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Normal Versus Pathological Mood: Implications for Diagnosis

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Abstract

Is there a clear line between normal and abnormal mood? Studies of manifest and latent structure provide strong support for a continuum that extends from asymptomatic to subsyndromal to syndromal cases of increasing severity. Subsyndromal symptoms are impairing, predict syndrome onset and relapse, and account for more doctor's visits and suicide attempts than the full syndromes, yet they are not recognized in the current classification. For most research and some clinical activities, dimensional diagnoses are recommended, and examples are offered for how such diagnoses could be made. For clinical activities requiring decisions, a multithreshold model is proposed in which both lower (e.g., mild depression, capturing subsyndromal cases) and upper (e.g., major depression, capturing clinically significant cases) diagnostic categories are used to inform clinical care. Beyond its implications for diagnosis, the dimensionality of depression and anxiety has implications for etiology and for research aimed at understanding how emotions become disrupted in psychopathology.

Contents

INTRODUCTION

Sadness, fear, and anxiety are universal emotions that are thought to serve important functions for individuals and for our species. At the same time, these emotions form the basis of depressive and anxiety disorders that are among the most prevalent, disabling, and costly diseases worldwide (Kessler et al. 2007, World Health Organ. 2017). Where to draw the line between normal and abnormal emotional experiences—and, indeed, whether a line exists at all—is a source of considerable debate. This is a decision with high stakes, influencing who is identified as having a disorder, which behaviors are considered to require treatment, how disorders are measured and studied, and who has access to care.

Given these stakes, it behooves us to reflect on what has been learned so far about the boundaries of depressive and anxiety disorders and to consider whether current diagnoses of these disorders align with the available evidence. To that end, this article reviews the state of the literature on normal and pathological mood. Although emotional disturbance can take many forms (Berenbaum et al. 2003), most studies to date have focused on depressive and anxiety disorders, and I focus my review on those conditions. Based on the conclusions of this review, I offer recommendations for improving the diagnoses used by researchers and clinicians. I close by proposing a new multithreshold model of diagnosis that is empirically informed while meeting the practical needs of decision makers in clinical settings.

WHAT HAVE WE LEARNED ABOUT THE BOUNDARY BETWEEN NORMAL AND PATHOLOGICAL MOOD?

Diagnosed Individuals Differ from Controls (but the Magnitude of the Difference Depends on How Controls Are Defined)

The typical study in this research area uses an extreme groups design in which a clinical group of depressed (or anxious) individuals is compared with a control group in which levels of depression (or anxiety) are low or absent. Studies published in top psychopathology journals generally use *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; Am. Psychiatr. Assoc. 2013)

criteria to form groups, with the clinical group qualifying for a diagnosis of major depressive disorder (MDD) or a specific anxiety disorder and the control group composed of healthy individuals carefully screened for psychopathology. Other studies create groups by placing cut scores on the continuous score distribution of a self-report scale, often selecting participants from the upper and lower extremes of the distribution. Investigations using these designs have revealed numerous differences between pathological and normal groups, not only on mood-related measures but also on measures of cognition, behavior, psychophysiology, neurobiology, and functioning. This is perhaps not surprising, as comparisons of extremes provide a fairly liberal test for differences. Unfortunately, these sorts of case-control designs also run the risk of exaggerating or distorting the true relationships between variables (Preacher et al. 2005). This is due not only to excluding the middle of the distribution, where most individuals reside, but also to the use of "supernormal" controls who, by virtue of having no history of psychopathology or very low levels of traits associated with emotional disorders (e.g., negative affect, worry), are quite atypical of the general population (Schaefer et al. 2017) and, arguably, not particularly normal.

These problems have spurred some investigators to adopt a more conservative research design, comparing disorder cases not with asymptomatic controls but with nonclinical controls who display core features of that disorder. An advantage of such designs is the potential to isolate, with greater precision, features uniquely associated with the disorder. For example, Gotlib and colleagues (1995) compared MDD-diagnosed individuals to nondiagnosed individuals with similar self-reported depressive severity. Analyses revealed many more similarities than differences between the groups on measures of psychosocial functioning and comorbid disorders. Parallel studies have been carried out for anxiety disorders, prompted by evidence that the core symptoms of these disorders—such as panic attacks, social fears, and excessive worry—are quite common in the general population. For example, research on social anxiety has shown that persons diagnosed with nongeneralized social phobia can be distinguished from supernormal controls with nongeneralized social fears, but not from subclinical controls with generalized social fears, on measures of psychopathology (Hofmann & Roth 1996). In a related line of work, our lab has shown that most severe worriers do not qualify for a diagnosis of generalized anxiety disorder (GAD), despite reporting trait worry levels as high as those of diagnosed individuals (Ruscio 2002). When compared to these worry-matched controls, GAD worriers are distinguished more by their perceptions of worry as uncontrollable and dangerous than by the actual frequency, intensity, or disruptiveness of their worry experiences (Ruscio & Borkovec 2004).

These findings suggest that the large differences usually observed between cases and controls can be attributed, at least in part, to sampling from the upper and lower ends of the distribution. Defining the normal group more conservatively results in fewer and smaller differences from the pathological group, hinting at a possible gradient of severity.

Research on Manifest (Observable) Structure Provides Strong Evidence for Continuity

Given the heavy reliance on case-control designs, evidence has been slow to accumulate about the nature and nosological significance of subsyndromal depression and anxiety. Progress has been slowed not only by the paucity of studies comparing syndromal with subsyndromal cases, but also by disagreements over which individuals belong in each of these groups. For syndromal depression, researchers have debated the appropriateness of studying depressive symptoms in nonclinical samples as an analog for clinical depression (Coyne 1994, Vredenburg et al. 1993) and of including bereavement-related depression in the depression syndrome (Horwitz 2015, Kendler et al. 2008). For subsyndromal depression, researchers have disagreed strongly over how

cases should be defined, leading to massive inconsistency in the samples used across investigations (Pincus et al. 1999, Rodríguez et al. 2012). Reviewing the studies published over a single five-year period, Pincus and colleagues (1999) observed that minor depression was defined in nine different ways, subthreshold depression was defined in five ways, and recurrent brief depression was defined in two ways; in some instances, different definitions were given the same name, and different names were used for the same definition. This variability has made it difficult to compare results across studies and to obtain a coherent picture of the boundaries of pathological mood.

Remarkably, despite this heterogeneity, results are strikingly consistent: Studies overwhelmingly support continuity between normal and pathological mood. Three major lines of evidence are summarized below, building on earlier reviews of the continuity of depression (Flett et al. 1997, Solomon et al. 2001) and extending them to anxiety. An important caveat is that all of the studies described in this section examined continuity in manifest structure, reflecting features of observable variables and measures that were used to represent mood. Manifest structure can provide clues to the underlying (or latent) structure of a construct, but does not test latent structure directly.

Evidence for a monotonic relationship between symptoms and outcomes. Studies have consistently revealed a dose-response relationship between increasing severity of depressive symptoms and increasingly adverse outcomes. For example, two investigations studied mutually exclusive groups defined by a progressively larger number of major depression symptoms: minor depression (2–4 symptoms), MDD with 5–6 symptoms, and MDD with 7–9 symptoms (de Graaf et al. 2010, Kessler et al. 1997). There were monotonic increases across the groups on measures of functioning (unemployment, quality of life, role impairment), clinical features (dysfunctional thinking, course of illness, comorbid disorders), risk factors (parental psychopathology), and treatment seeking. For most of these measures, differences between the two lower groups were no larger than differences between the two upper groups, with no evidence of a discontinuity at the MDD diagnostic threshold of five symptoms. Studies including a wider range of depression presentations have revealed the continuum more fully, showing that the odds of negative outcomes (e.g., functional disability, service utilization, public assistance, suicidal behavior) rise in tandem with the severity of depression (Chen et al. 2000, Cuijpers et al. 2004, Judd et al. 1997). For example, Judd and colleagues (1997) reported odds ratios for these outcomes largely in the range of 1–2 for the group with one depression symptom, 2–3 for the group with two or more symptoms but no depressive disorder, 3–4 for minor depression, 5–8 for dysthymia, and 9–15 for MDD or double depression (MDD superimposed on dysthymia) compared to the asymptomatic group.

The observed gradient extends to the bottom of the severity range: Interviewer-rated disability increases incrementally with the number (starting as low as one symptom), duration (starting as low as one day), and frequency of recurrence (starting as low as one episode per year) of depressive symptoms (Maier et al. 1997). The gradient extends to the top of the severity range as well: Even among inpatients hospitalized for MDD, there is a significant monotonic association between depression severity at the time of admission and impairment at the follow-up assessment four weeks after hospitalization (Goethe et al. 1993). In perhaps the clearest demonstration of the dose-response pattern, Angst & Merikangas (2001) found that the likelihood of family history of depression, history of suicide attempts, work and social impairment, and treatment seeking for depression—as well as the overall level of subjective distress—increased systematically with the number (0–9), duration, or recurrence of MDD symptoms. The general pattern is the same in adolescents, adults, and older adults (Lewinsohn et al. 2000).

Similar dose-response relationships have been reported for anxiety. For example, Kessler and colleagues (2006) compared four mutually exclusive groups defined by progressively more severe manifestations of panic and agoraphobia: isolated uncued panic attacks, panic attacks

with agoraphobia, panic disorder, and panic disorder with agoraphobia. Although all four groups reported high levels of comorbidity and treatment seeking, monotonic increases were observed across the groups in these correlates and in indicators of clinical severity, illness persistence, and role impairment. A similar pattern has emerged in research on GAD, where researchers have compared diagnosed individuals to individuals missing one or more DSM-IV criteria for the disorder. In adolescents, GAD missing one criterion had somewhat diminished associations with severity indicators such as chronicity and comorbidity, whereas GAD missing two criteria evidenced even smaller associations that differed more consistently from those observed for the full GAD syndrome (Beesdo-Baum et al. 2011). Comparable findings were observed in adults, where increasingly broad definitions of GAD were associated with gradual decreases in comorbidity (Ruscio et al. 2007). Above the diagnostic threshold, greater severity of GAD was associated with higher risk of onset of later disorders, with odds ratios increasing gradually across mild, moderate, and severe GAD cases (Ruscio et al. 2007).

Parallel results have emerged for social anxiety. Although nearly one-quarter of Americans report at least one excessive lifetime social fear that is associated with substantial anxiety or avoidance, only half that number qualify for a DSM-IV diagnosis of social anxiety disorder, with lifetime prevalence of the disorder increasing monotonically with number of social fears (Ruscio et al. 2008). Functional outcomes are best for asymptomatic controls (0 social situations feared) and grow progressively worse across individuals with social fears, social fears plus avoidance, and syndromal social anxiety disorder (Merikangas et al. 2002), with evidence of further worsening in syndromal cases with comorbid disorders (Wittchen et al. 2000). Among those diagnosed with social anxiety disorder, an increasing number of social fears is linearly associated with a more adverse clinical course as well as with greater avoidance, role impairment, comorbidity, and use of mental health services (Ruscio et al. 2008, Stein et al. 2010). The robustness of this pattern is noteworthy when contrasted with the numerous, mostly unsuccessful efforts to identify categorical social anxiety disorder subtypes based on the number (e.g., generalized versus nongeneralized) or type (e.g., performance versus interactional) of situations feared.

In summary, it is now clear that the cardinal symptoms of emotional disorders—such as depressed mood, panic attacks, and social fears—are found in many individuals without the full disorder. Dose-response relationships of symptoms with outcomes are the norm for both depression and anxiety, with little evidence of disjunctions in these relationships as a function of symptom number, severity, duration, or recurrence. Notably, even when controlling statistically for one another, depression and anxiety each continue to show monotonic relationships with outcomes (Balázs et al. 2013), confirming that the pattern is characteristic of both constructs.

Evidence for aggregation of syndromal and subsyndromal cases within families. Several of the studies reviewed above found elevated rates of parental psychopathology in general, and parental depression in particular, in participants with subsyndromal depression compared to healthy controls. Those studies generally relied on participants' reports about their relatives. However, a few studies have investigated familial aggregation using more direct assessment or verification of relatives' psychiatric history and have arrived at very similar conclusions. For example, family studies show both that (*a*) minor depression is elevated in the relatives of probands with MDD (Weissman et al. 1984) and that (*b*) MDD is elevated in the relatives of probands with minor or recurrent depression (Remick et al. 1996). These results extend to probands with subthreshold symptoms of depression, whose relatives have a rate of MDD that is intermediate to, and significantly different from, the rates in probands with MDD and probands with no lifetime mood disorder (Lewinsohn et al. 2003). When depression is measured dimensionally, a dose-response relationship emerges: In a sample of same-sex twins, the risk for lifetime MDD in the co-twin

increased monotonically as a function of the number of MDD symptoms reported by the index twin (Kendler & Gardner 1998). The study tested, but failed to find evidence for, discontinuity in the relationship; a single linear function fit the data best. Notably, family studies generally yield stronger evidence for the heritability of depression when depression in relatives is defined broadly, including subsyndromal as well as syndromal cases, rather than when the definition is narrowly restricted to MDD (Judd et al. 2002). This hints that efforts to uncover the origins of depression would be aided by taking a dimensional approach.

The evidence is more mixed regarding familial aggregation of syndromal and subsyndromal anxiety. Although some studies have reported elevated risk of the anxiety disorder under study among the relatives of participants with subsyndromal symptoms (e.g., Merikangas et al. 2002, Ruscio et al. 2005), they have tended to assess relatives' mental health history via participants' reports. Two family studies that interviewed relatives directly did not find increased risk for social anxiety disorder in the families of probands with subsyndromal social fears (Fyer et al. 1993, Knappe et al. 2009), although one of those studies did find elevated risk of other forms of psychopathology in relatives, including other anxiety, mood, and substance use disorders (Knappe et al. 2009). More definitive conclusions await investigation of other forms of anxiety, ideally involving direct assessment of relatives and measurement of multiple anxiety disorders at the syndromal and subsyndromal levels.

Evidence for spectrum models of depression and anxiety. Depressive disorders are represented in DSM-5 as separate categories, implying that they are distinct entities. An alternate conceptualization is that these disorders represent different manifestations of a single illness, with no sharp boundaries separating them from one another or from milder depressive states. Two lines of research provide strong support for this alternate conceptualization. First, apart from differences in the severity of depressive symptoms, minor depression is qualitatively very similar to MDD on a wide range of clinical features, personality correlates, and comorbidity patterns (Moore & Brown 2012). The similarities are equally striking between subsyndromal depression, dysthymic disorder, and MDD (Sherbourne et al. 1994), even when the subsyndromal group lacks the core symptoms of depressed mood and anhedonia (Sadek & Bona 2000).

Second, depressed individuals transition frequently and fluidly between depressive categories. In a landmark study, Judd and colleagues (1998b) analyzed the weekly symptom levels of a large cohort of patients who were diagnosed with MDD at intake and followed prospectively for 12 years. Over this period, patients spent approximately 15% of weeks at the MDD level, 27% at the minor depression or dysthymia level, 17% at the subsyndromal symptom level, and 42% without depressive symptoms, changing levels approximately twice per year on average. A similar dynamic course was observed in a community sample followed prospectively for one year, wherein more than one-third of individuals who began the year in one depressive category ended the year in another (Judd et al. 1997). This pattern has since been replicated in a primary care sample (Maier et al. 1997) and in representative community samples of adults (Chen et al. 2000) and adolescents (Angst & Merikangas 1997). Taken together, these findings provide compelling evidence for a unitary depression spectrum in which currently recognized syndromes shade into one another along a gradient of severity.

Research on anxiety has also shown that, despite their lower severity, subsyndromal anxiety symptoms exhibit substantively similar clinical characteristics, course features, and risk factors as the full syndromes, resembling diagnosed cases much more closely than noncases on these measures (e.g., Beesdo-Baum et al. 2011, Kessler et al. 2006, Ruscio et al. 2005, Wittchen et al. 2000). Far fewer studies, however, have followed anxiety disorders prospectively to detail their natural course, especially in comparison to subsyndromal forms of the disorders. A notable exception is a

large study by Merikangas and colleagues (2002) in which social anxiety symptoms were assessed repeatedly over 15 years in a representative community sample of young adults. The group with social anxiety disorder and the group with subthreshold fear and avoidance had similar family histories of phobias and very similar patterns of personality and sociodemographic correlates. While there was substantial within-group stability over the 15-year period, there was also substantial fluctuation between these groups and less severe symptom levels over time. Although preliminary, these results suggest that a spectrum model may be appropriate for anxiety as well as for depression. Intriguingly, a prospective study of individuals who had one anxiety disorder diagnosis at baseline found that many transitioned to a different anxiety disorder over a six-year follow-up, with some individuals having a different anxiety disorder at each two-year assessment and still others transitioning to multiple other anxiety disorders over time (Hovenkamp-Hermelink et al. 2016). These results raise the possibility that the different forms of anxiety may be represented more validly by one broad anxiety spectrum than by multiple, disorder-specific spectra.

Taken together, these three lines of evidence provide strong support for a continuum between normal and pathological mood. The continuum appears to extend below and above existing diagnostic thresholds, with no evidence of discontinuity at any level of severity. Different forms of depression appear to represent different levels or phases along the same continuum, with highly permeable boundaries between them. Similar continua are evident within, and perhaps across, anxiety disorders, further supporting a dimensional model.

The Preponderance of Research on Latent Structure Supports Continuity

The studies reviewed above describe the manifest structure of depression and anxiety—the surface structure observable in measures designed to assess these constructs. By contrast, latent structure reflects the underlying organization of a construct—the structure that exists out in the world regardless of how researchers choose to conceptualize or measure it (Meehl 1992). Importantly, manifest structure need not match latent structure. For example, the scores on a depression scale may be normally distributed within a sample, appearing continuous at the manifest level, yet that distribution may arise from a mixture of two latent groups (e.g., depressed and nondepressed) whose scores overlap due to measurement error (see Ruscio & Ruscio 2008). For this reason, researchers caution against inferring latent structure from manifest structure (Grayson 1987, Murphy 1964). Arguably, it is latent structure that is most important for understanding the nature of a psychopathological construct and, in turn, for enhancing the validity of its classification and diagnosis (Meehl 1995).

Many latent structures are possible, and different methodological approaches are appropriate for detecting and describing different structures (Haslam 2002, Ruscio & Ruscio 2004). As this article is concerned with the boundary between normal and pathological mood, I focus on an approach that was designed specifically to probe this boundary: the taxometric method (Meehl 1995, 1999; Ruscio et al. 2006; Waller & Meehl 1998). Taxometric procedures use the relationships among measured variables to determine whether there is a discontinuity (qualitatively discrete groups) or continuity (only quantitative differences between individuals) at a given latent boundary. Extensive Monte Carlo research has shown that taxometric procedures are capable of making this determination with a high degree of accuracy (Ruscio et al. 2010, 2018).

Depression is among the most-studied constructs in the taxometric literature (for a review, see Haslam 2011), reflecting the long-standing and contentious debate over the relationship between normal and pathological depression. Although some taxometric studies have focused on narrower depressive constructs, such as putative MDD subtypes (e.g., hopelessness depression) or vulnerability factors (e.g., negative cognitive styles), most investigations have examined the boundary

between MDD and normal mood, with nearly 20 such investigations published to date. Roughly two-thirds of these studies reached a dimensional conclusion, suggesting that major depression differs in degree, rather than in kind, from normal sadness. For the minority of studies that reached a categorical (taxonic) conclusion, their authors often acknowledged, and close inspection of the results reveals, a fair amount of ambiguity in the findings. Many of these studies were carried out before the publication of Monte Carlo research that substantially improved understanding of the factors that affect performance of taxometric procedures. In a quantitative review of taxometric studies of a broad range of constructs, Haslam and colleagues (2012) found that more recent taxometric studies involving new methodological safeguards and more favorable data conditions have tended to yield dimensional results. Importantly, dimensional findings for depression predominate in studies with clinical samples as well as in studies with community or college samples, arguing against the possibility that a depression class was not detected because of insufficient cases with clinically significant depression in the sample. Nevertheless, with few exceptions, clinical samples have consisted of outpatients, and measures have been limited to symptoms of syndromal depression. It remains possible that future taxometric studies using other samples and measures will discover reliable latent classes—corresponding, for example, to very severe depression (which may be too rare for taxometric procedures to detect outside of inpatient settings), to presentations involving pronounced symptoms other than those included in criterion A for MDD (e.g., psychotic features), or to more symptomatically or etiologically homogeneous forms of depression (e.g., melancholic depression, for which taxometric research has yielded tentative categorical evidence).

Similar conclusions have emerged from taxometric investigations of the boundary between normal and pathological anxiety. Most taxometric studies of anxiety have focused on vulnerability factors (e.g., anxiety sensitivity) or related constructs (e.g., health anxiety), rather than on specific anxiety disorders. Nevertheless, the roughly one dozen studies that probed the boundary of anxiety disorders with normal anxiety all provided support for dimensional structure, revealing continua for social anxiety disorder, agoraphobia, separation anxiety disorder, obsessive-compulsive disorder, posttraumatic stress disorder, and pathological worry, the cardinal feature of GAD (for reviews, see Haslam 2011, Haslam et al. 2012). The consistency of the findings is striking, although the small number of studies per anxiety disorder and the absence of studies for some disorders (panic disorder, specific phobias) point to the need for further investigation.

In summary, the weight of the evidence supports the continuity of pathological depression and anxiety with milder emotional experiences—not only at the manifest level, but also at the latent level of analysis. These results indicate that there is no underlying discontinuity that separates cases and noncases into distinct groups. Instead, any categories that are formed along these dimensions should be understood to represent practical, rather than theoretically or scientifically meaningful, types. As such, the categories should be viewed as provisional, contestable, and modifiable (Haslam 2002), valuable only to the extent that they prove more useful than their underlying dimensions or alternative categorical distinctions. It is to this question of usefulness that I turn next.

Capturing the Continuum May Improve Clinical Utility

The continued use of categories to represent and diagnose mental disorders is often defended on the grounds that categories are more useful than dimensions in clinical practice. There are reasons to believe, however, that a diagnosis that recognizes subsyndromal symptoms of depression and anxiety may be quite useful to clinicians. I summarize those reasons briefly below. Due to space limitations, I focus on depression for illustration, although similar results in the smaller anxiety literature hint that these observations may also apply to anxiety.

Patients often seek treatment for subsyndromal symptoms. A sizable proportion of individuals who seek treatment for depression fall short of the diagnostic thresholds for MDD and dysthymia (Angst & Merikangas 1997). In fact, patients with subsyndromal depression outnumber those with syndromal depression in some clinical settings, especially primary care (Johnson et al. 1992, Maier et al. 1997). A diagnosis that captures subsyndromal cases would dramatically improve coverage of treated depression and would reduce reliance on Other Specified or Unspecified Depressive Disorder (formerly NOS) diagnoses. An expanded diagnosis could also improve awareness and detection of subsyndromal symptoms, which—even when not the focus of treatment—adversely affect outcomes for other clinical conditions (e.g., Musselman & Nemeroff 2000).

Subsyndromal symptoms are associated with substantial impairment. Subsyndromal depression is disabling. The level of disability is typically lower than that associated with syndromal depression, but is substantial in absolute terms (Broadhead et al. 1990, Wagner et al. 2000) and higher than the disability associated with major chronic medical conditions such as diabetes and arthritis (Wells et al. 1989). Prospective follow-up of patients has shown that disability fluctuates with the severity of depressive symptoms (Judd 2012) and that the presence of even a few depressive symptoms is associated with significant decrements in functioning compared to symptom-free periods within the same patients (Judd et al. 2002). Subsyndromal cases also have elevated rates of suicidal behavior (Carrellas et al. 2017) and, because of their greater prevalence, account for more suicide attempts than do syndromal depression cases (Johnson et al. 1992).

Subsyndromal symptoms predict escalation to more serious conditions. A sizable number of subsyndromal depression cases progress to a major depressive episode. Individuals with subthreshold depression are approximately twice as likely as nondepressed individuals to develop MDD (Lee et al. 2019), showing elevated risk even in the absence of prior major depressive episodes (Horwath et al. 1992). In general, the greater the number or severity of depressive symptoms, the greater the future MDD risk (Kendler & Gardner 1998, Lewinsohn et al. 2000). Studies with lengthy follow-up intervals suggest that approximately one-third of subsyndromal cases will eventually develop MDD (Angst & Merikangas 1997) and that the risks of later mental health problems are similar to those of persons with MDD (Fergusson et al. 2005).

Residual subsyndromal symptoms strongly predict relapse. Approximately one-third of patients recover from major depressive episodes with residual depressive symptoms, and these symptoms carry important prognostic information. Residual subsyndromal symptoms powerfully predict a higher rate of episode relapse and a much shorter time to relapse, independent of prior history of recurrent episodes (Judd et al. 1998a, Paykel et al. 1995). These results have been interpreted as suggesting that, even when patients no longer qualify for an MDD diagnosis, subsyndromal symptoms signal that the depressive episode is still active and requires further treatment (Judd 2012).

Treatment improves subsyndromal symptoms and prevents syndrome onset. Until quite recently, depression treatments were developed and tested almost exclusively for MDD. Fortunately, growing awareness of the significance of subsyndromal depression has spurred interventions and clinical trials focusing on patients with milder depressive symptoms. The evidence for pharmacotherapy is equivocal in this population (Barbui et al. 2011). However, psychotherapy appears efficacious in reducing the depressive symptoms and is associated with decreased incidence of MDD

at six-month follow-up (Cuijpers et al. 2014). The effect sizes are reliably higher than those for wait list or usual care, but smaller than those observed for MDD, perhaps in part because lower depression severity allows less room for symptom change (Cuijpers et al. 2014). Notably, many studies have investigated low-intensity interventions—such as guided self-help (Willemse et al. 2004) or Internet-based therapy (Spek et al. 2008)—that are exciting for their potential to offer cost-effective treatment to large numbers of individuals.

In summary, despite falling below conventional thresholds of clinical significance, subsyndromal symptoms are clinically relevant. Affected individuals seek treatment for these symptoms, experience real impairment, and are at risk for poor clinical outcomes and escalation to more severe conditions. At a minimum, they require recognition and careful monitoring. Evidence that minimally intensive, relatively low-cost treatment might successfully address these problems further emphasizes the value of identifying those affected and offering appropriate intervention. Regrettably, too often, subthreshold symptoms are overlooked or dismissed by clinicians, researchers, and policy makers. Contributing to this oversight is a classification system that does not recognize symptoms below traditional diagnostic thresholds. Coupled with the dimensional findings reviewed above, this points to the need for a new approach to diagnosis.

IMPLICATIONS FOR DIAGNOSIS

The possibility of shifting to a dimensionally based classification of psychopathology has received increasing attention in recent years (Helzer et al. 2008a, Insel et al. 2010, Krueger et al. 2005). Whereas some proponents advocate for an entirely dimensional system (Brown & Barlow 2009, Kotov et al. 2017, Widiger & Samuel 2005), others have proposed more modest solutions, such as using dimensions alongside DSM diagnoses (Helzer et al. 2008b) or adding severity ratings above the diagnostic threshold so that the original DSM categories are preserved (Brown & Barlow 2005).

I submit that none of these solutions is ideal because no one system is appropriate for all the tasks to which diagnoses are applied. While some activities are best served by dimensional diagnosis, others require categories, even when the underlying condition is dimensional. I further suggest that the DSM continues to be used in part because it offers one major advantage over alternative systems: It provides a set of standardized, operational definitions for diagnosis that are used by almost everyone in the field. Standardized definitions provide a common language for clinicians, facilitating coordination of care among treatment providers, information sharing with patients and third-party payers, and consultation of treatment guidelines for informed clinical decision making. Standardized definitions also benefit researchers by allowing knowledge to accumulate across laboratories and studies and by ensuring that epidemiologic estimates are comparable across regions and over time. Thus, to be viable, diagnoses must (*a*) have utility—be useful for the major tasks facing mental health professionals, and (*b*) be standardized—provide consistent definitions that are widely adopted, enabling communication and advancement of knowledge. Below, I outline a proposal that addresses both of these priorities.

Researchers Should Use Dimensional Diagnosis

The literature reviewed above supports the use of dimensional diagnosis in research on depression and anxiety. Dimensional diagnosis, referring to the assignment of a dimensional score to represent a respondent's location on a continuum, has two major advantages for research. First, the goal of research is to represent reality as accurately as possible, and the best available evidence suggests that depressive and anxiety disorders are dimensional in nature. Second, advances are made when statistical power is adequate to detect associations of interest, and power is significantly weakened

by the artificial dichotomization of dimensional variables (Cohen 1983). The greater reliability and validity of continuous rather than discrete measures of psychopathology (Markon et al. 2011), especially for variables that are dimensional in nature (Ruscio & Ruscio 2002), suggest that dimensions will have greater utility than categories for studying pathological mood.

For a dimensional diagnosis to be viable, however, we will need a definition that is adopted widely and consistently. What should this definition be? Many options are possible, and the available research provides little guidance for choosing among them. Although we know a good deal about the dimensions that underlie psychopathology in general (Caspi et al. 2014, Kotov et al. 2017) and depression and anxiety in particular (Clark & Watson 1991, Watson 2005), there have been few attempts to specify how dimensional diagnoses should be constructed (for notable exceptions, see Brown & Barlow 2009, Klein 2008). This leaves several questions to be addressed before dimensional diagnoses are ready for use.

Should dimensions be based on, or independent of, DSM-5 constructs? The constructs of depression and anxiety have a long history in mental health research and practice (Crocq 2015, Horwitz et al. 2017). Further subdivision of these constructs, as when panic disorder was distinguished from GAD and social phobia was distinguished from simple phobia, occurred in response to observed differences in etiology and treatment. These constructs have been studied extensively, their symptom lists have been curated by expert scientists and clinicians, and they are already familiar to professionals working in the field. What's more, as shown above, dimensional forms of these constructs are robustly associated with a wide range of important validators and outcomes. Taken together, these considerations make a compelling case for basing dimensional diagnoses on DSM constructs.

At the same time, numerous authors have expressed dissatisfaction with the DSM representation of emotional disorders (Angst & Merikangas 2001, Brown & Barlow 2009, Maser et al. 2009). Although some of the criticisms would be addressed by switching from categorical to dimensional versions of the DSM disorders (e.g., failure to recognize subthreshold presentations), other criticisms may not (e.g., large associations and overlapping risk factors across disorders). Moreover, dimensionalizing existing DSM categories is viewed by some as a missed opportunity to derive dimensions empirically rather than perpetuating historical conventions (First 2005). For example, some structural (Watson 2005) and behavioral genetic (Hettema et al. 2005, Kendler et al. 2003) research has revealed that, rather than dividing into depression and anxiety factors, emotional disorders separate into distress (or anxious-misery) and fear factors, with the former including unipolar depression as well as GAD and posttraumatic stress disorder, and the latter including the phobias, probably social anxiety disorder and panic disorder, and possibly obsessive-compulsive disorder. Several dimensional alternatives to the DSM have emerged in recent years (Harvey et al. 2004, Insel et al. 2010, Kotov et al. 2017), each deemphasizing traditional syndromes in favor of dimensions that span diagnostic categories. Although each of these alternatives has promising features, all are in relatively early stages of development and testing, and it remains to be seen which, if any, will replace the DSM as the dominant classification in the field. Given the pressing need for dimensions that are ready for immediate use, as well as repeated demonstrations of the utility of dimensions derived from DSM disorders, dimensional diagnoses based on prevailing concepts of depression and anxiety offer a sensible, if temporary, way station to the dimensions that are ultimately shown to be most valid and useful.

How many dimensions are needed? **Table 1** presents several examples of dimensional approaches to diagnosing depression and anxiety that differ in their specificity. In the broadest example, depression is represented by a single severity dimension. This parsimonious option is

Table 1 Possible diagnostic dimensions for depression and anxiety at three levels of specificity

Level	Depression	Anxiety
Broad	Severity	Severity
Intermediate	Intensity (number/strength of symptoms) Course (chronicity/recurrence)	Intensity (strength of subjective fear/anxiety) Avoidance (overt or covert) Pervasiveness (number/range of situations) Course (chronicity/recurrence)
Narrow		Separation anxiety Selective mutism Specific phobia Social anxiety Panic Agoraphobia Generalized anxiety

supported by research showing that a simple count of the number of DSM MDD symptoms (from 0–9) is sufficient to powerfully predict family history of depression, history of suicide attempts, work and social impairment, and treatment seeking for depression (Angst & Merikangas 2001). In the intermediate example shown, separate dimensions are rated for intensity (reflecting the number or strength of current depressive symptoms) and course (reflecting the chronicity or recurrence of depression). This option is modeled closely after the two-dimension system proposed by Klein (2008) for classifying depressive disorders and is supported by demonstrations that adding a measure of course to a measure of symptom severity strengthens associations of depression with external validators and improves prediction of functioning and recovery (Angst & Merikangas 2001, Shankman & Klein 2002).

Parallel options for anxiety are proposed at the broad and intermediate levels. A single dimension of anxiety severity could be advocated given extensive evidence for shared phenomenology, neurobiology, and etiology—as well as high rates of comorbidity and transition between diagnostic categories—across all anxiety disorders (for a review, see Norton & Paulus 2017). At the same time, researchers commonly distinguish different aspects of anxiety severity that cut across disorders, suggesting that a more variegated representation of severity may enhance utility. Four intermediate dimensions are suggested by the literature. The most common are intensity (the strength of subjective fear and anxiety) and avoidance (the extent of overt or covert avoidance). An additional dimension of pervasiveness (the number or variety of situations that evoke anxiety) may be warranted to distinguish circumscribed from more generalized fears and to account for the concentration of multiple forms of anxiety in some individuals. As anxiety disorders are typically viewed as chronic conditions, a course dimension may be less informative for anxiety than for depression; however, indications that brief and recurrent anxiety episodes may be common in the community (e.g., Angst et al. 2006, Lee et al. 2009) tentatively suggest that a course dimension may prove useful for characterizing the full anxiety continuum. Finally, the narrowest level in the table includes the anxiety disorders recognized by DSM-5,[1] reflecting the myriad forms that

[1]In keeping with the decision to remove obsessive-compulsive disorder, posttraumatic stress disorder, and acute stress disorder from the anxiety disorders chapter in DSM-5, these conditions were excluded from the table, although their many similarities to anxiety disorders (see Abramowitz & Jacoby 2015, Jones & Barlow 1990) may ultimately support their integration into this framework.

anxiety may take. Dimensionalizing individual anxiety disorders preserves distinctions for which there is some genetic, neural, and behavioral evidence (Craske et al. 2009, Smoller et al. 2008) and conforms with descriptions of spectra at the disorder level (Cassano et al. 1997, Schneider et al. 2002).

All three of these levels are meaningful, and all have some empirical support. Which level, then, is most defensible as the basis for dimensional diagnosis? Narrower dimensions, when relatively orthogonal, convey more detailed information that may translate into improved utility. However, narrower dimensions are not always necessary; an analogy could be made to the psychotic disorders, where diagnosis focuses on intermediate processes (e.g., delusions, hallucinations) rather than on the specific content of those processes (e.g., persecutory, grandiose). Broader dimensions are more efficient and may better capture cases exhibiting a nonspecific or mixed pattern of symptoms. However, in the absence of data directly comparing these options, the most conservative choice is to begin at the most specific[2] level (that is, the intermediate two-dimension option for depression, and the narrow disorder-specific option for anxiety), as this aligns most closely with current practice. At the same time, we should vigorously pursue comparisons of dimensional diagnoses at this level with dimensions at the broader levels. If broader dimensions compare favorably to narrower dimensions in validity and utility, the broader dimensions should be preferred, as a system involving fewer dimensions would be less burdensome and more likely to be widely adopted.

A final point to be considered is whether functional impairment should serve as an additional dimension within any of these diagnostic options. While distress is intrinsic to the symptoms of depression and anxiety (Spitzer & Wakefield 1999), impairment varies widely across individuals and would aid in determining clinical significance, especially for individuals with fewer, milder, or more transient symptoms (Baumeister & Morar 2008, Pincus et al. 1999). Unfortunately, two individuals with identical symptoms may experience very different levels of impairment due to factors that may be unrelated to their symptoms, such as financial resources, social supports, coping strategies, and number and demandingness of roles. A purer measure of symptoms, unconflated with these factors, would likely prove more useful to researchers seeking to characterize symptom dimensions and identify their causal and maintaining factors. For this reason, it may be best to exclude impairment from dimensional diagnoses of depression and anxiety, although impairment will almost certainly play a role in defining categorical diagnoses for clinical decision making (see below).

How should dimensions be constructed? After deciding which dimensions to use, the question remains how those dimensions should be constructed. For example, how large a rating scale should be used to quantify each dimension? The more points there are on the dimension, the greater the potential sensitivity to differences between individuals and within individuals over time (Kraemer et al. 2004). This sensitivity must be balanced against the greater simplicity and efficiency of a smaller dimension. An elegant example that strikes this balance is a four-point scale proposed by Klein (2008) for rating the course of depression, which captures elements of both chronicity and recurrence; pending demonstrations of the validity of this scale, a similar scale may be appropriate

[2]Of course, even narrower dimensions than these are possible (e.g., specific phobia could be further subdivided into situational fears, animal fears, blood-injection-injury fears, and so on), as are broader dimensions (e.g., an overarching internalizing dimension). However, the improvement in prediction yielded by very narrow dimensions may be too trivial to warrant the increased diagnostic complexity, whereas the very broad internalizing dimension is neither specific to emotional disorders nor able to model the symptoms of mood disturbance with adequate fit (Waszczuk et al. 2017), suggesting that neither of these options is ideal as the basis for dimensional diagnosis of pathological mood.

for anxiety. Past studies have successfully measured the intensity and pervasiveness dimensions of anxiety by counting the number of symptoms (e.g., Yonkers et al. 1996) or feared situations (e.g., Ruscio et al. 2008), respectively, that are endorsed from a standardized list. A more sensitive and reliable assessment could be achieved by rating individual symptoms or situations on Likert-type scales and then summing across ratings, although it is critical that standardized scales be used to ensure consistency of diagnoses across studies. Research is needed to test whether the psychometric advantages of larger scales justify their greater assessment burden. Follow-up work could test whether more complex scoring algorithms that assign greater weight to some symptoms (e.g., suicidality) than to others (e.g., appetite disturbance, insomnia) (see Watson 2009) enhance validity without compromising feasibility.

A further consideration for intermediate and narrow dimensions concerns whether, and how, the dimensions should be combined to arrive at a dimensional diagnosis. Past proposals have recommended that each patient be described by a set of dimensions (Brown & Barlow 2009, Klein 2008, Shear et al. 2008). For example, Brown & Barlow (2009) proposed a system of 11 dimensions (including one depression, five anxiety, one mania, two temperament, and two avoidance dimensions), the scores of which are plotted on a common *y*-axis scale and interpreted as a profile. Separate dimensions are appealing for their preservation of distinct clinical features, especially if those features differ meaningfully in their associations with validators and outcomes. However, a set of dimensions is considerably more difficult to interpret, communicate, and aggregate across research participants than a single dimensional score (Ruscio 2008). It may be sensible to begin with the simplest system—one that combines the dimensions into a single composite score per individual—unless the dimensions are weakly correlated or unless multidimensional diagnoses are clearly superior in predicting outcomes of interest. All else being equal, preference should go to the system requiring the least time, effort, cost, and special expertise (Andrews et al. 2007, First 2005) to encourage uptake not only by researchers but also by clinicians, a topic to which I turn below.

Clinicians Should Use Dimensional Diagnosis for Some Purposes and Categorical Diagnosis for Others

The literature summarized above indicates that, for many clinical as well as research activities, dimensions will be more informative than categories. In particular, the demonstrated predictive power of severity scores—both below and above existing diagnostic thresholds—suggests that dimensional diagnosis should be favored for tasks that fundamentally involve a judgment (that is, a response along a continuum), such as prognosis estimation, preventive screening, and treatment planning. The routine use of dimensional measures like blood pressure, cholesterol levels, and body mass index in other areas of health care suggests that barriers to clinicians' use of dimensions (see First 2005) are not insurmountable, and that we should not be too quick to assume that researchers and clinicians differ in their diagnostic needs.

Nevertheless, there are clinical and administrative situations in which categorical diagnosis is unavoidable. These are generally situations that require a decision (that is, a choice between discrete options): whether to initiate treatment, to offer Treatment A or Treatment B, to hospitalize, to grant disability compensation, to provide insurance payment for therapy, and so on (Haslam 2002, Kraemer et al. 2004). In such situations, the focus should be on how to make the categorical decision most defensibly. For clinical conditions that are dimensional in nature, external criteria such as role impairment, treatment seeking, and the likelihood of progression to more serious conditions can be used to draw a threshold above which the condition is judged sufficiently severe to warrant diagnosis (Ruscio 2009, Widiger & Clark 2000). As with hypertension,

hypercholesterolemia, and obesity, clinical medicine offers numerous examples of dimensional measures on which thresholds are imposed for categorical decisions (Kessler 2002).

Problems with the current model of categorical diagnosis. If categories are required for clinical decisions, then which categories should be used? One option is simply to use the DSM-5 depressive and anxiety disorder categories. Although continued use of established categories has obvious advantages, two problems highlighted in the sections above suggest that a change is needed.

First, especially for depressive disorders, the DSM classification is cumbersome. Its separation of MDD and dysthymia/persistent depressive disorder from one another and from other forms of depression grouped in the Other Specified Depressive Disorder category is incompatible with the well-replicated finding that all of these conditions share spectrum relationships with one another and exist along a gradient of clinical severity. The data strongly suggest that these varied manifestations of depression would be represented more validly and parsimoniously as a single depressive construct.

Second, the DSM classification is incomplete. It appears to miss many individuals who fall short of current diagnostic thresholds yet are troubled by their symptoms and seek treatment for depression. These subthreshold cases fall in the nebulous gray zone between normal and pathological mood, between well-being and disorder. There is no place for them in the current classification system.

One possible solution is to lower existing diagnostic thresholds so that more individuals qualify for a diagnosis. However, a reduction that is substantial enough to address the false negatives problem would more than double the number of people who are identified as having a depressive disorder (Johnson et al. 1992, Judd et al. 2002). Many people who would be diagnosed by the expanded criteria have symptoms that are milder in severity and impact than is implied by the DSM-5 requirement of a "clinically significant disturbance" that is "associated with significant distress or disability" (Am. Psychiatr. Assoc. 2013, p. 20). Thus, lowering thresholds would deviate from the DSM convention of setting the diagnostic boundary at the point where symptoms are considered severe enough to require professional care (Haslam 2002). Unfortunately, leaving thresholds at current levels means that comparatively milder yet still troubling symptoms will continue to go unrecognized, despite their importance for prognosis and effective clinical management, and despite the possibility that low-cost interventions could successfully treat these symptoms and prevent their escalation to more serious problems.

A multithreshold model of diagnosis. Breaking free of the single-threshold model historically used by the DSM would open up the possibility of a different solution. As a single diagnostic threshold cannot serve all purposes well, a better option is to adopt a multithreshold model of diagnosis. The simplest version of this model, and the one that is best supported by available research on depression, is a dual-threshold model in which two diagnostic categories are formed along the depression continuum (see **Figure 1*a***). The upper category, corresponding to depression of moderate or greater severity, meets the conventional threshold for clinical significance and captures cases who would be recognized by DSM-5 as having a depressive disorder. I call this category major depression to preserve the familiar label for individuals experiencing a clearly pathological level of symptoms associated with marked impairment. The lower category, corresponding to depression of mild severity, includes cases whose symptoms fall below the conventional threshold for clinical significance yet are of sufficient intensity, duration, or impact that they warrant attention—whether in the form of watchful waiting or a low-intensity intervention (Hegel et al. 2006). I call this category mild depression to recognize its lesser severity while avoiding the implication of triviality or unimportance (Pincus et al. 1999).

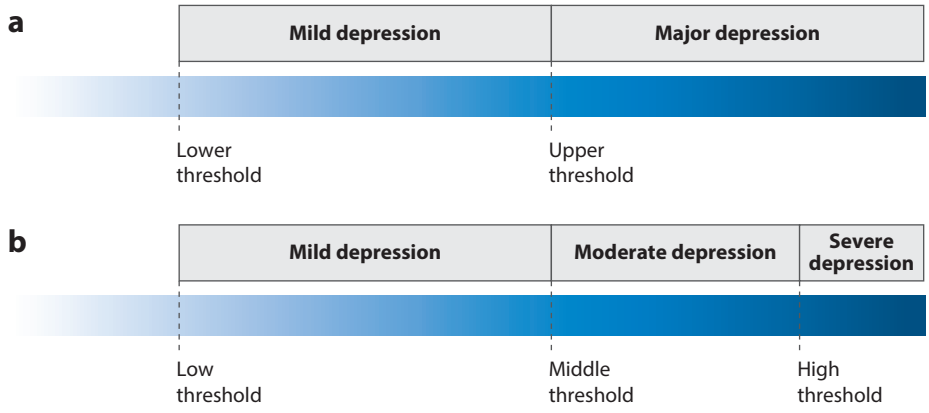

Figure 1

Schematic depicting a multithreshold model of diagnosis, illustrated here for depression. (*a*) A dual-threshold model in which two thresholds are superimposed on the underlying depression continuum to form diagnostic categories of mild and major depression. (*b*) A triple-threshold model in which diagnostic categories of mild, moderate, and severe depression are formed along the depression continuum.

This proposal differs in one key respect from those raised by other proponents of dimensional classification, who have generally advocated the flexible application of thresholds along the dimension on an as-needed basis for different types of decisions (e.g., Maser et al. 2009, Widiger & Samuel 2005). To prompt routine recognition of mild cases and permit communication and coordination across settings, we need formal, standardized, accessible diagnostic criteria for any proposed categories. Thus, mild depression, as well as major depression, should be included in future editions of the DSM. In principle, additional diagnostic categories could be formed along the dimension, although this should only be done if the improvement in clinical utility clearly outweighs the disadvantages of greater complexity (First et al. 2004). For example, some studies have found that severe depression, identified by a threshold higher than the DSM-5 requirements for MDD, is associated with sharply elevated familiality of depression as well as markedly worse psychosocial and clinical outcomes (for a review, see Solomon et al. 2001). If a diagnosis of severe depression would lead to different clinical decisions—indicating, for example, the need for different or more intensive treatment than moderate depression—then an additional, higher threshold may be warranted (see **Figure 1*b***).

Categorical diagnoses are intended for clinical decisions, so the categories should be actionable: Membership in one category should carry a different set of implications for clinical management than membership in another. A natural extension of this proposal is to align the diagnostic thresholds with stratified models or hybrid stepped/stratified models of care that provide guidance in choosing a level of care appropriate for the level of mood disturbance (see Clark 2018, Richards et al. 2012). For example, patients diagnosed with mild depression might be offered a low-intensity intervention, such as guided self-help, a psychoeducational group, or minimal-contact psychotherapy, with the high-intensity interventions of traditional individual psychotherapy or pharmacotherapy reserved for patients who are diagnosed with major depression or whose mild depression is not addressed adequately by less intensive care. An important question for future research is whether all mild depression cases should begin with a low-intensity intervention, given that such interventions have proved efficacious in the aggregate for subthreshold depression (Cuijpers et al. 2014, Willemse et al. 2004), or whether individuals with particularly concerning symptoms (e.g., suicidal ideation), a prior history of more severe

depression (i.e., residual cases), or other features indicating an especially high risk for progression to more severe depression (e.g., significant anxiety, family history of depression) (Klein et al. 2009) should be offered a high-intensity intervention from the outset. Enrolling mild cases in clinical trials would help generate knowledge about treatment need and treatment response at different levels of the depression continuum and would inform the construction of treatment selection models to aid in choosing the optimal intervention for each patient (Cohen & DeRubeis 2018).

Where should thresholds be placed? Several questions must be addressed before a dual-threshold model of diagnosis can be implemented. Perhaps the most challenging question is where to set the threshold for the mild depression category. There is presently no consensus definition for subthreshold depression, and the numerous proposed definitions vary widely in their criteria and corresponding prevalence (Cuijpers & Smit 2004, Pincus et al. 1999, Rowe & Rapaport 2006). Studies have shown that persons reporting as few as two symptoms of depression exhibit substantially poorer functioning than asymptomatic individuals and are at significantly elevated risk of developing MDD (Horwath et al. 1992; Judd et al. 1994, 1997), hinting that this could be a reasonable lower bound for mild depression. However, there is a need for research in which criteria are varied systematically within the same sample—and compared in their associations with clinically relevant outcomes—to determine the number, frequency, and course (duration, rate of recurrence) of symptoms that together define a sensible lower threshold.

Similar efforts are needed to establish the combination of symptoms that should define the upper threshold along the mood continuum. If, as proposed in this review, the new major depression category represents an amalgam of previously separate conditions (MDD, dysthymia, recurrent depressive disorder), their criteria will need to be consolidated, and a single threshold selected, to identify individuals with clinically significant depression. Evidence that many forms of depression can result in similar impairment (see Maier et al. 1997) raises the possibility of a new kind of diagnostic threshold, one that could be met by different combinations of criteria (e.g., many symptoms of relatively short duration versus fewer symptoms that are chronic or recurrent) rather than by just one criterion set.

Other authors (Kessler 2002, Kraemer et al. 2004, Swets et al. 2000) have described methods that can be used to locate optimal thresholds along continua for the purpose of decision making. The task is easier when there are nonlinearities in the relationship between the severity dimension and either soft (e.g., subjective distress, quality of life) or hard (e.g., unemployment, suicide attempt) outcomes. Unfortunately, nonlinearities have been hard to come by in research on depressive and anxiety symptoms (see Markon 2010). This suggests that clinicians will need to identify clinically meaningful levels of these outcomes that should be used to locate the threshold for each diagnostic category (Ruscio 2009).

Balancing costs and benefits. Like the selection of thresholds for continuous conditions in clinical medicine, the placement of the dual thresholds proposed here will depend on a cost–benefit analysis. Treating mild depression would make sense if the magnitude of (*a*) the current suffering or impairment or (*b*) the risk of progression to more severe illness makes the benefits of intervening outweigh the costs (Kessler et al. 2003). A variety of costs must be considered in this analysis, among them the financial cost of the intervention, the opportunity cost of diverting limited clinician time away from patients with greater need, the patient's cost in time and expenses, and the risks of any side effects that may result from the intervention. These need to be weighed against the possible costs of not intervening, both to the patient (e.g., suffering, poorer health care outcomes) and society (e.g., productivity loss, use of public assistance).

Importantly, costs and benefits evolve over time as new treatments become available, the cost of treatment changes, or shifts occur in the availability of public health funds or the political priority placed on mental health prevention and treatment (see Clark 2018, Cuthbert 2005). It follows that, to remain maximally useful for decision making, the categorical thresholds that we set now will need to be adjusted in the future. Recognizing that these thresholds are practical constructions that are imposed for specific purposes, rather than true boundaries that actually exist in nature, will help avoid reification (Hyman 2010) and prompt explicit consideration of costs and benefits as thresholds are revised.

CONCLUSIONS AND FUTURE DIRECTIONS

In the domain of depressed and anxious mood, there is little evidence for distinct states of normality and pathology. Instead, normality blends imperceptibly into abnormality along a continuum of severity. What will we do with this information? If we are serious about advancing knowledge of these conditions and their causes, we should use dimensional diagnosis in research. Diagnoses that reliably capture an individual's position along a dimension are capable of much finer distinctions than binary diagnostic categories, and these distinctions explain substantial variance in key outcomes. Evidence that dimensional measures of depressive and anxiety disorders outpredict categories derived from the same diagnostic interview (Prisciandaro & Roberts 2009, Ruscio 2010) further supports the use of dimensional diagnoses when investigating the nature, causes, and consequences of these disorders.

Prior attempts to encourage a switch to dimensional classification have not swayed large numbers of researchers to abandon the DSM categories in favor of dimensions. Despite well-documented limitations of DSM-5 diagnoses, we continue to use these categories for a reason: They provide standardized, operational definitions of clinically relevant phenomena that are useful and, because they are widely employed, allow findings to accumulate across the field. I have argued that dimensional diagnoses must meet the same needs before they are seriously considered as an alternative to DSM diagnoses in future research. For that reason, dimensions may have the most traction if they adhere closely to constructs that are already familiar to, and accepted by, many investigators. These include the dimensions of symptom intensity and course for depression and the dimensions corresponding to recognized disorders for anxiety. They are dimensions that researchers can begin using now while alternatives that are more ambitious or complex are being tested.

Further barriers to the adoption of dimensional diagnosis have been the assumptions that (*a*) diagnosis in research must look the same as diagnosis in clinical practice and (*b*) clinical practice is best served by categorical diagnosis. I have argued instead that (*a*) dimensional and categorical diagnosis are each useful for certain purposes, and (*b*) the critical distinction is not between research and practice but between judgments (which favor dimensions) and decisions (which favor categories). Given the clinical relevance of subsyndromal depression, a dual-threshold model of diagnosis may have greater utility than the traditional single-threshold model for categorical decisions. Adding a lower threshold—designating the mildest level of depression that warrants clinical attention—would expand coverage to treatment-seeking individuals whose symptoms are troubling but not markedly impairing and who may respond to less intensive interventions. At the same time, retaining an upper threshold—designating moderate to severe depression—would mean that that the major depression category need not be "watered down" to encompass milder symptom presentations, and could be defined instead by the level of depression at which more intensive intervention is warranted. For anxiety as well as depression, a multithreshold model would shift the conversation away from philosophical debates over the definition of abnormality and toward practical questions about when and how to intervene.

The current proposal represents a departure from the convention of equating diagnosis with a need for professional treatment (e.g., Regier & Narrow 2002). Instead, it takes the position, previously articulated by others, that the definition of a case and the need for treatment represent separate concerns (Kendler 2010, Kessler et al. 2003, Spitzer 1998). In that regard, this proposal may offer a useful model for other domains of psychopathology in which mild conditions, falling below current diagnostic thresholds yet associated with evident impairment and significant risk of progression to more severe conditions (see Insel 2010), are judged to merit formal recognition through standardized diagnostic criteria. The growing popularity of spectrum models (Maser & Patterson 2002) and emerging appreciation of a lifespan view of psychopathology in which prodromal and residual phases play an important role (e.g., Miklowitz & Cicchetti 2006) highlight the need for a diagnostic framework that can capture the severity level of a particular patient at a particular occasion. Emotional disorders are increasingly being viewed as chronic conditions that may wax and wane over time (Judd 2012, Maser & Patterson 2002, Yonkers et al. 2003), analogous to chronic physical conditions, such as asthma or diabetes, in which acute exacerbations periodically occur (Widiger & Clark 2000). The ideal diagnostic system would support investigation and characterization of the underlying continuum (via dimensional diagnosis) while also supplying thresholds along the continuum to aid decisions about the appropriate level of care given the current level of severity and phase of illness (via multithreshold categorical diagnosis).

The dimensional structure of depression and anxiety has implications beyond diagnosis, particularly for the study of etiology. As the causal processes that give rise to dimensions versus categories are often different, the latent structure of a construct constrains the plausible etiological models for that construct, with dimensions most likely to result from the additive contribution of many small genetic and/or environmental influences (Meehl 1992, Ruscio et al. 2006). This points to the need for etiological models that identify which factors, under what conditions, combine to determine where a person will fall along the depression or anxiety dimension (see **Figure 2**). One line

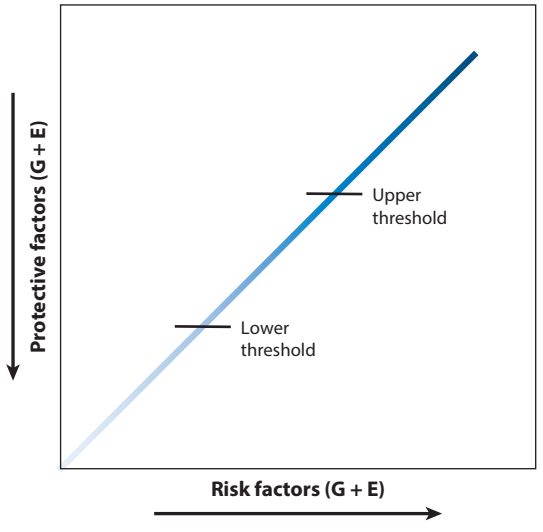

Figure 2

Multifactorial model depicting the aggregated causes of mood disturbance, illustrated here for depression. Current depression severity is represented as a function of risk and protective factors, both genetic (G) and environmental (E), that together determine an individual's position along the continuum and in relation to the dual diagnostic thresholds.

of research might seek to identify spectrum-relevant risk factors—those (mostly early-emerging and enduring) factors that are responsible for placing the individual above negligible levels on the dimension. A second line of research might investigate threshold-relevant risk factors—those (mostly proximal and acute) factors that propel the individual over the lower and, especially, the upper threshold reflecting clinically significant disturbance. That research could examine whether acute exacerbations in severity are more likely to occur when there is extreme deviation on multiple risk factors simultaneously, leading to a more catastrophic impact on functioning. Complementary lines of research could explore protective factors or counterforces that propel movement toward the lower end of the dimension, leading to reductions in severity and, potentially, increases in well-being (Siddaway et al. 2018).

Finally, recognition of a continuum connecting normal and abnormal emotional experiences encourages research aimed at a better understanding of emotion at all levels. Although investigations of pathological mood have focused disproportionately on depression and anxiety, many other forms of mood disturbance are important for psychopathology (Berenbaum et al. 2003). Mental disorders characterized by excessive anger (Cassiello-Robbins & Barlow 2016), disgust (Cisler et al. 2009), or positive emotions (Gruber 2011) represent promising targets for future extensions of this work. Given the central role of emotions in mental illness and the fundamentality of emotions to the human experience, delineating these boundaries could have a major impact on psychological theory and practice.

SUMMARY POINTS

1. Multiple lines of evidence support the continuity of syndromal depression and anxiety with milder emotional experiences.
2. Subsyndromal symptoms are disabling, responsive to treatment, and clinically informative, suggesting that capturing these symptoms would improve the clinical utility of diagnosis.
3. Researchers should use dimensional diagnosis when studying depression and anxiety.
4. Clinicians should use (*a*) dimensional diagnosis for activities requiring judgments and (*b*) categorical diagnosis for activities requiring decisions.
5. As different levels of severity may suggest different decisions, a multithreshold model of diagnosis could prove more useful for clinical decision making than the DSM's single-threshold model.

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