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DISCUSSION COMMENTARIES

MADNESS AND HYPNOSIS

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Introduction

In his paper, Wagstaff gives a detailed account of arguments considered in a now-famous High Court action, in which lawyers for Christopher Gates claimed that his schizophrenic illness had been caused by his participation in a stage show conducted by the hypnotist Paul McKenna (Wagstaff, 2000). The trial provided a remarkable opportunity for the causes of psychosis to be debated in public. Yet, the arguments made by lawyers for the plaintiff and the defendant created more fog than light. For this, the lawyers cannot be faulted. Their muddle and confusion reflected assumptions about severe mental illness that have been accepted uncritically by most psychologists and psychiatrists for more than a century. Tragically, this muddle and confusion has impeded the development of an adequate theory of madness, and so delayed the emergence of rational and humane treatments for people who are mentally ill.

Is schizophrenia a meaningful scientific entity?

A central assumption adopted by both the plaintiff and by Wagstaff in his commentary is that schizophrenia is a meaningful scientific entity, with properties that can be specified and compared with the properties of hypnosis. Many of the arguments pursued by the lawyers focused either on symptoms that were said to be common to schizophrenia and hypnosis, or on putative similarities at the neurophysiological level. For example, it was argued that hallucinations and other perceptual changes are a common property of both conditions, and that both involve the activation of inhibitory circuits in the frontal lobes.

The celebrated biologist C.H. Waddington (1977) used the term COWDUNG to refer to the conventional wisdom of the dominant group. In the case of schizophrenia, the dominant group has consisted of psychiatrists who have embraced and elaborated the system of psychiatric classification first developed towards the end of the nineteenth century by Emil Kraepelin (1899/1990). The group of American psychiatrists who developed the influential third and subsequent editions of the American Psychiatric Association's *Diagnostic and Statistical Manual* (DSM) explicitly styled themselves 'neoKraepelinians' (Blashfield, 1984). One of their number, Gerald Klerman (1978), went so far as to propose a neoKraepelinian manifesto. The three most important tenets of the manifesto were:

1. There is a boundary between the normal and the sick.
2. There are discrete mental illnesses.
3. The focus of psychiatric physicians should be particularly on the biological aspects of mental illness.

The concept of schizophrenia exemplifies these assumptions. When first describing the disorder under the term 'dementia praecox' (senility of the young), Kraepelin believed that he had identified a cluster of symptoms that occurred together and which had a predictable (poor) outcome. Most psychiatric researchers since his time have either tacitly or explicitly accepted this model. For this reason, the most common research design adopted by psychopathologists studying psychosis has involved comparing patients who have a diagnosis of schizophrenia with various control groups (patients with another diagnosis, normal individuals, or both), on the assumption that the patients with schizophrenia share something in common which has aetiological significance and which is absent in the comparison groups. I have detailed the folly of this approach elsewhere (Bentall, 1990, 1993). In brief, empirical research shows that the tenets of Klerman's manifesto provide a poor basis for developing a scientific account of madness.

First, over the past decade or so it has become apparent that there is no clear dividing line between madness and sanity. Population surveys show that a sizeable minority of individuals who have not sought psychiatric help experience 'first rank' psychotic symptoms such as hallucinations (Tien, 1991) or delusions (Verdoux, Maurice-Tison, Gay, Van Os, Salamon and Bourgeois, 1998). An even more substantial proportion of the population report 'schizotypal' traits, which seem to be attenuated versions of the symptoms reported by patients with schizophrenia (Claridge, 1990). It therefore seems that we must recognize 'the essentially dimensional nature of disorder with severe schizophrenia at one end of that dimension and somewhere along that dimension the milder madnesses of belief in horoscopes and magical intervention before reaching the sanity of the less interesting members of the population' (Venables, 1990: 294).

Second, the division of the psychoses into the two main categories proposed by Kraepelin – dementia praecox and manic depression – has seemed increasingly problematic. Schizophrenia seems to consist of a number of clusters of relatively independent symptoms (Liddle, 1987), which are also found in other diagnostic groups. Not surprisingly, many patients show a mixture of affective and psychotic symptoms, leading some authors to propose that there exists a schizoaffective spectrum of conditions, instead of the discrete illnesses envisaged by Kraepelin (Crow, 1991; Brockington, 1992). Alarmingly, for patients seen by psychiatric services, neoKraepelinian diagnoses do not seem to be a good predictor of response to psychiatric drugs (Johnstone, Crow, Frith and Owens, 1988). In the light of these observations, a number of researchers, including myself, have argued that psychopathologists should pay less attention to broad diagnostic categories and should instead attempt to construct explanatory models of specific psychotic symptoms such as hearing voices, paranoid beliefs or incoherent speech.

If schizophrenia is a disease with no particular symptoms, which has no particular course, no particular outcome, and responds to no particular treatment, it is not surprising that aetiological research has so far failed to find evidence that it is the product of a specific cause (Bentall, Jackson and Pilgrim, 1988). Under these circumstances it is difficult to see what can be gained by comparing the properties of schizophrenia with the properties of hypnosis (a phenomenon which is also poorly understood). Indeed, the barristers who debated this issue in the High Court might be seen as victims of an unintentional con, perpetrated by 'experts' who have continued to adhere to a paradigm which is well past its use-by date.

Could Gates have been driven mad by his experiences on stage?

I turn now to the third of Klerman's manifesto points. Recently, it has become fashionable to regard psychosis as the outcome of faulty neurodevelopment (Keshavan and Murray, 1997). There is some merit in this approach, as there is consistent evidence of markers of early brain damage in children who later become psychotic (Green, 1998). However, these findings are non-specific and have also been found in people who later develop affective disorders (Nasrallah, 1997). Moreover, the strength of these effects seems to be quite modest. The best interpretation of the evidence is that early brain damage confers a mild and non-specific risk of serious psychiatric disorder in later life.

The neoKraepelinian commitment to a biological approach has impeded research into social and environmental determinants of psychosis. Indeed, textbooks commonly suggest that such determinants either do not exist or are yet to be discovered. In fact, when relevant research has been carried out, remarkably consistent evidence that life experiences contribute to the onset of psychosis has been collected.

Some well-designed prospective studies have implicated the family environment in psychosis (Goldstein, 1987; Wahlberg et al., 1997). However, for the purpose of this discussion I would like to draw attention to research indicating that trauma can play a role in bringing about psychotic symptoms. In a review of the literature on women with severe mental illness, Goodman, Rosenberg, Mueser and Drake (1997) found that 51–97% have experienced severe sexual or physical abuse at some time in their lives. This finding has been replicated in well-designed studies carried out since. For example, in a survey of more than 200 severely ill psychiatric patients, Mueser et al. (1998) found that 52% of the women and 35% of the men had been sexually abused in childhood. In this study, only 2% of patients had not experienced some kind of severe trauma at some point in their lives. Although only 3% of the patients had a concurrent diagnosis of post-traumatic stress disorder (PTSD) in their notes, 40% of those with a primary diagnosis of manic depression and 28% of those with a primary diagnosis of schizophrenia also met the diagnostic criteria for PTSD. These figures are so much higher than those found in the general population that they cannot be dismissed lightly.

These findings have been corroborated by studies in which trauma survivors have been followed up to observe their symptoms. Positive symptoms of psychosis (hallucinations and delusions) have been found in victims of sexual abuse (Ensink, 1993; Ross, Anderson and Clark, 1994) and in survivors of warfare (Butler, Mueser, Sprock and Braff, 1996). Interestingly, a number of these studies report that the severity of positive symptoms correlates with the severity of the trauma experienced.

In citing this evidence I do not mean to imply that trauma is *the* cause of psychosis. However, it is clear that adverse life experiences can play an important role in the complex network of causal relations that culminates in madness. This brings me back to the case of Christopher Gates. Whereas we can be certain that hypnosis does not, in general, lead to psychotic states, I do not see how we can be sure that the events on the night of 10 March 1994 – which may have been experienced by Gates as quite traumatic – did not play a role in triggering his psychotic episode. A court, on listening to the evidence I have just outlined, may well have reached the conclusion that such a triggering effect was unproved, but who is to say for certain that it did not happen?

Conclusions

Scientific evidence is always vulnerable to misinterpretation in the courtroom. However, this problem is accentuated when the evidence in question has been collected and interpreted in the context of a failed paradigm. In this case, unquestioning acceptance of the COWDUNG model of psychosis resulted in a muddled debate that was incapable of shedding light on an issue of importance not only to Gates, but also to society as a whole.

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