Distress and fear disorders: an alternative empirically based taxonomy of the 'mood' and 'anxiety' disorders†

LEE ANNA CLARK and DAVID WATSON

Summary The nosological organisation of DSM–IV and ICD–10 does not capture the empirical structure of the mood and anxiety disorders. Instead, they form a broad group of 'internalising' disorders with two subclasses: distress disorders and fear disorders. This empirical structure should form the basis for revised taxonomies in DSM–V and ICD–11.

Declaration of interest None.

As workgroups begin the task of revising the taxonomy of mental disorders and diagnostic criteria for DSM–V and ICD–11, the field has the opportunity to bring these classification schemes in line with current empirical research. Even if the DSM–V Task Force adopts a conservative approach, revising only those elements for which there is strong empirical support, certain sections stand to be radically revised. Only if non-scientific considerations play an important part in the revision – or lack thereof – will these sections see minor rather than major changes. We address here two such sections of DSM–IV: mood disorders and anxiety disorders.

DEVELOPMENT OF THE CURRENT TAXONOMY

With the advent of DSM–III, a strong separation was made between 'affective' and 'anxiety' disorders, with hierarchical exclusion rules virtually dictating that the former trump the latter in cases in which both types of disorder were present. Research ignoring these rules found no empirical basis for them, however, so they were eliminated in DSM–III–R. Once these exclusion rules were relaxed, research reports on diagnostic comorbidity flooded the literature. Clark & Watson (1991) and Barlow and colleagues (e.g. Barlow *et al*, 1996) offered theoretical models to explain these comorbidity findings, proposing that anxiety and depressive disorders were linked through a shared personality dimension of negative emotionality (or neuroticism; N/NE), and distinguished on the basis of unique factors – anhedonia or low positive emotionality in depression and autonomic arousal in anxiety.

During the 1990s, the US National Comorbidity Survey data revealed that major depressive disorder had very different comorbidity rates with various anxiety disorders, ranging from an odds ratio of 6 with generalised anxiety disorder to 4 with panic disorder and 3 for simple and social phobia (Kessler *et al*, 1996). Results of genetic studies paralleled the US survey data in that major depressive disorder and generalised anxiety disorder were found to share a single genetic diathesis, which also was linked strongly to the N/NE personality trait (e.g. Kendler, 1996). In contrast, the genetic overlap of major depressive disorder and other anxiety disorders was lower (Kendler *et al*, 1995) or even negligible (Pauls *et al*, 1994). Moreover, a review of the voluminous comorbidity literature by Mineka *et al* (1998) revealed that, although either type of disorder conveyed an increased risk for later development of the other, anxiety disorders were significantly more likely to appear first, and cases of pure depression were more rare than pure anxiety, raising the possibility that anxiety disorders represented a less severe form of a single spectrum.

These results led Mineka *et al* (1998) to propose an integrative hierarchical model of anxiety and depression with N/NE as a common genetic substrate, and various specific factors differentiating individual disorders. Specifically, anhedonia/low positive emotionality is conceptualised as the specific factor in depression, whereas autonomic arousal represents the specific component in panic disorder (not anxiety disorders in general, as in the original model of Clark & Watson, 1991). Other anxiety disorders such as phobias or obsessive–compulsive disorder also are presumed to have their own (currently undetermined) specific factors. Fergusson *et al* (2006, this issue), using structural equation modelling on data from a 25-year longitudinal birth cohort study, found evidence consistent with this model. Specifically, he demonstrated that a common factor ('internalising,' on which we expand subsequently) explained both symptom comorbidities and continuity over time for major depressive disorder, generalised anxiety disorder, phobias and panic disorder; at the same time, however, he found across-time continuity in disorder-specific components of major depressive disorder and phobias. Although this model explains many aspects of the data well, the exact nature of the additional specific factors (e.g. whether they are only phenotypic or also have a genetic basis) remains unclear.

RECENT ADVANCES IN UNDERSTANDING THE STRUCTURE OF PSYCHOPATHOLOGY

An important related question is how the genetic and structural findings for anxiety and depression fit into the broader domain of psychopathology. The answer to this question has emerged over the past decade. During this period, six large-sample independent studies (Lahey *et al*, 2004; see Clark, 2005 for the five others) have examined the structure of psychopathology by studying diagnostic comorbidity patterns phenotypically and/or genotypically, each using a set of common mental disorders that largely overlapped across studies. The results have revealed a remarkably consistent structure: a hierarchical model with two broad factors – externalising and internalising. Substance dependence, attention-deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder/antisocial personality disorder define the externalising factor. The internalising factor subsumes two highly related subfactors: 'distress/ misery' – comprising generalised anxiety disorder, over-anxious disorder and depressive disorders

†See pp. 540–546, this issue.

– and 'fear', which includes simple and social phobias, separation anxiety disorder and panic disorder. Slade & Watson (2006) additionally showed that this structure fitted both DSM–IV and ICD–10 conceptualisations of these disorders, with neurasthenia representing a manifestation of distress/misery in the latter. Finally, it is noteworthy that this alternative hierarchical scheme consistently captures the comorbidity data better than the DSM model, which separates these syndromes into 'mood' and 'anxiety' disorders.

The recognition of this structure has engendered further questions about the nature of the internalising and externalising dimensions themselves. Based on an extensive review, Clark (2005) proposed that both personality (e.g. N/NE) and psychopathology derive from innate general temperament dimensions, including negative and positive temperament, which differentiate through development into the full range of adult personality and also are the diatheses from which psychopathology develops in response to a sufficiently stressful environment. In this model, internalising emerges largely from negative temperament and externalising from temperamental disinhibition, alone or in combination with negative temperament.

IMPLICATIONS FOR DSM–V/ICD–11

Moreover, this robust structure has two important implications for DSM–V and ICD–11. First, the hard separation between mood disorders and anxiety disorders introduced in DSM–III, with particular diagnoses assigned to each group, is shown to be a pseudo-hierarchical, rational folk system. It now is abundantly clear that these two types of disorders are strongly related and should not be artificially separated into different diagnostic classes. Moreover, the current distinction between mood disturbance (the defining element of the current mood disorders) and anxiety/avoidance (the characteristic features of the current anxiety disorders) is unsound and does not provide an optimal arrangement of these disorders (Watson, 2005). To the extent that the DSM and ICD purport to be empirical documents, the current folk taxonomy must be abandoned and replaced with a data-driven, scientifically supported taxonomy. Second, mental disorders are hierarchically arranged: that is, the evidence establishes that most disorders co-occur and are empirically related, but that some disorders are more highly comorbid than others. The taxonomic structures of official diagnostic manuals need to reflect this fact.

What this would mean for DSM–V/ICD–11, for example, is that instead of grouping generalised anxiety disorder, panic disorder, and so on together under the heading of 'anxiety disorders', as they are now in DSM–IV, generalised anxiety disorder and overanxious disorder would be grouped with major depressive disorder/dysthymia (in what Watson, 2005, labels the 'distress disorders') because they share more variance with these depressive disorders than with other anxiety disorders. One clear advantage of such a hierarchical structure is that it reconciles the long-standing tension between 'lumpers' (who value broad diagnostic categories) and 'splitters' (who argue for fine-grained diagnostic specificity) by encompassing both at different levels of the diagnostic hierarchy. Thus, depending on the nature of the problem at hand, clinicians and researchers can choose to focus on a few broad non-specific classes of psychopathology (e.g. distress disorders, externalising disorders), individual disorders, or some combination of the two. Note also that a hierarchical model easily can be extended further to encompass subtypes within current disorders (e.g. subtypes of specific phobia; see Watson, 2005).

The primary immediate change would be organisational, with more highly comorbid disorders placed together and those with less overlap falling farther apart in the hierarchical structure. However, although none of the current diagnoses necessarily would disappear if the empirically revealed structure were implemented in DSM–V/ ICD–11, it is likely that moving to a more thoroughly empirically based taxonomy eventually would result in more radical diagnostic revisions. In particular, data-based considerations eventually would create pressure to replace currently heterogeneous syndromes (such as many of the current personality disorders) with more homogeneous diagnostic groups, or at least ones in which observed heterogeneity reflected more peripheral variation with little implication for differential treatment. For example, when relations between various personality and psychosocial variables and treatment outcome were examined in a sample of patients with recurrent major depression, it was the common, overlapping variance that carried the predictive weight (Clark *et al*, 2003).

There are likely to be pressures from various constituencies to maintain the status quo, but their bases will be pragmatic rather than scientific. For example, directors of anxiety disorders clinics may resist revision for fear that the loss of generalised anxiety disorder to the distress disorders will reduce their client base. Pharmaceutical companies may express concerns that extensive (translation: expensive) clinical trials will need to be conducted to examine the effectiveness of their current 'antidepressant' drugs for generalised anxiety disorder. Even further, the fact that the distress and fear disorders are themselves collapsed together at a higher level in the hierarchy has implications for the cross-effectiveness of 'antidepressant' and 'anti-anxiety' drugs. Of course, practising clinicians have known for years that there is no clear one-to-one correspondence between the formal DSM diagnoses they give their patients and the prescriptions they write for them, and the pervasive phenomenon of 'comorbidity' is well known to those who are on the front lines of mental-disorder treatments. Thus, these pragmatic concerns should not hinder the development of an empirically adequate and clinically useful psychiatric classification scheme.

LEE ANNA CLARK, PhD, DAVID WATSON, PhD, Department of Psychology, University of Iowa, Iowa City, USA

Correspondence: Lee Anna Clark, E11 SSH, Department of Psychology, University of Iowa, Iowa City, IA 52242-1407, USA. Email: la-clark@uiowa.edu

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