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The Kraepelinian dichotomy

McDonald *et al* (2005) investigated the Kraepelinian dichotomy of psychosis using brain imaging. They reported distinct grey matter volumetric deficits in patients with schizophrenia and those with psychotic bipolar I disorder but common white matter abnormalities in the two disorders.

Kraepelin distinguished dementia praecox and manic–depressive psychosis on the basis of symptomatology, course and outcome. He wrote that the basic disturbances in dementia praecox were the 'impoverishment of those feelings and strivings which continually stoke the furnace of our will' and 'a loss of the internal integrity of comprehension, emotion and volition'. Furthermore, his description of manic–depressive psychosis included cases of 'periodic and circular insanity, simple mania, melancholia and affective changes that could be regarded as rudiments of more severe disasters' (Berner *et al*, 1992). This formulation is what we would today consider a spectrum concept of manic–depressive illness. A test of the Kraepelinian dichotomy would thus be better served by the use of patients with affective disorders rather than bipolar I disorder (with psychotic symptoms) as the comparator group.

The non-significant differences in grey matter between patients with bipolar I disorder and healthy volunteers could be a result of sampling bias. Recruitment of patients from voluntary support groups might have resulted in inclusion of those with less-severe illness. In addition, depression, anxiety, medical disorders (e.g. hypertension, diabetes mellitus) and seizures, which can give rise to structural abnormalities on magnetic resonance imaging, were not excluded in the 'healthy volunteers'. The mean IQ and ethnicity of patient groups and the healthy volunteers were not given. These variables are important as they may contribute to differences in brain structure among groups (Thase, 2000). Similarly, the use of spoiled gradient recall echo sequence instead of inversion recovery sequence might have led to type 2 errors in comparisons of white matter volumes between patients with schizophrenia and those with bipolar I disorder (Karson & Renshaw, 2000).

The statistical analysis used the analysis of covariance (ANCOVA) model for differences between each patient group and the healthy volunteer group and differences between the two patient groups. Risk of type 1 errors would have been lower in a single ANCOVA (3×2) model.

Finally, it would be interesting to know whether 'normalisation' using the International Consortium for Brain Mapping data-set instead of the Talairach space would have made a difference to the results and whether some of the results were confirmed by the 'region of interest' methodology, which is known to be more accurate.

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Authors' reply: Drs Sharan and Bharadwaj object to our representation of Kraepelin's manic–depressive illness with DSM–IV psychotic bipolar I disorder because Kraepelin used the term to refer to a broader spectrum of affective disorders. By this logic our inclusion of patients fulfilling modern diagnostic criteria for schizophrenia rather than dementia praecox should be equally unacceptable to them. However, the Kraepelinian dichotomy continued to stimulate controversy over the past century precisely because the evolution of diagnostic criteria for these syndromes consistently failed to fully separate the disorders on clinical and neurobiological grounds. Thus 'the Kraepelinian dichotomy' has come to refer to the distinction between schizophrenia and bipolar disorder (Craddock & Owen, 2005). Furthermore, there is considerable morphometric heterogeneity between bipolar disorder and major depressive disorder (Strakowski *et al*, 2002), which underlines the need for more homogeneous rather than broader-spectrum affective disorder patient groups for magnetic resonance imaging studies.

Their hypothesis that our failure to identify grey matter abnormalities in bipolar disorder may result from recruiting patients with less-severe illness and a group of healthy volunteers with conditions associated with structural abnormalities is difficult to reconcile with our success in identifying white matter abnormalities in the same patients and typical grey matter deficits in patients with schizophrenia, who were recruited in a similar manner.

Moreover, there is no reason why healthy volunteers would have higher rates of the conditions suggested than the patient groups. Ethnicity is given in the cited associated paper (McDonald *et al*, 2004). Although type 2 errors are frequently possible, the magnetic resonance sequences used are common for computational morphometry studies and successfully detected differences in patients and healthy volunteers. The ICBM152 template was indeed used, as is standard with the SPM99 (Statistical Parametric Mapping 99) package, to create the customised template. We accept that the risk of type 1 errors would be lower with a single screening analysis of covariance but we hypothesised changes in a voxelwise comparison between each patient group and the control group and thus reported these results.

Although results from computational morphometry have been interpreted

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