

Two Sides of Depression: Medical & Social

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Two models have dominated portrayals of depression. The medical model views depression as a disease that has distinct symptoms with predictable courses and outcomes. It typically relies on brain-related explanations and responses, although many adherents also use social and psychological causes and treatments. A second model conceives of depression as the result of external stressors, loss events, and other problems of living that naturally subsides when these conditions improve. In this view, optimal responses lie in addressing the social conditions that underlie depressed states. In this essay, we examine how each edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) since DSM-III in 1980 has blurred the medical and social approaches and conceived of all sorts of depressive symptoms as needing medicinal responses. Although the distinction between the social and medical types is often difficult to make, it is an essential first step in developing accurate conceptions of the two sides of depression.

The question of whether it is most useful to treat depression as a medical or as a social problem has generated considerable discussion. On one side, most psychiatrists and many patient advocates view depression as a disease that has distinct symptoms with predictable courses and outcomes. They typically search for brain-related causes and apply somatic treatments to this condition. A variant of this approach, typically called biopsychosocial, sees some combination of biological, psychological, and social factors as responsible for depression and its treatment, but usually holds that medical diagnosis of depression is warranted.¹ On the other side, many social scientists and critics of psychiatry see depression as resulting from external stressors, loss events, and other problems of living, and as subsiding when these conditions improve. In their view, optimal responses lie in addressing the social conditions they believe lead to depressed states.

In this essay, we focus on how the criteria for major depressive disorder (MDD) in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* since its third edition (DSM-III) in 1980 abandoned the well-established distinction between medical and social depressions.² Consequently,

social forms of depression have been seen as medical disorders, resulting in mistaken views of its prevalence, etiology, and treatment.

The medical view of depression dominates public discourse. According to this approach, we are in the midst of a tidal wave of depressive disorder that should be addressed with an equally forceful medical response. In contrast, just a few decades ago, prior to the use of current diagnostic criteria, depressive disorder was considered a serious but relatively uncommon disorder, affecting perhaps 2–3 percent of the population over a lifetime.³ This situation drastically changed after 1980 when DSM-III inaugurated symptom-based criteria for MDD in order to improve the reliability of psychiatric diagnosis. This meant abandoning traditional standards for validity that required more subjective judgments, such as “with or without cause” or “unexpected duration” that had previously separated medical from social forms of depression.⁴ The unintended result was to combine situational responses to external losses with long-standing individual dysfunctions without distinguishing these two very different conditions.

Population surveys using DSM measures soon found that huge proportions of people met MDD criteria. The major survey of mental disorder in the United States conducted after 1980 indicated that over 20 percent of community members had suffered from MDD.⁵ MDD’s Janus-faced nature allowed researchers to downplay its severity when explaining how it could afflict such a substantial portion of the population. For this purpose, it was the common cold of psychiatry. Yet when it was advantageous to emphasize its devastation, depression was labeled a major scourge of humankind. The World Health Organization (WHO) declared depression to be the world’s most disabling condition after it combined the large group of people who met the MDD criteria and assumed their severity was comparable to paraplegia or blindness!⁶ While this might be justified for the relatively small number of serious cases of depression, the same can hardly be said for someone who was sad, fatigued, unable to concentrate, and had sleep and appetite problems for two weeks after facing a major loss event. Yet this situation was favorable for many groups with interests in promoting the widespread and severe nature of depression: practitioners, professional organizations, government agencies concerned with mental health, the WHO, drug companies, and patient advocacy groups, among others.

Remarkably, those initial studies *underestimated* DSM-defined depression rates because they relied on people’s recollections of depressive symptoms from years before. Current estimates based on more methodologically sophisticated techniques that repeatedly interview individuals over time suggest that half or more of the entire population suffers from depressive disorder at some point in life.⁷ A corresponding result is that a substantial proportion of the population now takes antidepressant medications. For example, from 2017 through 2018, about 14 per-

cent of all U.S. adults and 19 percent of all women used antidepressants within the past thirty days.⁸ This is despite the facts that the average effects of antidepressant medication over a placebo are minimal and that the potential negative side effects are considerable.⁹

Against this medicalized approach, we will argue for a more nuanced view that is empirically better supported, conceptually more defensible, and more beneficial for patients and society than either pole of the medical-social dispute: both sides are partly right. There are genuine medical disorders of depressive emotion, as there are of almost any biological system. However, there are also much more frequent, expectable, depressive reactions to perceived social conditions that trigger depressive feelings. Correspondingly, some depressive conditions are best approached and treated as medical conditions while others are better addressed as social, nonmedical problems. We will focus on how inadequately addressing the medical-social distinction has led to the massive medicalization, misdiagnosis, and pharmacological treatment of what are often externally triggered normal-range depressive responses that are best addressed through social interventions. Accordingly, because a disorder diagnosis presumes that something has gone wrong within the individual, little research addresses the social dynamics of depressive feelings and the way that social conditions might be altered to minimize them.

Importantly, we do *not* argue that people with normal-range, socially embedded sadness should not be able to get professional help and support. The U.S. medical insurance system often requires “medical necessity” for treatment to qualify for reimbursement, and thus pushes psychiatry to draw the boundaries of medical disorder as broadly as possible. The problem is that this approach influences the type of help that is offered based on incorrect attributions of medical disorder. Rather than falsely categorizing people as having depressive disorders, mental health treatment systems should be revised to allow appropriate support of and research into both social and medical sources of depression.

The emotions of sadness, grief, and depression – including feelings that can be quite intense – are often normal biologically designed features of human psychological functioning, and not inherent disorders. Evidence for this view includes the prevalence of such feelings in response to suitable triggers in our society. These responses persist across cultures (although the events that set off such responses vary enormously due to different cultural meanings), in nonhuman primates (a point observed from facial expression and behavior by Darwin, and confirmed by modern hormonal and behavioral studies), in many other animals (as in recent stories in the popular press about lengthy grief in elephants and whales), and even in human infants prior to socialization when they are separated from their attachment figures.¹⁰

Whether depressive emotions are normal depends on the situation. Four specific qualities indicate that depressive reactions, like other emotions, are evolutionarily designed responses to particular circumstances.¹¹ First, these reactions are highly context-specific, emerging in response to losses and other stressors, including loss of relationships, status, resources, and meaning. This fits the evolutionary understanding that each emotion is biologically designed as an adaptive response to a particular kind of challenge and is thus triggered by specific types of events. Like many other clearly biologically designed features – for example, sleep – the adaptive purpose of sadness, grief, and depressive feelings remains disputed. Various explanations include disengagement from valued goals that have become hopeless, withdrawal when a loss of status or resources places us in danger if we continue in the fray, as well as warnings that things are not going well and need our attention and signs that we need to devote our mental processing toward rumination on complex problems in our social relationships.¹² Whatever the precise answer, for better or worse, sadness and grief are part of our natural humanity.¹³

The second indication that depressive reactions are evolutionarily designed is that the symptomatic intensity of the emotional response is roughly proportional to the magnitude of the loss that triggers it, subject to individual and cultural variability. From an evolutionary point of view, the greater the adaptive challenge, the greater the strength of the emotion that might be warranted. The third indication is that once triggered, symptoms persist in accordance with external contexts, but then naturally remit when the context changes for the better or as people reconstruct their lives and meaning-systems to adapt to their losses. Deviating from emotional neutrality has biological costs, and once the adaptive challenge subsides, so do emotions. And the fourth indication: for an emotional response to effectively deal with environmental challenges, many different physical and psychological mechanisms must be coordinated. The remarkable orchestration of psychological, physiological, and behavioral variables that occurs in emotional experiences implies the evolution of emotions as superordinate programs that coordinate multiple mechanisms.¹⁴

In contrast to normal depressive reactions, depressive disorders lack at least one basic quality of designed reactions, and thus qualify as true medical disorders. Typically, such failures involve reactions that are too intense or lengthy given the triggering context. Normal emotional reactions vary greatly both individually and culturally, so, given our ignorance of emotional mechanisms, in practice there will be fuzzy boundaries between medical and social types of depression. Nonetheless, the distinction still determines a range of clear cases on either side of the medical-social boundary, just like other useful distinctions with fuzzy boundaries (for example, night/day, child/adult, orange/red). However, this fuzziness also means there will be ample room for disagreement and controversy.¹⁵

Of course, emotions are often unwanted or distressing without being disordered. Given that our environment is so different from the environment in which the human species evolved, there will be mismatches between the way we have evolved to react and the transformed social environment in which we find ourselves. Such mismatched reactions can be normal but no longer useful, and we may want to treat them while recognizing that no medical disorder is present.¹⁶

We reject the common idea that when depressive feelings are reactive to some situation rather than unprovoked, they are normal. Social triggers can cause both normal and disordered depressions. The majority of cases of both kinds start with a stressor, so very few depressions are completely out of the blue. Most cases described since antiquity arise after hearing news of the death of a loved one or some other major loss. So, the medical or social distinction must lie elsewhere.

Instead, a crucial difference between medical and social depressions is whether symptoms respond to changing external conditions, as they are biologically designed to do. For example, cases that develop after people have lost jobs or romantic relationships should remit when they enter new jobs or new involvements. In other cases, such as grief reactions, symptoms should gradually dissipate with the passage of time and the construction of new meaning-systems. In contrast, depressive disorders are unresponsive to positive changes in the initiating circumstances and persist over long time periods regardless of the social environment.

There is a long history of medical recognition and treatment of depressive disorder, known in antiquity as “melancholia” or “black bile disease” after the most popular theory of its cause.¹⁷ Classic medical texts also emphasized the distinction between melancholia and normal-range but symptomatically similar conditions, citing famous clinical cases in which depression due to stressful situations, such as unrequited love, were diagnostically distinguished from melancholia.

The modern concept of depressive disorder emerged most directly from the work of psychiatrist Emil Kraepelin (1856–1926), who was so influential on recent thinking that the present diagnostic system is commonly referred to as “neo-Kraepelinian.”¹⁸ His approach reflects an understanding of normal social sadness and medically disordered depression as described above. Like his medical predecessors since ancient times, Kraepelin believed in the necessity of taking context into account when diagnosing depressive disorder and differentiating it from normal sadness: “Morbid emotions are distinguished from healthy emotions chiefly through the lack of a sufficient cause, as well as by their intensity and persistence. . . . Again, morbid emotions sometimes attach themselves to some certain external occasions, but they do not vanish with the cause like normal feelings, and they acquire a certain independence.”¹⁹ Eminent twentieth-century psychopharmacologist Donald Klein echoed Kraepelin’s last point, that a key

distinction between medical and social depressions is whether the condition responds to changing conditions: “Once the episode is underway, it is autonomous, that is unresponsive to changes in the initiating circumstances. If the patient with a depressive episode [as opposed to a depressive reaction] regains his job the illness continues.”²⁰

Kraepelin offered illustrations that emphasize the ambiguities diagnosticians face and the need for them to consider social context in discriminating disorder from normality:

Several times patients have been brought to me, whose deep dejection, poverty of expression, and anxious tension tempt to the assumption of a circular [pathological] depression, while it came out afterwards, that they were cases of moodiness, which had for their cause serious delinquencies and threatened legal proceedings. As the slighter depressions of manic depressive insanity, as far as we are able to make a survey, may wholly resemble the well founded moodiness of health, with the essential difference that they arise without occasion, it will sometimes not be possible straightway to arrive at a correct interpretation without knowledge of the previous history in cases of the kind mentioned.²¹

Kraepelin was also careful to report how initial intuitions of social causes of a depressive condition can turn out to be misleading: “In another case the sale of a property, which was regarded as the cause of a depression, was successfully made null and void, but without any influence on the disease; later on there were further manic and depressive attacks.”²² Kraepelin approached the diagnosis of each case of depression as a matter of testing which hypothesis, a normal emotion or an emotional disorder, best explained the patient’s suffering.

Sigmund Freud, Kraepelin’s main contemporary rival, agreed with Kraepelin on this point. Although he recognized that grieving could become pathological, in his essay “Mourning and Melancholia,” Freud strongly endorsed the normality of intense grief:

Although grief involves grave departures from the normal attitude to life, it never occurs to us to regard it as a morbid condition and hand the mourner over to medical treatment. We rest assured that after a lapse of time it will be overcome, and we look upon any interference with it as inadvisable or even harmful.²³

Freud emphasized that grief is not a medical disorder that represents the breakdown of a biologically normal response. Thus, it does not require medical treatment. Indeed, Freud indicated that it would never occur to us to provide medical treatment to the bereaved. In addition, he stressed that grief is naturally self-healing, so that with time, the mourner would return to a normal psychological state. Medical intervention, he suggested, could harm the grieving person through interfering with this natural process.

The current high prevalence rates of depressive disorder are not surprising once one understands the criteria being used for the identification of cases. DSM diagnosis of MDD requires the presence for at least a two-week period of symptoms from five (or more) of the following symptom groups, with at least one of the symptoms being either depressed mood or loss of interest or pleasure in usual activities: 1) depressed mood, 2) diminished interest or pleasure in activities, 3) weight gain or loss or change in appetite, 4) insomnia or hypersomnia (excessive sleep), 5) psychomotor agitation or retardation (slowing down), 6) fatigue or loss of energy, 7) feelings of worthlessness or excessive guilt, 8) diminished ability to think or concentrate or indecisiveness, and 9) recurrent thoughts of death or suicidal ideation or suicide attempts. Additionally, the symptoms must cause distress or social role impairment – but this added requirement has been shown to be redundant because if one has five of the symptoms, one is sure to experience distress or a decrease in role functioning (for example, family, school, occupation).²⁴ Indeed, even those having normal grief or depressive feelings experience significant distress and decrements in role functioning.

These acontextual criteria disregard pre-DSM III understandings of the social circumstances that allow for an inference to depressive disorder: “A depression is judged to be pathological if there is insufficient specific cause for it in the patient’s immediate past, if it lasts too long, or if its symptoms are too severe,” one summary read.²⁵ In contrast, the DSM criteria ignore the context in which symptoms arise, require just a two-week period of duration, and do not mandate the presence of any especially severe symptoms. The result is to conflate depressive symptoms that are normal, proportionate responses to situations of loss with depressive disorders.

The original MDD criteria did recognize the importance of context in a single instance known as the “bereavement exclusion.” They did not diagnose depressive symptoms arising from bereavement if they were not unduly severe or prolonged. This exclusion resulted from DSM Task Force member Paula Clayton’s findings that normal grief often met the proposed criteria for depressive disorder, but naturally resolved over relatively short periods of time. Bereavement, however, was the sole exclusion: symptoms resulting from other kinds of loss were not proscribed. Yet Clayton had proposed that bereavement might be a model for other losses: “We believe that a specific, carefully defined model for a reactive depression can be delineated. . . . It is conceivable that there are situations other than bereavement caused by financial problems, problems with children, the death of a President, etc. which precipitate a similar reaction with similar mild symptoms and course.”²⁶

Rather than following up Clayton’s proposal for an expanded exclusion, the DSM went in the other direction. DSM-5 eliminated the bereavement exclusion so that depressive feelings meeting MDD criteria during grief are categorized as

depressive disorders: “The DSM-5 Mood Disorders Work-group has recommended the elimination of the bereavement exclusion criteria from major depressive episodes in light of evidence that ‘the similarities between bereavement related depression and depression related to other stressful life events substantially outweigh their differences.’”²⁷ This rationale begged the crucial question of whether other mild depressive reactions to stress (caused by losses and stressors other than bereavement) were different enough from other MDD conditions to suggest they are normal emotional responses to social events.

In response to intense criticism over eliminating the bereavement exclusion, the editors of DSM-5 added a note acknowledging that its criteria may invalidly diagnose normal-range social reactions as medical disorders: “Responses to a significant loss (e.g. bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in [the symptom criteria], which may resemble a depressive episode.”²⁸ The note advises the clinician to judge this issue using “clinical judgment based on the individual’s history and the cultural norms for the expression of distress in the context of loss.”²⁹ The problem is that the note contains no measures and is not part of the formal MDD diagnostic criteria; thus, it has no impact on scientific research and likely little effect on clinical diagnosis. That the DSM acknowledges that its criteria misclassify some normal-range social depressions as mental disorders is a useful starting point. The puzzle is why neither the DSM nor psychiatry more generally refuses to see this as the serious medical, scientific, social, and ethical issue that it is.

The problems with the MDD criteria can be traced to their origins. They emerged from studies aimed at formulating criteria to distinguish depressive disorders from physical medical problems in hospital settings.³⁰ However, the criteria were not designed for their current function of distinguishing depressive disorder from normal intense distress and grief in the community, and they fail to do so.

MDD symptom criteria are invalid for two basic reasons. First, they do not consider the context of the symptoms. Thus, one cannot judge whether an emotional response is more likely a normal-range proportional response to circumstances or an expression of an emotional disorder. Second, the DSM criteria include many symptoms that also occur in normal distress, from sadness, moderate role impairment, and insomnia to decreased appetite, difficulty concentrating, and fatigue. Diagnosis requires any five symptoms, and this threshold can be reached by symptoms that are all signs of normal distress and thus indicate a normal-range response. The five-symptom threshold thus fails to perform its basic function of validly indicating disorder.³¹

Yet another problem with the DSM criteria lies in the lifetime trajectories of people who qualify as disordered. When Kraepelin developed the notion of depressive disorder, the single feature that most convinced him that he was justified in attributing medical disorder was the actuality or expectation of an eventual recurrence. The research literature regularly describes depression as recurrent as a rationale for its being pathological. Eminent psychiatric researcher Kenneth Kendler explains: “For Kraepelin, the ‘construct’ of . . . manic-depressive insanity assumed a relapsing disorder without deterioration” and thus “course and outcome would be the most important validators.”³² Recurrence is interpreted as evidence of an ongoing internal dysfunction that disposes the individual to new episodes so recovery from depression is interpreted as “recovery from the episode, not from the illness per se.”³³ Consequently, treatment should focus on preventing recurrence, often by extending services beyond recovery.

However, recent analyses of the literature reveal that over half – likely approaching 60 percent – of all depressive episodes are the only ones that the individual experiences during lengthy follow-up periods.³⁴ That means that most cases of what is diagnosed as depressive disorder do not satisfy the crucial criterion, recurrence, that persuaded Kraepelin to consider this condition a mental disorder, and that current researchers cite as justifying its pathological status.

As a result, many MDD diagnoses are questionable as medical pathologies. For example, a recent national epidemiological survey found that about 13 percent of individuals diagnosed with MDD had their depressive episodes only after the deaths of loved ones and these episodes lasted less than two months.³⁵ There is no rationale for diagnosing such individuals on that basis alone as having a mental disorder as opposed to a natural reaction to a social loss. And that figure stems from just acute grief and ignores transient normal-range reactions to other stressors. Note that some of the grief cases had more than one episode because the subject lost more than one close person. This indicates that even recurrence must be looked at carefully and not taken mechanically as an indicator of disorder because many people react with normal-range distress to repeated losses.

Much of the data that is most relevant to evaluating whether current MDD criteria validly distinguish social from medical depressive episodes were generated during the debate over the bereavement exclusion. However, these results transcend that specific debate. According to the bereavement exclusion, depressive symptoms during bereavement are considered normal-range and only qualify as MDD if “after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.”³⁶ These criteria – that is, having any one or more of the six specified more severe symptoms – were used to define

what became known in the literature as “complicated” depressive episodes, as opposed to “uncomplicated” episodes that did not have any of the six specified severe features. Note that instead of the specified duration threshold of longer than two months, the studies described below often used the more demanding longer than six months as the duration criterion for complicated depression on the assumption that many normal episodes of distress can last beyond two months.

Initially, studies examined whether uncomplicated depression during bereavement and reactions to other losses are similar or different. We conducted the first major study, along with fellow psychiatrists Michael First and Mark Schmitz, to examine whether depressive reactions to other stressors – such as loss of a valued job, marital dissolution, financial ruin, loss of possessions in a natural disaster, and negative medical diagnoses in oneself or a loved one – also could be divided into the same pattern of milder uncomplicated responses to social losses and more severe complicated and possibly disordered responses.³⁷ We found that all kinds of loss-triggered episodes of depression that were not especially severe or prolonged, and met the six requirements for being “uncomplicated,” had similar symptoms, durations, treatment histories, and degree of impairment as bereavement and looked very different from complicated depressions. Several follow-up studies confirmed these results and suggested that all uncomplicated conditions formed one homogeneous category. However, they did not conclusively resolve which kind of category they found: mild depressive disorder or normal-range nonmedical depression.

The debate over possible problems with DSM depression criteria was transformed when studies used longitudinal data to evaluate the crucial feature of “predictive validity”: whether later outcomes after a depressive episode confirm that it was likely a medical disorder. The most characteristic feature of depressive disorder – indeed, for many diagnostic theorists from Kraepelin onward, *the* defining feature – is recurrence of depression over time.

Ramin Mojtabai was the first researcher to take a predictive-validity approach to bereavement-related depression. Mojtabai used the two-wave National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), a survey of a nationally representative U.S. community sample that followed respondents three years after the initial interview. He divided the sample into five groups: those with a history of 1) a single uncomplicated bereavement-related depressive episode, 2) a single uncomplicated bereavement-unrelated episode, 3) a single longer depressive episode, 4) recurrent depressive episodes; as well as 5) those with no life history of depression. He then compared these groups for their experiences of depression during the three-year follow-up period between wave one and wave two. His most striking finding was that participants who at the initial interview had experienced a single lifetime uncomplicated bereavement-related depression were *not* significantly more likely to experience a depressive episode during the

follow-up period than those who had no lifetime history of depressive episodes (4.3 percent versus 7.5 percent, respectively). In contrast, all the other categories of depression history at baseline had significantly higher three-year recurrence rates, ranging from 14.7 percent to 27.2 percent. Mojtabai concluded that his findings supported the validity of the bereavement exclusion.³⁸

To demonstrate that Mojtabai's remarkable findings were no fluke, Wakefield and Mark Schmitz replicated his analysis using a different dataset: the Epidemiological Catchment Area (ECA) study, which included a one-year follow-up period.³⁹ The same findings emerged. The recurrence rate in the uncomplicated depression group (3.7 percent) was not significantly different from the rate for the group with no history of depression (1.7 percent), and both were significantly and substantially lower than the other depression groups studied (14.4 percent and 16.2 percent). This was powerful evidence that eliminating the bereavement exclusion leads to misdiagnosing normal-range depressive feelings as depressive disorders.

Recall that our earlier study had established that other social-triggered uncomplicated depressive episodes are generally similar to uncomplicated bereavement-related episodes – so much so that they seem to form one uniform category – and are quite dissimilar to complicated episodes. This suggests that independent of issues concerning bereavement, the DSM criteria misdiagnosed intense social depressive reactions as medical disorders. Consequently, the studies of predictive validity were expanded beyond bereavement-related depression to examine all stress-triggered depressive episodes. The question became not just whether the bereavement exclusion itself is valid and should be retained, but whether the validity of DSM criteria requires that the exclusion be extended to all major social stressors.

In their studies using both the NESARC and ECA datasets, Wakefield and Schmitz addressed this broader question. They found that the results related to bereavement strongly generalize to all stressors. The NESARC data could examine the three most distinctive and problematic known outcomes of depressive disorder: recurrence, suicide attempt, and anxiety disorder. The results were that “for all validators, 3-year rates for single episode uncomplicated cases were not significantly different from no-MDD-history rates, but significantly lower” than the rates for the other groups studied.⁴⁰ Moreover, “mild” depression defined according to the APA's standards in terms of number of symptoms did not yield the same results. The quality of the uncomplicated symptoms mattered. For technical reasons, the NESARC study was limited to single-episode uncomplicated cases, but the ECA study indicated that a multiple-episode history of uncomplicated episodes does not significantly predict higher recurrence of depression than single-episode cases (3.7 percent versus 3.0 percent, respectively), which makes sense if these are basically normal-range reactions that do not indicate dysfunctions of emotional mechanisms. In sum, studies by Wakefield and his collaborators sup-

port the conclusion that people who develop uncomplicated depressions (that is, those with no prolonged duration or any especially serious symptoms), both during bereavement and after all kinds of losses and stressors, are more similar in outcome to those who were *never depressed* than those who had complicated depressive conditions (in other words, with either extended duration or at least one very severe symptom or both), which strongly suggests that most uncomplicated depressions are social conditions, rather than medical. These studies provide the most conclusive evidence we have so far that current DSM criteria misdiagnose social depression as a medical disorder.

These findings can be extended to further subsets of DSM-defined depressive disorders, but that research remains to be done. Meanwhile, by ignoring these results, psychiatry overlooks information that could allow some patients to avoid medication or go off medication sooner due to a lack of any raised likelihood of recurrence or other negative consequences given their symptom profile. In addition, it privileges medical over social responses, such as participation in self-help and support groups, referrals that enhance social resources, educational and relationship counseling, engagements with clergy and other spiritual advisors, and diet and exercise programs.⁴¹ As a result, resources are misallocated and individuals inappropriately treated. This situation is especially true in non-Western societies that are less likely than Western cultures to medicalize depressive conditions and more likely to employ group modes of treatment.⁴²

Some defenders of the medical approach argue that physicians diagnose a heart attack irrespective of whether its causal factors include poor diet or smoking.⁴³ But heart attacks are clear failures of cardiac functioning, whatever their cause. Unlike heart attacks, depressive feelings during grief and in response to other losses are naturally designed emotions that are generally self-limiting – but like all natural systems, they can go wrong. The diagnostician has the responsibility of distinguishing normal reactions from dysfunctions. A more appropriate cardiac analogy is that physicians do not diagnose rapid heart rate as an arrhythmia if it only occurs when the individual is vigorously exercising and stays within normal-range bounds for such cardiac adjustments to physiological demands. To routinely diagnose depressive episodes in reaction to stressful situations as depressive disorders makes no better sense than diagnosing increases in heart rate during exercise as heart disorders. Yet this is what American psychiatry has insisted on doing, resolutely confusing social and medical depressive conditions and muddying research, treatment, and epidemiology as a result.

DSM-5 perpetuates psychiatry's refusal since 1980 to take seriously the problem of distinguishing normal emotions from psychiatric disorders. Its criteria for depression do not separate medical disorders from natural responses to loss. The result of mixing normally distressed individuals with tru-

ly disordered ones is to preclude research from establishing the etiology, course, treatment effectiveness, and possible biomarkers of depression. Far more research is needed that explores questions such as when natural responses to social losses become medical disorders, the reasons for the high variability of depression rates across cultures, and the relative effectiveness of medical and social responses to depression. Although the distinction between social and medical forms of depression is often difficult to make, it is an essential first step in developing accurate conceptions of the two sides of depression.

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ENDNOTES

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