-level 'psychotic' experience stretching back into early adolescence (Poulton *et al*, 2000). These childhood antecedents of a developing psychosis will unfold in a social environment and there is now considerable evidence that social factors influence morbidity and outcome of psychosis, for example urban living, particularly deprivation, membership of marginalised social groups, the impact of migration and the (favourable) correlates of 'developing-nation' status (Harrison *et al*, 2001). These unfolding antecedents of psychosis and the social risk factors will also affect 'normal' social and psychological development, leading to low self-esteem, difficulty in establishing relationships and susceptibility to stress. The science of 'developmental psychopathology' (Rutter, 2000) shows that there exists continuity between adolescent and adult emotional disorder, including depression and risk of suicide, which occurs in a dimensional and not a categorical way, influenced by social and familial context (Fombonne *et al*, 2001). Discontinuity between adolescent and adult emotional functioning is also possible; for example,

Andrews & Brown (1995) showed that positive life events in late adolescence can help to restore a disturbed developmental trajectory to within normal limits. The domains of emotional functioning in adolescence also interact; for example, social anxiety increases the developmental risk of adolescent depression.

Developmental trauma and difficulty can act as risk factors for adult emotional disorder: for example, childhood abuse or neglect and problems of parental attachment can predispose to adult depression. There is evidence of a high rate of traumatic histories in people with psychosis, including sexual trauma and emotional neglect, unwanted pregnancy and dysfunctional parental attachment (e.g. Greenfield *et al*, 1994). These traumatic histories may also render patients prone to post-psychotic depression and other emotional disorders: in PTSD, for example, traumatic responses to violent crime have been shown to be more likely in people with a history of childhood trauma, who appraise the traumatic event as more personally threatening (Andrews *et al*, 2000).

In the cognitive framework, traumatic histories and developmental anomalies may influence the cognitive schemas that govern the processing of self and social information. Such schemas have been observed to be active in the emotional response to psychosis in the way in which the person hearing voices appraises their interpersonal significance (i.e. their power and omnipotence; Birchwood *et al*, 2000b), and also in the distress and persistence of voices in young adolescents (Escher *et al*, 2002).

In sum, this pathway to emotional disorder in first-episode psychosis involves developmental disturbance triggered by an emerging psychosis, childhood trauma, or both, leading to dysfunctional cognitive schemas that affect adaptation to psychosis and its symptoms, and to adolescent emotional disorders that continue into adulthood. The therapeutic implications of this pathway lie in the focus on disturbances in 'normal' developmental processes in adolescence and their continuity with emerging psychopathology, with a particular therapeutic focus on dysfunctional schemas of self and others.

CONCLUSIONS

Distress from psychotic experience and the disorders of emotion in first-episode

psychosis may arise from three overlapping processes: those that are intrinsic to psychosis, those that are a psychological reaction to psychosis and patienthood, and those arising from anomalies of childhood and adolescent development, triggered by an emerging psychosis, childhood trauma or both. The application of cognitive-behavioural therapy (CBT) in psychosis focuses on reducing psychotic symptoms as a means of alleviating the distress and emotional problems to which they give rise. The outcomes of CBT for psychosis have not shown any consistent effect on depression or other emotional dysfunction; the same can be said for neuroleptic pharmacotherapy. This analysis suggests a complementary and perhaps more fruitful focus on the concomitant emotional dysfunctions and their developmental and psychological origins. This will require not only the adaptation of tried and tested CBT developed for non-psychotic emotional disorder, but also – crucially – a recognition that the powerful evidence for biological processes in psychosis sits comfortably beside the findings in developmental psychology and psychopathology; and patient services have much to gain from a cross-fertilisation of approaches between child and adult mental health services in the management of first-episode psychosis.

DECLARATION OF INTEREST

None.

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