A history of confusion

In 1976, Robert Kendell published a seminal article in the Journal outlining the many problems (the ‘contemporary confusion’) relating to the classification of depression. Some of these, he argued, had implications for the classification of mental illness as a whole. Farmer & McGuffin revisited and updated the issues in 1983. Particularly, the institution of operational diagnoses along with the contentious issues of subdividing major depressive disorder and its characterisation on a dimensional as opposed to a categorical scale.

Subdivisions and overlaps

Kendell proposed that a bipolar/unipolar subdivision of affective disorder had a persuasive research base and further supporting
Evidence, including data from genetic studies, has firmly established the taxonomy in the later review by McGuffin & Farmer. The authors proposed a two-threshold model whereby unipolar and bipolar disorder are quantitatively rather than qualitatively different, with both disorders lying on the same continuum of liability but bipolar disorder being the more extreme or severe form. Recent genetic advances have shown this model to be overly simplistic. In particular, a twin analysis convincingly refuted the two-threshold approach, whereby the data were better explained by a model of a distinct, but partly overlapping, set of genes. The genetic correlation between mania and depression was 0.65 but approximately 71% of the genetic variance of liability for mania was not shared with depression. A separate set of twin analyses, that involved relaxing the inbuilt hierarchy of diagnosis in DSM-IV, strongly suggested that there are also partly distinct and partly overlapping genes contributing to bipolar disorder and schizophrenia. Although considered controversial at the time, this was soon followed by supporting molecular genetic evidence from linkage and association studies. Yet other twin studies suggest that there is also considerable overlap between risk factors for anxiety and depression.

The genetic overlap between depression and anxiety is hardly surprising given the high rates of comorbidity that exist between the two disorders, as findings from the National Comorbidity Survey testify. This poses important questions for the treatment of comorbidity and also future diagnostic systems. Moffitt and colleagues recently attempted to address this issue from a prospective, longitudinal approach. They reported that 72% of individuals with generalised anxiety disorder (GAD) were diagnosed with comorbid depression and 48% of individuals with depression also had GAD. Evidently there is a strong association between the disorders in this large sample, and it would be informative to ascertain how near the diagnostic thresholds those not classified as comorbid are. The authors claim that there is sufficient evidence to use the umbrella term of ‘distress disorders’ to cover both syndromes of depression and anxiety. They go on to say that the current hierarchical superiority of depression over anxiety may also be questionable as the two disorders show symmetrical developmental trajectories, so there is no evidence to prioritise one over the other. These conclusions should be of interest to those researchers charged with revising the classification systems of depression and anxiety. However, the key point from a clinical perspective is that the rates of comorbidity in depression are often underestimated, and that the treatment and management of depression is likely to vary dependent on whether the comorbidity is with anxiety or with other common psychiatric disorders.

Another pertinent issue concerning how depression is subdivided arises from the recent revival of interest in the concept of melancholia. The rationale behind this resurgence is that major depressive disorder is too heterogeneous a concept, while a melancholic subtype offers a reliable, valid and more homogenous group with specific treatment recommendations, specifically tricyclic antidepressants and electroconvulsive therapy. The editor of Acta Psychiatrica Scandinavica even felt sufficiently compelled by the concept of melancholia to devote an entire recent issue to the topic. The intentions of the authors to help homogenise depression for both clinical and research purposes are admirable. However, some of the fundamental assumptions, such as the underlying neurobiology of melancholia being well-defined and distinct from other mood disorders, is somewhat dubious, as is the evidence presented in the issue, particularly regarding the epidemiological findings. It appears as though melancholia is merely another term for severe major depressive disorder, and there seems little point in reintroducing the term as a confusing synonym for an already well-established concept.

**Categories v. dimensions**

The operational approach to defining depression is categorical in that it defines cases or non-cases of disorder. However, when originally proposed by Hempel, he accepted that the individual items of psychopathology that make up each definition were dimensional, where experience ranged in severity from mild, self-limiting and present in the general population to severe, persistent and pathological. Hempel suggested that for each criterion the rater had to ask ‘does the respondent exhibit this amount of (symptom) X?’ In terms of criteria for depression, this translates as ‘has the individual experienced at least 2 weeks of unremitting low mood?’ This imposes a severity threshold on a continuous variable: in this case, mood. Thus, recognition that symptoms of mental health are continuously distributed across individuals within the general population as well as in those with disorders has been implicit since operational definitions were first proposed. However, depression is a ubiquitous syndrome that is associated with various mental disorders (schizophrenia, substance misuse, eating disorders, anxiety) along with physical disorders such as Parkinson’s disease, myocardial infarction, stroke and obesity. Not all of these different manifestations of depression respond well to antidepressant medication, suggesting that they may be etiologically distinct. Depression also ranges in severity and symptom profile from the severe psychotic type of disorder found in bipolar illness to mild neurotic depressive symptoms associated with some personality disorders. Recent taxonomic studies exploring whether there is a discontinuity between clinical depression and subthreshold depressive symptoms have been inconclusive.

In fact, the ICD–10 classification of depression incorporates a quasi-dimensional approach, inasmuch as there are mild, moderate and severe forms which some authorities suggest have implications for treatment. For example, the UK National Institute for Health and Clinical Excellence (NICE) guidelines indicate that the preferred first line of treatment for mild depression should be psychological therapy and that drug treatment should be reserved for those with moderate or severe forms of illness.

Arguing for dimensions v. categories becomes somewhat spurious since both approaches are necessary. Certainly, there is a clinical requirement to define cases of a particular disorder, since this determines management and prognosis as well as facilitating service planning. However, defining psychopathology dimensionally probably has greater utility for basic science research especially neuropsychology and genetics. The dimensional structures of the psychopathology of unipolar depression, as well as their bipolar counterparts (L. Forty, personal communication, 2007), have already been established using multivariate statistical approaches. Thus, there is an accepted dimensional structure for depression that can provide the continuous measures required in certain spheres of research. This indicates that DSM–V and ICD–11 probably need to incorporate both categories and dimensions; not one or the other.

**Conclusions**

So what has changed over the past 30 years? Is there still confusion over the classification of depression? The answer to the first question is that the main taxonomies have not been altered drastically. An operationalised approach to diagnosis is still predominant in the main nosologies, although this may be challenged in the next revision of the DSM. The subdivision of unipolar and bipolar depression is well established by the research evidence and the melancholic definition of depression is also receiving a
The classification of depression: are we still confused?

Farmer AE, Wessely S, Castle D, McGuffin P. Methodological issues in using a dimensional approach to diagnosis, it is now evident that both have their specific applications and both are necessary to psychiatry. It seems highly likely at the time of writing that DSM–V will be cast in both dimensional and categorical formats. Are we still confused? Since it remains the case that we still have little understanding of the precise aetiology of depression, the answer to this question is surely ‘yes’. Our current nosologies remain as ‘working hypotheses’ and have no greater validity than the definitions of depression that existed when Kendell wrote in 1976. Consequently, the ‘true’ classification of depression remains as elusive as it was 30 years ago, though this is by no means reason to give up hope for the future.

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References

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