When does depression become a mental disorder?

Mario Maj

Summary
How can we differentiate a depressive disorder from ‘normal’ sadness? This editorial summarises three approaches: the first emphasises the context in which depressive symptoms occur; the second postulates a qualitative difference between the two conditions; and the third argues that the distinction should be based on pragmatic grounds.

Major depressive disorder is reported to be the most common mental disorder, with a lifetime prevalence in the community as high as 17% in the USA1 and even higher in some other countries.

These rates are frequently quoted in the literature, but are viewed by many, both outside and within the psychiatric field, with some degree of scepticism. In particular, it has been argued that ‘the DSM definition of major depressive disorder fails to exclude intense sadness arising from the way human beings naturally respond to major losses;’2 ‘normal’ sadness may be, therefore, treated as if it were a depressive disorder, which ‘may undermine normal recovery by disrupting normal coping processes and use of informal support networks;’2 This argument is likely to be increasingly endorsed by public opinion in the years to come. It is therefore crucial to articulate a convincing response to the question ‘When does depression become a mental disorder?’ In this editorial, three approaches regarding this issue are summarised.

The contextual approach

This approach argues that depression, contrary to normal sadness, is either unrelated to a life event or disproportionate to the preceding event in intensity, duration and degree of the functional impairment it produces. This argument is certainly appealing to many clinicians and lay people, but has some important weaknesses.

First, the presence itself of a depressive state may affect the individual’s accuracy in reporting life events. Many patients with depression try to find a meaning in their depressive state, by attributing significance to events which are in themselves neutral. Indeed, the experimental induction of depressed mood leads to a significant increase in reports of recent stressful events.3

Second, the presence itself of a depressive state may expose a person to adverse life events. In fact, the relationship between depression and ‘dependent’ events (i.e. events which can be interpreted as a consequence of the depressive state, such as being fired from a job or being left by the partner) is stronger than the relationship between depression and other events.

Third, what is a ‘proportionate’ response to a given event remains to be clarified: even when exposed to the most extreme adverse event, the majority of people do not develop a depressive state. Fourth, whether a life event has been really decisive in triggering a depressive state may be difficult to establish in several cases, and in any case will require a subjective judgement by the clinician, with a high risk for a low reliability. Finally, currently available research evidence suggests that response to antidepressant treatment in major depressive disorder is not related to whether or not the depressive state was preceded by a life event.

In a clinical reality in which the majority of people fulfilling the current diagnostic criteria for major depression report their state to have been triggered by a life event, it would be unwise to disallow the diagnosis on the basis of a ‘contextual’ criterion of doubtful reliability and clinical utility.

The qualitative approach

This approach, endorsed by several European psychopathologists, assumes that there is always a qualitative difference between ‘true’ depression and ‘normal’ sadness. It is argued that this difference has been lost in the recent process of oversimplification of psychopathology related to the development of operational diagnostic criteria. The oversimplification has allegedly occurred at two levels: at the level of individual phenomena, where the need to distinguish psychopathological symptoms from other expressions of impaired mental well-being has been de-emphasised; and at the level of syndromal description, where the fact that there is a gestalt of the depressive syndrome, beyond the sum of depressive symptoms, has been ignored.4

The nature of this postulated qualitative difference between depression and ‘normal’ sadness is usually not specified. However, it has been explored in several studies. For example, in a study in which patients with depression were asked to describe in their own words their current state, ‘the commonest description was of a sense of detachment from the environment’; ‘the next most common description was of a specific inability to envisage the future’; ‘the next most common descriptor was of physical changes that were described in terms of feeling that the subject was coming down with a viral illness, either influenza or glandular fever, along with descriptions of aches and pains and, in particular, headaches or numbness of the head or tight bands.
around the head.5 These descriptions appear different from those that people who are simply sad would offer spontaneously.2

It has been also argued that a person with depression has lost the ability to experience pleasure generally, whereas a person who is just demoralised is still able to experience pleasure when distracted from demoralising thoughts. Furthermore, the demoralised person feels inhibited in action by not knowing what to do, feeling helpless and incompetent, while the person with depression has lost motivation and drive, and is unable to act even when an appropriate direction of action is known.6

The fact is, however, that taxonic research on depression, based on Meehl’s taxometric methods or latent class analysis, has failed to support the idea that a latent qualitative difference exists between major depression and ordinary sadness, arguing instead for a continuum of depressive states, with the possible exception of a subtype, grossly corresponding to DSM-IV major depression with melancholia, which may be qualitatively different. Further research is certainly needed to explore the nature of the subjective experience of people with depression, and its differences with respect to the perception of ordinary sadness. A more precise characterisation of the individual depressive symptoms is needed, as well as an exploration of the predictive value of each symptom, and of clusters of symptoms. Further studies on the validity and clinical utility of the construct of melancholia are also warranted. For the time being, however, it would be hard to maintain that the difference between depression and ordinary sadness is always a qualitative one.

The pragmatic approach

This approach assumes that, since there is a continuum of severity and pervasiveness from ordinary sadness to clinical depression, the boundary has to be fixed on pragmatic grounds (i.e. giving priority to clinical utility). This is what the DSM-IV actually tries to achieve, regarding depression as a ‘disorder’ when it reaches a given threshold in terms of severity, duration and degree of suffering or functional impairment, thus deserving clinical attention.

The problem is, however, that the threshold fixed by the DSM-IV for the diagnosis of major depression is not based on solid pragmatic grounds. The duration and impairment criteria have never been validated, and the requested number of depressive symptoms (at least five) does not have acceptable empirical support. Indeed, a monotonic increase has been found in the number of episodes, impairment and comorbidity as we go from people with two to four depressive symptoms to those with five or six symptoms, to those with seven to nine symptoms.7 When a point of rarity has been reported, it has usually corresponded to a threshold higher than that fixed by the DSM-IV. For example, the risk for future depressive episodes was found to be substantially greater in individuals with seven or more depressive symptoms than in those with six symptoms.8 Furthermore, among patients with a diagnosis of major depressive disorder, those with a score of <20 on the Hamilton Scale for Depression (who made up more than 60% of the sample) did not recover more frequently with imipramine than with placebo plus clinical management, suggesting that the DSM-IV threshold may be too low.9 On the other hand, the psychosocial impairment associated with the presence of two to four depressive symptoms has been repeatedly reported to be comparable to that associated with the presence of five or more symptoms,10 arguing for the existence of depressive states below the DSM-IV threshold which require clinical attention.

These findings seem to indicate that more than one threshold may be needed in the characterisation of depressive states, in order to maximise clinical utility. The threshold for a depressive state deserving clinical attention may be lower than that fixed by the DSM-IV, but the threshold for a depressive state requiring pharmacological treatment is likely to be higher. These thresholds may need to be based on the overall severity of depressive symptoms rather than, or in addition to, their number.

Conclusions

Of the three approaches considered, the first two, which are probably more appealing to several clinicians and lay people, are not supported by currently available research evidence, whereas the third has some empirical support. An analogy seems to emerge between depression and common physical diseases such as hypertension and diabetes, which also occur on a continuum, with at least two identifiable thresholds: one for a condition deserving clinical attention and another for a state requiring pharmacological treatment. The introduction in the DSM-5 of a dimension of severity, with clear anchor points, may help to address this reality, but the current threshold for ‘caseness’ may still need to be reconsidered.

References

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