

Sadness or sickness?



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A new book provides another challenge to the current thinking on depression.

IN the last year, one book has rattled the ranks of the psychiatric profession at an unprecedented level. In this book, *The Loss of Sadness*, sociologist Allan Horwitz and social worker Jerome Wakefield argue that psychiatry has progressively pathologised sadness.

They observe that to be human is to react with sadness to a range of negative life events. By contrast, they argue that clinical depression is a state that appears without apparent cause or is grossly disproportional to any preceding stressor.

Their central concern is that contemporary psychiatry has confused normal sadness with clinical depression by ignoring the relationship of symptoms to the context in which they emerge. As noted in the foreword by Robert Spitzer (architect of the DSM-III approach to classification), major depression was “based on the assumption that symptoms alone can indicate that there is a disorder”.

SADNESS EXPLORED

The authors open with an intriguing question concerning the central character in Arthur Miller’s play, *Death of a Salesman*. Is Willy Loman (low man?) weighed down by failed hopes and aspirations in his life or does he suffer a psychiatric illness?

They reveal that when the script was sent to two psychiatrists, Loman was classified as having a depressive disorder. Predictably, Miller objected to this characterisation.

Essentially, the authors argue that the distinction between sadness and clinical depression lies in the context. They suggest that sadness has three necessary features: first, specific environmental triggers (principally losses); second, a response that is roughly proportional in intensity

to the experienced loss; and third, that it dissipates when the loss is accommodated, neutralised or ends.

DEPRESSION DEFINED

The authors argue that depression has gained “an iconic status in both the contemporary mental health profession and the culture at large”, with experts claiming that it is a “dire public health problem that affects a large proportion of the population”.

While conceding the reality of depressive disorder, they argue that “an inadequate conceptual distinction between disorder and non-disorder is a crucial weakness in the entire clinical and research industry”.

Finally, they claim that all discussions about depression ignore “the critical question of when depressive symptoms indicate a mental disorder and when they are disordered responses to loss”.

They review historical definitions of depression and the diagnostic systems introduced in the last few decades and conclude that the “massive pathologisation of normal sadness” has made “depressive diagnosis less rather than more scientifically valid”.

They criticise progressive dimensionalising of depressive disorders into minor and sub-syndromal conditions and the consequent “article of faith” that early treatment of mild disorders in community settings will prevent conditions emerging, and they offer a brilliant criticism of the fallacies behind the movement to accord clinical status to minor depression.

They are especially critical of screening instruments – particularly in risking over-diagnosis – and detail consequences of viewing both sadness and depressive disorders as equivalent conditions in terms of treatment.

CHALLENGES

In effect, the authors rework the old binary model of depression which contrasted endogenous and reactive depressive conditions.

They then make two assumptions that are worthy of challenge. First, they argue that states of sadness and melancholia comprise the “same sorts of symptoms”. However, while patients with melancholia are highly likely to report profound anergia, psychomotor retardation or agitation, impaired concentration and a non-reactive and anhedonic mood, those

with non-melancholic disorders are more likely to report a reactive mood, irritability and, often, anger.

Second, the authors’ emphasis on context is problematic. While the older term for melancholia was ‘endogenous’ – suggesting that causes lay within – multiple studies have shown that those with melancholic depression are almost as likely to report stressors as those with non-melancholic disorders.

The relationship of stressors to melancholia is complex. While significant antecedent stressors are often found for the first melancholic episode, once an individual has developed the condition, episodes tend to occur more autonomously.

The authors’ emphasis on the depressive reaction being disproportionate to the stressor is also clinically appealing but is not supported by research. The authors’ suggestion that clinical depression and states of sadness can therefore be distinguished by respective absence or presence of the cause is both simplistic and problematic.

However, aside from such caveats, Horowitz and Wakefield have delivered a refreshing work that rebalances the clinical scales. 