

## REVIEW ARTICLE

# The Boundaries of Depressive Illness

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### Abstract

**Background:** There is increasing recognition of the overlap between depression and a variety of psychiatric disorders. This has important implications for nosology, treatment, service utilisation and prevention of depression. **Aims:** To study the boundaries of depression with other psychiatric disorders. **Methods:** This selective review, a mixture of classic and recent papers, focuses on the boundaries between depression and normality, subthreshold disorders and variety of psychiatric disorders. **Results:** The review indicates that there is considerable overlap at the boundaries of depression. Current data suggests strong relationships with anxiety disorders, personality disorders and bipolar disorder. There is no evidence to support the 'drawn boundary' with subthreshold depressive disorders. The relationship with grief and schizoaffective disorders is inconclusive.

**Conclusions:** The disputes at the boundaries of depression highlight the limitations of the categorical system of classification. These disputes result from implementation of diagnostic criteria, the fundamental nosologic process and phenomenon themselves. There is thus a further need to explore alternatives in defining and understanding psychiatric diagnosis (German J Psychiatry 2007;10: 79-87).

**Keywords:** depression, classification, Dysthymia, bipolar, review

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## Introduction

The lack of distinct boundaries between depression & the major classes of psychiatric conditions have been recognised for more than a century. Clinical experience suggests that symptoms of depression overlap substantially with those of other psychiatric disorders. The advent of structured instruments and standardised diagnostic criteria that delineate discrete syndromes has substantiated this observation (Wittchen, 1989).

The overlap between depression and various psychiatric disorders have important implications beyond nosology. Depression including their different clinical manifestations is particularly important as they are amongst the most frequent psychiatric illnesses both in the community and in a variety

of clinical settings. In addition to their frequent and serious complications that include suicides and substance abuse, they are strongly associated with limitations in well-being and daily functioning that are equal to or greater than those of severe chronic medical conditions. According to the World Development Report 1993, depression ranks fifth among women and seventh among men as a cause of morbidity (World Bank, 1993). Understanding the boundaries appear to be particularly important as they influence treatment, service utilisation, and help understand aetiology of psychiatric disorders (Wittchen, 1989).

This selective review, a mixture of classic and current papers, attempts to integrate the current understanding of the phenomenology, importance and controversies concerning the boundaries between depression and psychiatric disorders. The strengths and limitations of the current classificatory systems are discussed in this context.

## Sadness/Grief

The normal emotions of sadness are part of everyday life and should be differentiated from major depressive disorder. Sadness is a universal human response to loss, defeat, disappointment, or other adversities. The response may be adaptive or might signal the need for support from significant others. Transient depressive periods also occur as reaction to certain anniversaries (anniversary reaction), as well during the premenstrual phase (premenstrual tension) and the first week postpartum (maternity blues).

Normal bereavement or grief occurs in response to significant separations and losses, such as death, divorce or catastrophes. In addition to depressed affect that is appropriate to the loss, bereavement reactions are characterized by the prominence of sympathetic arousal and restlessness believed to represent, from an evolutionary perspective, physiological and behavioural mechanisms to facilitate the search for the lost object. In most cases grief and sadness do not generally seem to cause depression except in those predisposed to mood disorder. However, increasing research into catastrophes like the civil war in Lebanon (Karam, 1994) & the earthquake in Armenia (Armenian, 2002), have shown the boundary with clinical depression are blurred and they serve as potent forces in depression formation.

While the distinction of sadness with depression is mostly straightforward it is sometimes difficult. It is dependent on the severity, duration and the required number of symptoms as defined by the different classificatory systems, often producing ambiguities and varying prevalence.

## Subthreshold Depressive Disorders

Classification of subthreshold depression has received little attention and has been mainly defined as residual categories. This approach has often been based on expert opinion rather than on empirical testing and makes an assumption of thresholds for caseness (Liebowitz, 1993).

The boundary of depressive disorder with dysthymia is a true continuum. The core concept of dysthymia refers to a subaffective disorder with an insidious onset and low-grade chronicity for at least two years, which follow an intermittent or persistent course with origins in childhood or adolescence (DSM-IV). The profile of dysthymic disorder overlaps with that of major depressive disorder but differs from it in that symptoms tend to outnumber signs. Prospective studies on children have revealed that about 75% of dysthymics tend to develop major depression in their lifetime (Klein, 2006). The rate of major depression in the relatives of early-onset dysthymic probands is significantly greater than in the relatives of normal controls (Klein, 2006). Studies also indicate that the rate of dysthymia is significantly greater in the relatives of dysthymic probands than in relatives of both major depressive probands and normal controls (Klein 2004, Klein 2006).

Sleep EEG data indicate that many persons with dysthymia exhibit sleep patterns of those with acute depression (Kocsis, 1987). Dysthymia responds to treatment with antidepressant with studies indicating a 70% remission rate (Kocsis, 1987). There are only a few differences in clinical features, family history, and treatment response between patients with dysthymia and major depressive disorder.

Another subthreshold depressive disorder is the residual diagnostic category of mixed anxiety-depressive disorder (MADD) in ICD-10 (included in DSM-IV appendix), which is used to characterize a sizeable proportion of patients with substantial levels of anxiety and depression, not meeting the diagnostic thresholds for formal anxiety or depression. Persons diagnosed with MADD have both anxious and depressive symptoms that warrant clinical intervention (Tyrer, 2003). The field trial for MADD for DSM-IV showed that patients presenting with subthreshold affective symptoms to be at least as common as patients with DSM-III-R anxiety and depressive disorders (Zinbarg, 1998).

A twin study (Kendler, 1998) evaluating the nature of boundaries between depression and milder cases, found no evidence for the DSM-IV 'stated boundary' of 2 weeks duration, five symptoms or clinically significant impairment and suggested that the criteria for major depression as articulated by DSM-IV appears to be a diagnostic convention artificially imposed on a continuum of depressive symptoms. Individuals with subthreshold depression show substantial disability and poor social functioning (Wells 1989, Spitzer 1995). It accounts for similar, or even greater, overall impairment, service utilization and psychotropic drug use in the population compared to formal affective disorders, due to its higher prevalence (Broadhead, 1990).

Taken together, these data call into question the validity of the distinctions between the various forms of depression and suggest that depression is better conceptualized as a single condition with varying levels of symptom severity.

## The Unipolar-Bipolar Distinction

The Unipolar-Bipolar distinction has proved to be of great heuristic value for clinical and therapeutic research central to the understanding of mood disorders. The distinction is validated to an extent by each of the classic criteria (Robins, 1970) but in most cases there is a substantial overlap making them equally compatible with continuum models.

The phenomenology at the boundary between unipolar and bipolar disorder is the sharing of symptoms (ICD-10). Unipolar depressive disorder remains a provisional diagnosis especially during the period that represents the broad range of susceptibility to mania because of the possibility of developing a later manic episode.

Unipolar disorder is more prevalent clinically and probably etiologically more heterogeneous than bipolar disorder. Epidemiological studies show consistent differences between unipolar and bipolar disorder in sex ratio, age at onset, and frequency of affective episodes (Goodwin, 1990). Depressive

episodes in bipolar disorder appear to be more severe, psychotic and to have the so-called reverse neurovegetative signs (Akiskal 1983, Coryell, 1987). Follow-up data suggest that between 5% and 20% of unipolar patients switch to a bipolar course. Data have shown that unipolar individuals who switch at follow-up are younger, more likely to be males, have more severe episodes, an earlier age at onset and more prior episodes (Tsuang 1981, Akiskal 1983) with the 'switch' developing within five years in two-thirds (Akiskal, 1983). In addition, the unexpected crossing from dysthymic disorder to hypomanic or manic episodes have been described suggesting some forms of dysthymic disorder are subaffective precursors of bipolar disorder (Akiskal, 1995).

There is clearer evidence for genetic transmission in bipolar disorder. Most studies of the relatives of bipolar probands have shown a two to threefold increase in the prevalence of unipolar disorder in comparison to unipolar probands who have shown similar prevalence as control probands for bipolar disorders (Rice, 1987). Though these findings have been replicated in numerous other studies, the genetic evidence is not straightforward. Tsuang and colleagues (1980) found an increase of bipolar relatives in the families of unipolar probands. Akiskal's prospective study (1983) of unipolar depressives suggested that probands having three or more relatives affected were at higher risk of developing bipolarity. Conversely, the increase in unipolar disorder in bipolar probands tends to diminish if more stringent criteria are used, for example if three or more episodes of depression are required for a diagnosis of unipolar depression. Also new research comparing monozygotic and dizygotic twins has demonstrated that the genetic propensity to mood disorders embraces entities that extend beyond the classical endogenous depression to subsume a larger variety of depressions (Akiskal, 1996).

There is some evidence that subjects with unipolar and bipolar disorder differs on neurotransmitter metabolites and neuroendocrine response but much of this evidence lack robustness and replicability (Goodwin, 1990). With regards to treatment, bipolar disorders have a better response to mood stabilisers. Mood stabilisers especially lithium have a role as adjuvant in treatment of unipolar depression (Goodwin, 1990). Antidepressants, which are primary modalities of treatment in unipolar depression, tend to produce manic or hypomanic episodes in bipolar depression (Kupfer, 1988).

It appears that many of the subtypes of affective disorders are not pure entities and that unipolar-bipolar distinction fails to capture the heterogeneity that exists among mood disorders. Thus the evidence varies, as do interpretations, and the data do not stand the stringent test to decide between continuum and separate disorder models.

## **Schizodepression – Bridge between schizophrenia & depression?**

Clinicians have no difficulty in distinguishing the phenomenology of classic schizophrenia from depression and the distinction appears stable over time. Delusions & hallucinations in the psychotic subtype of depression are generally

mood congruent. But about 20% of psychotic depressives develop mood incongruent psychotic features including first ranking symptoms (Goodwin, 1990). Most schizophrenics experience some depressive symptoms during the course of their lifetime. About one-third to one-fourth of patients with schizophrenia develops major depression (Siris, 2000). The significance of depression in schizophrenia has been a matter of some debate. It has been proposed by different experts that it should be regarded as the prodrome, a post-illness reaction, a secondary post-antipsychotic medication reaction, or demoralization in the setting of severe mental illness. Studies comparing brain region volumes found enlarged ventricles in schizophrenia and psychotic depression but not in non-psychotic depression. Psychotic depression was also comparable to schizophrenia in hippocampal and temporal lobe asymmetry (Salokangas, 2002). Depression during the acute phase of schizophrenia may be associated with a favourable course and outcome (Vaillant, 1964), but several studies have suggested that depression during the chronic phase of schizophrenia has often been associated with worse outcome, impaired functioning, personal suffering, higher rates of relapse and suicide (Vaillant 1964, Heila 1997).

The overlap between schizophrenia and depression have long been conceptualised around the concept of schizoaffective disorders. Several researchers have proposed that schizoaffective disorder represents a bridge between affective disorders and schizophrenia (Kendler, 1986).

Schizodepressive patients experience a phenomenological overlap with prominence of both schizophrenic and depressive symptoms in the same episode of illness (ICD-10). Family studies support the notion that schizoaffective patients are a heterogeneous group with several different genetic dispositions (Kendler 1986). A number of studies have found that relatives of patients with schizodepressive disorder have greater risk of developing both unipolar depression and schizophrenia (Kendler 1986, Rice 1987). Schizodepressive patients have an outcome between patients with major depressive and schizophrenia with patients with mood-incongruent features faring worse than patients with mood-congruent features. A five year follow-up also found that schizoaffective patients who were categorised as chronic had a worse outcome, while the non-chronic schizoaffectives had a similar outcome to psychotic major depressive patients (Coryell, 1984). Abnormalities in platelet 5-HT intake, CSF tryptophan, plasma tryptophan and 5HIAA, clonidine induced increase in growth hormone, non-suppression of DST have been reported both in schizoaffective and affective disorders. Other findings like CSF noradrenaline, platelet serotonin content and PGE-1 stimulated adenylate cyclase activity in schizodepressives show similar values as in schizophrenics (Meltzer, 1986). Studies of response to treatment suggest that antidepressants which are the primary modalities for treatment in depression when used alone are of little benefit in schizoaffective disorder – depressive type which responds to a combination of antidepressant and antipsychotic (Levitt, 1988).

Summarizing the above evidence is fraught with methodological issues owing to the significant conceptual and definitional shifts that schizoaffective disorder has undergone (Lapensee, 1992). There is still little support for schizoaffective

tive disorders as a separate entity with many researchers considering it as a buffer zone between schizophrenia and affective disorders (Blacker, 1992). Given the paucity of specific data, investigators have been unable to reach a consensus on either the status of schizodepressive disorder or its boundary with depression.

## The Anxiety-Depression Picture

The psychopathological differentiation of anxiety and depressive states has not been entirely resolved. Cognitive factors like hypervigilance, severe tension, perceived danger, phobic avoidance, doubt and uncertainty seem to characterise anxiety in comparison to sadness, perceived loss, hopelessness, self-deprecation that characterise depression (ICD-10).

Multiple international studies (Sartorius 1983, Lepine 1993), investigating common psychological problems in epidemiological samples have found about a quarter of primary care attenders' worldwide have an ICD-10 diagnosis of 'current depressive episode' with the most frequent overlap being anxiety. The NIMH epidemiological catchment area study also showed that an anxiety-depression syndrome characterized a substantial proportion of depressed community respondents (Kessler, 1993). The analysis of lifetime DSM-III-R diagnoses in the NCS data showed that 58% of individuals with major depression also met criteria for a comorbid anxiety disorder (Kendler, 1996). Conversely, most individuals with diagnosed anxiety disorders also meet criteria for depression although comorbidity varies widely across different anxiety disorders (Mineka, 1998). Finally, both mood and anxiety disorders are highly comorbid with substance use disorders, eating disorders, somatoform disorders and personality disorders (Mineka, 1998). In spite of the data, anxiety symptoms are not included in the main diagnostic criteria for depression both in ICD-10 & DSM-IV. Also, the hierarchical exclusion rules of DSM-IV and ICD-10, makes it difficult to diagnose anxiety disorders in the presence of significant depression.

Of the various anxiety disorders, generalized anxiety disorder (GAD) poses a more significant boundary issue than other anxiety disorders. GAD is very strongly linked to the unipolar mood disorders, both phenotypically and genotypically. Analyses of twin data have found that major depression and GAD are genetically indistinguishable; i.e. The genetic correlation between them essentially is unity, indicating that they reflect a single common genetic diathesis (Kendler 1996, Mineka 1998). At the phenotypical level examination of NCS data showed a lifetime diagnosis of GAD had tetrachoric correlations of .64 and .59 with dysthymia and depression (Krueger, 1999). Confirmatory factor analysis to estimate the latent correlations among major depression and various anxiety disorders showed GAD correlated more strongly with depression ( $r=0.63$ ) than with OCD ( $r=0.52$ ), panic/agarophobia ( $r=0.5$ ) or social phobia ( $r=0.37$ ) (Brown, 1992).

Literature suggests that depression with comorbid anxiety disorder is associated with poorer outcome. Depressed outpatients with comorbid anxiety disorders compared to depressed outpatients without generalized anxiety disorder, had a younger age of onset, higher level of suicidal ideation, poorer social functioning and a higher morbid risk for illness onset in first-degree relatives (Brown, 1992; Sherbourne, 1997).

Benzodiazepines, which are highly effective in treatment of all anxiety disorders, have shown to be of little benefit in depression (Brown, 1992). Alprazolam, a high potency benzodiazepine, is an exception, with evidence showing that it has some antidepressant properties (Haefely, 1985). Antidepressants drugs have been shown to be effective in the treatment of anxiety disorders (Gorman, 1987).

Thus, research into the relationship between anxiety and depression provided evidence for the absence of clear boundaries both at symptom and at syndrome levels.

## Personality Disorders and Depression

Personality functioning and depressive illness are complexly interrelated. Research on the prevalence of personality disorders among depressives generally suggests a 30-70% comorbidity rate. Zimmerman and Coryell (1989) investigating the relationship between Axis I and II disorders found that of those who had a diagnosis of personality disorder, 38.5% had a history of major depression and 14% had a history of dysthymia. They further reported that among those who were diagnosed of having a history of major depression or dysthymia, 47.4% and 47.6% had a concurrent Axis II disorder (Pfohl 1984, Shea 1987).

The available data suggest that those depressives with a personality disorder when compared to those without have an earlier onset for first depressive episode (Pfohl, 1984) and a significantly longer duration of current depressive episode (Shea, 1987). Anorexia, guilt and features of somatisation disorder have been observed more frequently among depressive spectrum patients and anxiety, paranoia, and psychoticism as assessed by the HSCL occur more frequently among depressives with any personality disorder (Shea, 1987). Mixed findings have been obtained on whether having a personality disorder increases the severity of the depressive episodes (Charney 1981, Shea 1987). Suicidal ideation, serious and non-serious suicide attempts have been observed to be more among depressive outpatients with a concurrent Cluster B (Dramatic-erratic) personality disorder (Charney 1981, Pfohl, 1984).

Most of the available family data is with regards to the relationship of depression with borderline personality disorder. Some studies show an increase in depression clustered in relatives of borderline patients who have depression (Pope, 1983) while others have shown higher rates of bipolar disorder (Akiskal, 1985). Rates of borderline personality disorder are not reported in most family studies of affective disorders

but Coryell and Zimmerman (1989) did not report increase in borderline or other cluster B personality disorders in relatives of patients with major depression. The family data is thus unclear.

There is an indication that those depressives with a personality disorder generally respond less favourable to treatments for depression than do other depressives. Tricyclics an important modality of treatment for depression has shown no benefit in 'pure' borderline personality disorder (Zimmerman, 1989). Studies (Charney 1981, Pfohl 1984) assessing antidepressant response found that there was a response rate of 76% in pure depressives in comparison to 36% in depressives with comorbid personality disorder. But this finding has not been universally replicated and negative findings have also been reported (Zimmerman, 1989). There are anecdotal reports of borderline patients improving with carbamazepine or lithium (Blacker, 1992).

This area of research is fraught with methodological problems. The boundaries of personality disorders themselves are not very clear. Individuals often have features of more than one personality disorders and this makes the differential diagnosis with affective disorder more difficult. The majority of research of personality assessments in patients with depression has been based on self-reports and is uncontrolled for the significant effects that depression and current difficult life circumstances may have on personality measures (Farmer, 1990).

DSM-IV has included mood disorders on Axis I and personality disorders on Axis II, implicitly suggesting that there exists a clear boundary between them. The present evidence suggests that the constructs of personality and mood overlap and the distinction between personality and depression though clinically useful is not an absolute trait-state distinction (Widiger, 1993). There is a need thus for prospective studies assessing personality prior to the onset of depression, family & outcome studies to understand this relationship better.

## Depression and somatisation – cultural equivalents?

The phenomenon of depression is recognisable in any culture in which it has been sought, although its clinical presentation may vary widely. It has long been suggested that somatisation was the cultural equivalent of depression in non-western cultures. This is reflected in the results from the Cross-National epidemiology study of major depression & bipolar disorder (Weissman, 1996), which showed marked differences in the prevalence of depression at different centres, even after correction for between-centre differences. Leff (1977) pointed out that people from traditional cultures tend to express distress in somatic terms or express dysphoria in ways different from western cultures. In China four-fifth of psychiatric outpatients were diagnosed as neurasthenic, of which half of them was self diagnosed. The Chinese concept of neurasthenia includes somatic, cognitive, emotional and depressive symptoms. Significantly, Chinese

people have lower rates of depression (Zhang, 1998). Studies from the Indian subcontinent have shown similar patterns. Pain was the most common physical symptom in depressives. This represents in Asian culture 'suffering' and 'dependency' while disguising the affective aspects of common mental disorder (Bhugra 1997a). Different levels of awareness, popular perception of illness, role of doctor, different pathways of care and culture could possibly explain the varying presentations. Another issue that could contribute to cross-cultural variations are methodological problems. Ballenger (2001) suggest that variation noted may be the consequence of differences in population sampling and methods of clinical assessment, differences in classification and lack of culturally appropriate instrument or problems related to validation.

Researchers seem to agree that each culture has its own emotional lexicon that encodes socially and morally significant values and its own idioms of distress – cultural ways of talking about distress (Bhugra, 2004). These idioms have not been included in either ICD-10 or DSM-IV TR. Even while the current diagnostic systems are necessary for comparisons between different cultures, defining concepts of depression pluralistically in accord with both psychiatric and indigenous belief systems will help to better our understanding of the heterogeneous expression of depression across cultures. (Bhui, 1999).

## Organic Depression

Organic depressive disorder appears to affect men and women equally, in contrast to major depressive disorder, which predominates in women. Depression accompanies a range of medical problems. Many endocrine, pharmacological and structural factors lead to organic depressive disorder (Caseem, 1990). This issue has additional importance, as there is potential impact in improving our understanding of disease process and aetiology. When depressive symptoms occur in the setting of physical illness, it is not always easy to determine whether they constitute a genuine depressive disorder especially in the medically ill elderly. Pain, discomfort, anorexia & weight loss, demoralisation and medication adverse effects in the medically ill should not be mistaken for the somatic symptoms of depression, while anhedonia, psychomotor disturbances, convictions of failure, worthlessness or guilt & suicidal ideation should arouse the suspicion of clinical depression (Lishman, 1998).

There are important questions yet to be answered concerning the link between organic disorders and depression. Is the mood disturbance a physiological or psychological reaction to the medical condition? Are the two conditions concurrent sequelae of another factor? Is the mood syndrome responsible for exacerbation of the symptoms associated with the medical condition, as might be seen in depressed individual with fatigue and somatic complaints? Though the area of research is very broad, currently the data is mostly limited to case-reports and case series. Further research focussed on these areas would probably help to understand the relationship better.

## Discussion

In the last century, the discovery of biological markers for diseases like syphilis, multiple sclerosis and connective tissue disorders, all of which had 'myriad clinical presentations' have clarified diagnostic and nosological issues leading to better understanding of pathophysiology and improved management of these conditions. It was hoped, at the beginning of the last century, that similar discoveries would shed light on disagreements about diagnosis and nosology in psychiatry. In the absence of agreements on biological mechanisms or markers, controversies continue to plague diagnostic systems. This is reflected in the significant ambiguities in the boundaries of the diagnosis of depression. The boundaries of depression with many of the disorders appear to represent a true continuum or overlapping symptoms and are not consistent with the conceptualisation of depression as a discrete diagnostic category. This overlap is not unique to depression and is reflected by the high degree of comorbidity among psychiatric disorders in community studies. The NCS data showed that 56% of people with psychiatric diagnoses had comorbidity (Kessler, 1993) while Rush (2005) found that among depressives 20% had three or more comorbid disorders. This may be partly may be an artefact of drawing an artificial line between two aspect of illness with a common aetiology like depression and personality disorders or depression & generalized anxiety disorder.

The major classificatory systems are categorical rather than dimensional, and binary rather than probabilistic (ICD10 & DSM-IV). When Robins & Guze (1970) described their classic criteria for validity of psychiatric disorders they implicitly assumed that psychiatric disorders are discrete entities. They did not consider that disorders might merge into one another with no natural boundary in between. Several attempts to demonstrate natural boundaries between related syndromes or between a common syndrome such as major depression and normality either by locating a zone of rarity between them or by demonstrating a nonlinear relationship between symptom profiles and validating variables such as outcome or heritability have been failures (Kendell, 1970; Kendler, 1998). This lack of evidence of the validity of the diagnostic categories and their boundaries continues to be a critical issue.

A dimensional classification on the other hand could provide a more valid and internally consistent means to describe a patient's psychopathology. It could provide a means for recognising assessing and tracking subthreshold conditions and might facilitate the development of more precise, consistent, and uniform points of demarcation between normal and abnormal psychological functioning (Kessler, 2002). In addition a dimensional classification could also provide a more specific and individualized profile of patient's psychopathology with different cut-off points along distribution of dimensions of functioning that could be more meaningful and specific to different social and clinical decisions.

In spite of the inherent advantages of the dimensional system little has emerged with regard to exactly how dimensional classification could be introduced in the DSM and ICD. The

dimensional classification was rejected in the preparation of DSM-IV in part because "there is yet no agreement on the choice of the optimal dimensions to be used for classification purposes" (American Psychiatric Association, 1994, p. xxii). There have been suggestions that dimensions corresponding to broader biologically and environmentally based constructs of temperament and personality like neuroticism and externalisation must drive the classificatory systems (Clark, 2005). This is a more challenging endeavour because broader behavioural phenotypes not currently recognized by the DSM must be identified, validated, and measured in a manner that is feasible in clinical practise and research.

Binary systems in which the patient is judged to have either the disorder or not, has clinical utility but fails to take into account the multiple uncertainties involved in psychiatric diagnoses. Psychiatric diagnoses involve assessment of symptoms that is dependent on patient's description and also clinician's interpretation of that description. The diagnostic values of the symptoms are also currently unknown (Ohayon, 1990). Many attempts have been made to incorporate this uncertainty into diagnostic measures. It has long been argued that diagnosis should be represented as a vector of probabilities where each component of the vector represents the probability that an individual has the disorder (Dunsmore, 1966). Such probability scales have been proposed as weighting schemes for instance weights based on the probability of underlying bipolar disorder among unipolar relatives in a linkage study (Baron, 1990) and latent class analysis for depression (Wainwright, 1997). This could prove to useful in research settings but currently its utility in clinical settings is limited.

The current diagnostic categories, inspite of their questionable validity have high clinical utility. They remain a useful framework for organising and explaining the complexity of clinical experience. They have helped to improve communication, standardize research, treatment and organise services. Given the advantages of each of the systems the choice of one or other approach can be defined by specific purpose – categorical approaches could be preferable in clinical decision making for an individual patient; dimensional approaches could be applied to research settings. Another practical alternative imbibing the two systems would be to introduce dimensional severity rating to the extant diagnostic categories and/or the constituent symptom criteria (Brown, 2005).

It is distinctly possible that all of the systems to conceptualise psychiatric disorders are not mutually exclusive and may describe different segments of psychiatric morbidity. Discrete disease entities and dimensions of continuous variation are not mutually exclusive means of conceptualising psychiatric disorders. Both are compatible with a threshold model of disease and may account for different or even overlapping segments of psychiatric morbidity. Psychometric and statistical methodology demonstrates that categorical and dimensional approaches are fundamentally equivalent and mutually convertible (Kramer, 2004). Every dimensional diagnosis can be converted to a categorical diagnosis by setting an appropriate cut-off point, and creating an ordered set of categorical diagnoses along a dimension.

In spite of the importance, there has been little focus on determining the natural boundaries within the territories of existing diagnostic categories including depression. There is need for research that requires multidisciplinary approaches including genetic and psychobiological, to address the problem areas resulting from the diagnostic criteria, the implementation of the criteria, the fundamental nosologic process and the phenomena themselves. Till that point a classificatory system that suits everyone's different goals and needs may remain distant and hence it is important to be aware of the limitations and strengths of the current classificatory systems.

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