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• Forum •

Subthreshold depression

Understanding subthreshold depression

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The concept of subsyndromal, subthreshold or subcase depression has received considerable attention in recent decades. Judd and colleagues[1] — who reported that patients with depression are symptomatic more than 60% of the time and that most of that time is spent in subthreshold states — proposed the following operational definition of subsyndromal depression: 'two or more simultaneous symptoms of depression, present for most or all of the time, at least 2 weeks in duration, associated with evidence of social dysfunction, occurring in individuals who do not meet criteria for diagnoses of minor depression, major depression, and/or dysthymia.'

Distinguishing major depressive disorder from subthreshold depression and distinguishing the different types of subthreshold depression from each other is one example of a fundamental limitation of our current psychiatric nosology. The elaborate classification systems currently in use in psychiatry are based on subjective descriptions of symptoms. The detailed phenomenology includes descriptions of multiple overlapping clinical subtypes, but there are no biological characteristics that distinguish one subtype from another. Moreover, different disorders can exhibit similar clinical symptoms and the same disorder can have different symptomatic patterns in different individuals.[2] In the absence of biological markers, establishing thresholds in terms of frequency, duration or severity of symptoms that define when a symptom cluster becomes 'psychopathological' and that distinguish one symptom cluster from another symptom cluster will always be problematic.

The original hope for ICD-10 and DSM-IV was that the diagnostic emphasis on observable behaviors (rather than on presumed cognitions) would advance the reliability and validity of the diagnostic categories. Based on this approach, the core entity of depressive disorders in DSM-IV became a uniform major depressive disorder with modifiers for different subtypes (e.g., psychotic or atypical) and for circumstances presumed to have clinical significance (e.g., seasonal or postpartum depression).[3-5] The authors of ICD-10 recognized the need for a more comprehensive understanding of affective disorders but did not feel that the related science was sufficiently mature: 'the relationship between etiology, symptoms, underlying biochemical processes, response to treatment, and outcome of mood [affective] disorders is not yet sufficiently well understood to allow their classification in a way that is likely to meet with universal approval'.[5,9] The reported emphasis of the upcoming DSM-5 on the dimensional aspects of psychopathology may provide an opportunity to more clearly characterize the different manifestations of the different subthreshold conditions along the depressive continuum, but this remains to be seen.

It is my view that the depressive symptoms at the major, minor, dysthymic or subsyndromal levels are all integral components of the longitudinal clinical structure of major depressive disorder (MDD), with each symptom level representing a different phase of illness intensity, activity and severity.[1,2,6,7] The different depressive disorders included in official diagnostic systems (i.e., MDD, minor depression, dysthymia) and subsyndromal depression do not represent discrete disorders but, rather, are stages along a dimensional continuum of symptomatic severity.

Overcoming the current nosological problems will require changing the current phenomenology-based classification system to a classification system based on reliable neurobiological findings that takes into consideration the remarkable synchronization of psychological, social and cultural factors with biochemistry and physiology.[3,4] Studies of the relationship of stress and illness[2,4] that clarify the inter-related roles of social, psychological and biological factors in the cause, course and outcomes of mental disorders provide one clear example of the potential benefits of this more integrative approach to the conceptualization of mental disorders.

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It is now broadly accepted that psychological stress may change the internal homeostatic state of an individual. During acute stress adaptive physiological responses occur, including the increased adrenocortical secretion of hormones, primarily cortisol.[2-4] Several authors hypothesize that prolonged dysfunction of the stress response system, characterized by either hyper- or hypo-activity of the hypothalamic-pituitary-adrenal (HPA) axis, plays a central role in the etiology and course of mood disorders. There is accumulating evidence supporting the suggestion that depressive disorders include individuals with different treatment response profiles who have abnormalities in HPA axis activity and in immune functions.[3-5,8,9]

Despite the fluctuating operational criteria and the current lack of definitive biomarkers for subthreshold depressive states, it is clear that the various subthreshold depressive conditions have a high prevalence in the community and are all associated with psychosocial disability, poor quality of life, and high healthcare costs that increase incrementally in parallel with increases in symptom severity.[1,6,7] Moreover, individuals with any of the subtypes of subthreshold depression are more likely to experience full-criteria episodes of MDD and to progress into a persistent, fluctuating depressive condition. Thus, research advances in understanding the neurobiology of subthreshold depression and the related development of evidence-based psychosocial and pharmacological interventions for these conditions could substantially decrease the morbidity associated with subthreshold depressive symptoms that, in spite of their high prevalence and impact, are often overlooked, underdiagnosed, and undertreated.

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