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) deficit 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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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 greater 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.


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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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.

‘Reliability’ 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 adherance 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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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
International Diagnostic Interview (CIDI), lacks sensitivity because of the strict diagnostic rule. This could account for the low prevalence rate in our study and the fact that we may have picked up only those most seriously affected. However, we do not doubt the presence of depression in our society. The impetus for our study came from the ‘lived lives’ of Ethiopian psychiatrists working within Ethiopia who commonly encounter people presenting with symptoms according to a ‘Western’ construct of depression in a tertiary care setting. These people respond to anti-depressants by showing good recovery from symptoms and regaining their original level of functionality. In an ongoing intervention programme, we have found the same for people with depression identified by the CIDI in Butajira (study ongoing).

The CIDI was translated, back-translated and modified by experienced Ethiopian psychiatrists who considered the symptom questions to have face validity and applicability. In addition, convergent validity of CIDI-defined depression was indicated by our finding of strong associations between depression and disability. We believe that the CIDI is unlikely to be merely detecting physical ill health because first, it incorporates specific measures to screen out symptoms that seem to have a physical cause and second, our study participants with persistent depression were most disabled in social domains rather than in those domains of functioning more likely to be influenced by physical impairment (e.g. mobility).

We believe that the difference in mental health across cultures is mainly in the presenting features, not in the nature of the disorder. In low- and middle-income countries it has been said that people tend to present with somatic symptoms (Mumford et al., 1997; Parker et al., 2001). However, this view of cultural difference between the West and the rest of the world was challenged by the World Health Organization cross-cultural study in primary care (Gureje et al., 1997). Although the presentation of depression clearly does vary across cultures, in an African setting depression was found to be better characterised by core depressive symptoms than by somatic complaints (Okulate et al., 2004).


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Suicide risk and perinatal circumstances
Riordan et al (2006) present evidence that maternal circumstances and foetal experience may have an impact upon the subsequent mental health of the offspring. Many studies describe gestational insults, obstetric complications and perinatal environment as risk factors for mental illness in later life. Such evidence has often been based upon longitudinal cohort studies which have the advantage of large sample sizes and masked assessments at both exposure and outcome. Such strategies have generated evidence supporting the neurodevelopmental hypothesis of schizophrenia (Done et al., 1991; Jones et al., 1994). There can be little doubt that such epidemiological evidence can lead to hypotheses of the pathogenesis of psychiatric illness. As suggested by Riordan et al., foetal nutrition, intra-uterine stressors, hypothalamic–pituitary–adrenal axis dysfunction and attachment theory may all be putative mechanisms by which the foetal–maternal interaction contributes to future psychiatric illness.

However, there are several inherent limitations to this methodology. Longitudinal studies have traditionally concentrated on descriptions of the progeny. Data relating to many maternal factors in birth cohort studies are limited or unavailable. Important confounding factors cannot be, and have not been, eliminated in such work. Social class, alluded to by Riordan et al., cannot be ignored as an important confounder for all of the findings. Riordan et al concede that assessment of economic circumstances is based only on parental occupation. Is such a measure valid over several generations? How can social class (a factor relating both to parity, and to young maternal age and depression and suicide) not be considered an important confounding variable? The importance of housing and maternal diet, alcohol, smoking and drug use will certainly influence birth weight and depression and suicide in later life. Family history of psychiatric illness is probably the most important confounding factor that has not been, and unfortunately cannot be, assessed in this study. Maternal depression may have an impact upon birth weight as an environmental factor, but may exert a genetic effect on psychiatric illness and suicide of the offspring. Although such a study does suggest important epidemiological trends, hypothesising that biological mechanisms are involved in psychiatric illness of offspring is premature without controlling for these important confounders.


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Authors’ reply: Dr Baig may be correct in reiterating the inherent weaknesses of longitudinal birth cohort studies, but we do not accept that it is premature to hypothesise. Future studies into these potentially important epidemiological trends will require modified study designs and therefore hypotheses to guide these. We have discussed a heterogeneous group of potential confounding and mediating factors, biological influences being just one possible aspect of what is probably a complex picture of multifactorial aetiology. Hypothesising about the exclusive involvement of biological factors would indeed be premature, but not to consider them at all would place undue restrictions on future study design.

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Agoraphobia in an evolutionary context

I was interested in Bracha et al's postulation that agoraphobia might be a result of evolutionary bias towards safety-seeking among plain-living descendants. I am keen on evolutionary explanations of psychiatric phenomena, despite the fact that they cannot be proved, because they make so much sense to patients. They also allow a different perspective on distressing difficulties, even suggesting some benefit in what had previously been experienced as purely negative phenomena – for example, anxiety conferring a safety advantage in past millennia.

However, I am not so convinced by the explanation given. Many of the patients I see with agoraphobia are most phobic not about open spaces, but about places where they might be seen by others to ‘embarrass themselves’, usually by vomiting, fainting, or screaming out loud. Their overwhelming desire to return home seems to be as much about being hidden from others of the same species as about safety from predators. The explanation I favour is that signs of illness would possibly result in, at best, being excluded from the tribe, and, at worst, being killed and disposed of, both because of the risk of infecting or damaging the rest of the tribe. This is also, of course, related to past and present stigma and understanding of mental illness and unusual phenomena.

My patients have found this a very helpful, rational explanation which has helped them to start making sense that feels like irrational, uncontrollable behaviour. I will now also include Bracha et al’s explanation and see which has more face validity.


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Authors’ reply: The patients Cornish describes probably have the post-traumatic stress disorder-like secondary agoraphobia (also known as conditioned, acquired, post-traumatic, memory–trace–overconsolidation-based agoraphobia), not the rarer primary (also known as innate prepotented) agoraphobia.

As we have proposed (Bracha, 2006; Bracha et al., 2006a), both primary and secondary agoraphobia should be taken out of the panic disorder section of DSM–V/ICD–11. Primary agoraphobia (i.e. with no prior criterion A traumatic event) should be categorised with the other innate specific phobias and secondary (post-traumatic) agoraphobia should be categorised alongside (a more narrowly defined) post-traumatic stress disorder (PTSD) in a new sub-category of anxiety/stress/fear disorders entitled ‘overconsolidational fear disorders.’ Contrary to dogma, evolutionary hypotheses are testable (Bracha et al., 2005; Bracha, 2006; Bracha & Hayashi, 2006b).

Panicky attacks away from home are not the sole Criterion A event which (if untreated) are often followed by secondary (memory–trace–overconsolidation-based) agoraphobia. Secondary agoraphobia frequently follows embarrassing experiences away from home, related to psychiatric and non-psychiatric conditions such as chronic motor or vocal tic disorder, trichotillomania, narcolepsy, grand mal seizures, etc. Criterion A experiences (‘events’) such as being bullied, ridiculed, threatened or physically assaulted by school or neighbourhood peers are also often followed by the PTSD-like secondary agoraphobia.

Another diagnosis Cornish considers is social phobia (i.e. innate fear of simultaneous visual scrutiny by a large group of strangers). During much of the human era of evolutionary adaptedness, being stared at by a large group of non-smiling, non-kin conspecifics was more likely than not to be followed by negative consequences (Bracha, 2006). Evolution is not forward looking and could not anticipate a future where being stared at by a large group of non-smiling strangers might be followed by receiving an honorarium rather than by injury or death.

Most importantly, both the dimensional and categorical approaches planned for DSM–V/ICD–11 should include an evolution-inspired ‘innateness axis’ modelled on Axis V (the global assessment of functioning axis). The dimensional innate-ness axis score would reflect the clinician’s estimate (based on past psychiatric history, genetic history, age at onset, etc.) of the likely ‘hardwiredness’ of a particular patient’s symptoms (with low scores indicating a mostly post-traumatic, overconsolidational aetiology and high scores indicating a mostly evolutionarily hardwired aetiology).


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Access the most recent version at DOI: 10.1192/bjp.190.4.361

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