The New Psychology and Treatment of Psychosis

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Our understanding of the psychotic disorders is undergoing a remarkable transformation. Twenty years ago, it was widely accepted that diagnoses such as "schizophrenia" and "bipolar disorder" identified a series of discrete conditions which were mainly caused by genetically-determined neurocognitive dysfunction, and which were resistant to psychological intervention. Of course, this view is still held by many psychiatrists, but a critical mass of conceptual arguments and empirical findings now challenge the conventional account of psychosis, and promise to bring about a paradigm shift.

For example, it is now evident that the neo-Kraepelinian diagnostic system, enshrined in successive editions of the American Psychiatric Association's Diagnostic and Statistical Manual (American Psychiatric Association, 2000) has almost no scientific or clinical value. Research demonstrating that hallucinations and delusions are much more widely experienced in the general population than was hitherto thought (van Os, Hanssen, Bijl, & Ravelli, 2000), and that these symptoms exist on continua with more mundane types of experience (Claridge, 1990), has shown that it is misleading to draw a clear line between "schizophrenia" and normal functioning. The parallel discovery that soft bipolar symptoms are widely experienced (Angst, 2005) and that hypomanic personality traits are approximately normally distributed (Meyer & Keller, 2003) has similarly dissolved the boundary between bipolar disorder and ordinary mood states. At the same time, the existence of patients with mixed bipolar and schizophrenia symptoms (Kendell, 1991) and others whose symptoms stride the distinction between bipolar and unipolar depression (Akiskal, Benazzi, Perugi, & Rihmer, 2005) implies that psychotic conditions cannot be subdivided into neat categories. More crucially from the point of view of the patient, diagnoses appear to be poor predictors of response to antipsychotic medication and mood-stabilizing drugs (Johnstone, Crow, Frith, & Owens, 1988).

Recent genetic research has added to the woes of the neo-Kraepelinian system. Molecular studies have failed to find genes of major effect for any diagnosis, and those genes of minor effect that have been identified seem to confer risk for a variety of conditions (Craddock & Owen, 2005). Moreover, it is clear that numerous methodological errors have led researchers to exaggerate the role of genetic causation and neglect the extent to which environmental factors are also important (Marshall, 1990). Foremost amongst these is the flawed assumption that apparently high heritability estimates (often calculated on the basis of flawed data; (Joseph, 2003)) preclude strong environmental determinants (Dickins & Flynn, 2001). Reflecting this assumption in their recent attempt to defend the schizophrenia concept, American psychiatrists Robert Leiberman and Michael First argued that "Schizophrenia is not caused by disturbed psychological development or bad parenting" (Lieberman & First, 2007). However, there is compelling evidence that being unwanted at birth (Myhrman, Rantakallio, Isohanni, & Jones, 1996), early separation from parents (Morgan et al., 2006), sexual abuse (Bebbington et al., 2004; Morrison, Read, & Turkington, 2005) and other kinds of adverse family relationships (Wahlberg et al., 1997) all increase the risk of psychosis in adult life. Interestingly, there appear to be specific associations between chronic victimization and paranoia (Janssen et al., 2003) and between sexual trauma and hallucinations (Hammersley et al., 2003; Read, Agar, Argyle, & Aderhold, 2003).

The role of neurocognitive dysfunction in psychosis is far from clear. Although high risk of psychosis is associated with low IQ (Davidson et al., 1999), in comparisons between schizophrenia and bipolar patients similarities in neuropsychological functioning are more evident than differences (Hoff et al., 1990; Nuechterlein, Dawson, Ventura, Miklowitz, & Konishi, 1991). Moreover, neurocognitive impairment seems to play little or no role in positive symptoms (Green & Nuechterlein, 1999; Keefe et al., 2006). When individual symptoms of psychosis have been studied, specific biases in reasoning and information processing have been identified. For example, paranoid delusions are associated with a tendency to jump to conclusions when reasoning about sequentially presented information, a bias towards explaining negative events in terms of external factors, and an impoverished ability to understand the intentions of others (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001). Verbal hallucinations, on the other hand, seem to be the consequence of misattributing inner speech (verbal thought) to external sources, which in turn appears to reflect an impairment in monitoring the source of experiences (Bentall, 2000). Elsewhere, I have argued that it may be possible to construct a complete and satisfactory account of severe mental illness by studying the psychological processes involved in individual symptoms; once all the symptoms have been explained in this way there will be no schizophrenia or bipolar disorder left over to explain afterwards (Bentall, 2003).

It is against this background that the new psychological interventions for psychosis – especially cognitive behaviour therapy – have been developed. Indeed, many clinicians working on the mechanisms involved in hallucinations, delusions and other psychotic symptoms have been involved in the development of this type of treatment. The contributors to this special issue on psychosis clearly describe the substantial achievements in treatment that have been made so far. Although recent meta-analyses confirm that CBT for psychosis is effective, this is no time for complacency, as the impact on symptoms is greater than the impact on relapse, and the effect sizes achieved in the largest and most methodologically sound trials are modest (Pilling et al., 2002; Tarrier & Wykes, 2004). Future research that further advances our understanding of the psychological mechanisms that give rise to symptoms is likely to inspire further innovations in treatment, thereby improving the quality of life of patients who have been ill-served by conventional psychiatry.

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Ricard P. Bentall is a professor of clinical psychology at the University of Manchester. Much of his empirical research has focused on psychological mechanisms responsible for specific psychotic symptoms. In studies of auditory hallucinations he has explored cognitive failures which lead the hallucinating individual to misattribute their inner speech to an external source. In studies of persecutory delusions, he has investigated social reasoning (especially attributional and theory of mind) biases which lead the deluded person to attribute malevolent intentions to others. Recently, this work has been extended to examine mechanisms responsible for manic symptomatology. He has also carried out research into the treatment of chronic fatigue syndrome.

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