Correspondence

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Symptom dimensions and the Kraepelinian dichotomy

The recent paper in which Dikeos *et al* (2006) investigate the distribution of symptom dimensions within a psychosis sample is a valuable contribution to the literature, and we fully support their observation that bipolar disorder is a much more solid construct than schizophrenia.

There are two important issues that were not discussed which we believe deserve consideration. The first is a major limitation of the current conceptual framework of psychopathology where definitions of psychopathology items are not independent of diagnostic concepts. Consider, for example, the relationship between items that measure course of illness and items that represent occurrence of reduced affective response and drive. Episodes of reduced affective response and drive with inter-episode recovery are likely to be interpreted as consistent with the presence of mood disturbance and indicative of a relatively good outcome. In contrast, chronically reduced affective response and drive which may be qualitatively identical to that in the previous example but without inter-episode recovery is likely to be interpreted as consistent with the negative features of a (schizophrenic) defect state and taken as evidence of a relatively poor outcome. In this example, recovery becomes part of the definition of two similar states. It is hardly surprising that one predicts poor outcome. We could give other examples. The only way to overcome difficulties such as these will be to use a set of clinical descriptors that do not have definitions that are enmeshed in our traditional diagnostic concepts. We believe such approaches are needed.

The second issue concerns validity. Dikeos *et al* addressed validity by considering prediction of clinical characteristics, some of which cannot be considered independent of the other items of psychopathology used to make the predictions. A key goal of diagnosis should be to identify clinical entities that are helpful for making management decisions. Recent developments in neuroscience in general, and molecular genetics in particular, offer the realistic prospects that over the coming years we will be able to identify domains of psychopathology that are associated with abnormal action in specific biological systems (Craddock *et al*, 2005). This will provide truly independent validators against which to examine the relative merits of diagnostic categories versus psychopathological dimensions (Craddock *et al*, 2006), will allow us to escape from our historical strait-jacket of traditional psychiatric thinking (Craddock & Owen, 2005; Marneros, 2006) and has the potential to lead to major benefits for our patients.

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N. Craddock, M. C. O'Donovan, M. J. Owen
Department of Psychological Medicine, School of Medicine, Cardiff University, Heath Park, Cardiff CF14 4XN, UK. Email: craddockn@cardiff.ac.uk
doi: 10.1192/bjp.190.4.361

Authors' reply: We agree that definitions of psychopathology items are not independent of diagnostic concepts and that this is a limitation of the current conceptual framework of psychopathology. It must be noted, however, that although the DSM and ICD classification systems were based largely on expert opinion, with the aim of improving reliability, and were not the outcome of rigorous nosological validity studies, they cannot be considered entirely arbitrary. Indeed, there are studies which provide support for some validity in terms of temporal stability of diagnosis and long-term outcome (Mason *et al*, 1997; Amin *et al*, 1999). In addition, the current widespread use of these two main diagnostic systems and the huge impact they have on psychiatric training make it difficult to use any set of clinical descriptors that are really free from their influence.

The second point raised by Craddock *et al* concerns the need for independent external validators of psychopathological dimensions. We agree fully with this comment. Our aim is to further the analysis of the dimensions we have identified by examining them against those validators that are currently considered the most objective, such as neuroimaging, genotypic, neuropsychological and neurophysiological data.

Like Craddock *et al*, we hope that future developments in molecular genetics and neuroscience will provide grater insight into the aetiology of psychiatric disorders. However, we would point out that one of the leading American psychiatric geneticists, Ken Kendler, has recently cautioned against an expectation that genetics will provide definitive answers to the complex and multifaceted problems currently facing psychiatric nosology (Kendler, 2006). Nevertheless, we retain our hope that the analysis of psychopathological dimensions, even if the latter are based on symptoms influenced by the current nosological categories, will help to clarify heterogeneity among patients with psychotic illnesses and facilitate our understanding of the underlying pathophysiological pathways.

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D. G. Dikeos, C. McDonald, M. Walshe, C. Sham, R. Murray Division of Psychological Medicine, Institute of Psychiatry, SGDP Building, De Crespigny Park, Denmark Hill, London SE5 8AF, UK. Email: d.dikeos@iop.kcl.ac.uk
doi: 10.1192/bjp.190.4.361a

Prion disease in Sri Lanka

Butler (2006) emphasises the importance of psychiatrists being aware of prion disease. We feel that psychiatrists in low- and middle-income countries also need to be aware of these disorders. The low prevalence rate in such countries might be attributable to underdiagnosis and underreporting. Prion diseases are not included in the list of notifiable diseases in countries such as Sri Lanka and even diagnosed cases are not notified.

Butler & Fleminger (2001) stated that approximately two-thirds of patients with new-variant Creutzfeldt–Jakob disease (CJD) present with psychiatric symptoms such as anxiety, depression, apathy and withdrawal. Somatic symptoms are a common presentation of depression in countries such as Sri Lanka. Even neurological symptoms such as pain and headache can be features of depression and the diagnosis of prion disease might be easily missed.

Two cases of prion disease have been diagnosed in the psychiatry unit at North Colombo Teaching Hospital over the past 10 years. Both patients were referred for the assessment of depression and later developed neurological symptoms such as myoclonus. Electroencephalography revealed a characteristic pattern of CJD (further details available from the authors). Other patients with CJD who presented with psychiatric symptoms have been reported from different units in Sri Lanka (Gunathilake *et al*, 1998). All these cases appear to be of the sporadic type.

Although CJD is a known cause of dementia, a patient presenting with dementia might not always be investigated for prion diseases because of the perceived low prevalence of the disease in low- and middle-income countries.

Moreover, CJD is a transmissible disease, and a lack of awareness of its true prevalence might lead to a lax attitude regarding precautions against spread. Prion protein is not destroyed by ordinary sterilisation procedures but requires sophisticated methods of sterilisation which might not be available in low- and middle-income countries. Prion diseases can also be transmitted through meat. Although there are regulations regarding meat production and sale, these are not strictly adhered to in most low- and middle-income countries, so although prion diseases might not be common in these countries, the risk of transmission might be higher. Furthermore, the healthcare systems might be unprepared to meet the challenges of an epidemic. Therefore, it is important to raise awareness of prion diseases among clinicians worldwide.

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K. A. L. A. Kuruppuarachchi, L. T. Wijeratne Department of Psychiatry, Faculty of Medicine, University of Kelaniya, Ragama, Sri Lanka. Email: lalithkuruppu@lycos.com
doi: 10.1192/bjp.190.4.362

'Major depression' in Ethiopia: validity is the problem

Mogga *et al* (2006) like the majority of published studies of people from low- and middle-income countries rely exclusively on Western measures of psychopathology (Hollifield *et al*, 2002). Culture is seen as mere packaging and is disregarded while standardised methodologies ('reliability') applied to universal psychobiological man get at the 'real' problem (Summerfield, 2004). This is a form of imperialism.

'Reliablity' cannot redeem a study that commits a category error: the assumption that because phenomena can be identified from one setting to another, they mean the same everywhere. African cultures emphatically do not share a Western ethnopsychology that defines 'emotion' as a feature of individuals rather than situations, being internal, often biological, involuntary, distinct from cognition, a cause of pathology and targetable by technical interventions (Lutz, 1985). 'Major depression' is not a timeless, free-standing, internally coherent, universally valid, pathological entity requiring medical intervention (Summerfield, 2006).

The hard truth, which if owned would totally disrupt business as usual, is that psychiatric measures are the products of a Western epistemology, including models of mind and definition of personhood. They simply cannot be turned into universally valid instruments – no matter how much tinkering with criteria and translation.

Noting the raised 'disability' scores and increased attendance at traditional healers, I do not doubt that something was ailing some of those with 'persistent depression'. However, it is likely that this was a very heterogeneous group and that undiagnosed physical illness, particularly the diseases of poverty, was a major determinant. The only solution offered was antidepressants and it is no surprise that adherence was poor.

In the last few lines Mogga *et al* state that 'more information is needed regarding the characteristics, beliefs, knowledge and illness attributes' of the population. These domains should have been the point of departure of the study, not a mere afterthought. What can emerge when researchers know so little of the lived lives of participants?

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Mogga, S., Prince, M., Alem, A., *et al* (2006) Outcome of major depression in Ethiopia. Population-based study. *British Journal of Psychiatry*, **189**, 241–246.

Summerfield, D. (2004) Cross-cultural perspectives on the medicalisation of human suffering. In *Posttraumatic Stress Disorder. Issues and Controversies* (ed. G. Rosen), pp. 233–245. John Wiley.

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D. Summerfield Institute of Psychiatry, London SE5 8AF, UK. Email: derek.summerfield@slam.nhs.uk
doi: 10.1192/bjp.190.4.362a

Authors' reply: We agree that there is inevitably a limitation in the use of measures developed in a different cultural setting. Our measure of depression, the Composite