The Loss of Sadness

How Psychiatry Transformed Normal Sorrow
Into Depressive Disorder

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The Concept of Depression

The poet W. H. Auden famously deemed the period after World War II the "age of anxiety."[1] For Auden, the intense anxiety of that era was a normal human response to extraordinary circumstances, such as the devastation of modern warfare, the horrors of the concentration camps, the development of nuclear weapons, and the tensions of the cold war between the United States and the Soviet Union. Were Auden still alive, he might conclude that the era around the turn of the twenty-first century is the "age of depression."[2] There would, however, be a crucial difference between the two characterizations: whereas the age of anxiety was viewed as a natural response to social circumstances that required collective and political solutions, ours is viewed as an age of sadness that is abnormal—an age of depressive psychiatric disorder that requires professional treatment.

Consider Willy Loman, the lead character in Arthur Miller's classic play *Death of a Salesman* and possibly the fictional character most representative of American life during the decades following World War II.[3] As he enters his 60s, despite his fervent belief in the American dream that hard work will lead to success, Willy Loman has never accomplished very much. He has heavy debts, his health is failing, he is barely able to continue working at his job as a traveling salesman, and his sons despise him. When he is finally fired from his job, he is forced to admit to himself that he is a failure. He kills himself in an automobile accident in the hope of getting his family some money from an insurance settlement. The tremendous popularity of *Death of a Salesman* on its introduction on Broadway in 1949 stemmed from Willy Loman's embodiment of the Everyman in American life who embraced the goal of achieving great wealth but found himself destroyed by it.

Death of a Salesman received a very different response during its revival 50 years later.[4] According to a piece in *The New York Times* titled "Get That Man Some Prozac," the director of the revived version sent the script to two psychiatrists, who diagnosed Loman as having a depressive disorder.[5] The playwright, Arthur Miller, objected to this characterization, protesting: "Willy Loman is not

a depressive. . . . He is weighed down by life. There are social reasons for why he is where he is." The response of the psychiatrists is as exemplary of our time as Loman was of his. What our culture once viewed as a reaction to failed hopes and aspirations it now regards as a psychiatric illness. The transformation of Willy Loman from a social to a psychiatric casualty represents a fundamental change in the way we view the nature of sadness.

The Ubiquity of Depression

The ascendancy of depressive disorder is a major social trend manifested in a variety of ways:

Amount of depression in the community. Many researchers claim that substantial and growing proportions of the population suffer from depressive disorder. Estimates from epidemiological studies indicate that Major Depression afflicts about 10% of adults in the United States each year and nearly a fifth of the population at some point in their lives.[6] Rates among women are even higher, about twice as high as in men.[7] Depending on the definition employed, depression can afflict as many as half of the members of some groups, such as female adolescents and the elderly.[8] Moreover, these numbers seem to be steadily growing. For the past several decades, each successive birth cohort has reported more depressive disorders than previous generations showed.[9] Although these rising rates are more likely to be an artifact of the way community surveys measure this condition than to reflect an actual increase,[10] there is a widespread perception that depressive disorder is growing at an alarming pace.

Number of patients in treatment for depression. The number of persons treated for depression in the United States has grown explosively in recent years. Most depressed people are treated in outpatient settings, where treatment of depression increased by 300% between 1987 and 1997.[11] By 1997, fully 40% of all psychotherapy patients, double the percentage of a decade before, had diagnoses of a mood disorder, the larger category that comprises mainly depression.[12] The overall percentage of the population in treatment for depression in a particular year grew from 2.1% in the early 1980s to 3.7% in the early 2000s, an increase of 76% in just 20 years.[13] Some groups experienced a much greater increase; for example, in just the period between 1992 and 1998, health care providers diagnosed 107% more elderly persons with depression.[14]

Prescription of antidepressant medication. Although medication has been a common treatment for life problems since the 1950s, its use has undergone a staggering growth in recent years. Antidepressant medications, such as Prozac, Paxil, Zoloft, and Effexor, are now among the largest selling prescription drugs of any sort.[15] Their use among adults nearly tripled between 1988 and 2000.[16] In any given month, 10% of women and 4% of men now use these drugs.[17] During

the 1990s, spending for antidepressants increased by 600% in the United States, exceeding $7 billion annually by the year 2000.[18]

Estimates of the social cost of depression. Depression is believed to be the source of huge social costs. The World Health Organization (WHO), the leading international body that deals with health, projects that by 2020 depression will become the second leading cause of worldwide disability, behind only heart disease. The WHO estimates that depression is already the leading cause of disability for 15- to 44-year-olds.[19] In the United States, economists estimate that depression is responsible for $43 billion in costs every year.[20]

Scientific publications on depression. Research on depression has become a major industry.[21] In 1966, 703 articles containing the word *depression* in their titles were published in medical journals. In 1980, the year in which the American Psychiatric Association (APA) published its landmark third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*) with new definitions of depressive disorder, 2,754 articles on depression were published. This number steadily increased over the following 15 years, and then exploded in the mid-1990s. By 2005, there were 8,677 articles about depression published, more than 12 times the number in 1966. The number of articles concerned with depression is now far higher than any other psychiatric diagnosis and has grown far more rapidly than the general growth in psychiatric research publications.

Media attention to depression. Depression has become a central concern in the culture more generally. Popular television shows, best-selling books, and major articles in national magazines often feature this illness. Many memoirs about personal experiences of depression, including William Styron's *Darkness Visible,* Kay Jamison's *An Unquiet Mind,* Elizabeth Wurtzel's *Prozac Nation,* and Andrew Solomon's *The Noonday Demon,* have reached the best-seller list. A look at the new books in the psychology sections of bookstores reveals a virtual tidal wave of books on how to prevent or cope with depression of all sorts. The acclaimed television series, *The Sopranos,* features as its central character a Mafia boss who has—among other psychiatric conditions—depression and whose consumption of antidepressant medications is a major theme of the show. A number of prominent public personalities, including Tipper Gore, Mike Wallace, and Brooke Shields, have received massive publicity after disclosing their depressive conditions.

Normal Versus Disordered Sadness

Although the belief that depression is a widespread phenomenon is new, the symptoms we now associate with it, including intense sadness and the many other emotional experiences and physical symptoms that often accompany sadness, have been noted since the beginning of recorded medical history.[22] Yet, in attempting to understand the recent upsurge in diagnosed depressive disorder,

it is important to recognize that until recently, two broad types of conditions that manifest these same symptoms were sharply distinguished from each other. One, normal sadness, or sadness "with cause," was associated with experiences of loss or other painful circumstances that seemed to be the obvious causes of distress. The response to such normal reactions was to offer support, to help the individual cope and move on despite the loss, and to avoid confusing the person's sadness with illness.

The other kind of condition, traditionally known as *melancholia,* or depression "without cause," was a medical disorder distinguished from normal sadness by the fact that the patient's symptoms occurred despite there being no appropriate reason for them in the patient's circumstances. These conditions were relatively rare but tended to be long lasting and recurrent. Because they were not proportional reactions to actual events, such conditions were assumed to stem from some sort of internal defect or dysfunction that required professional attention. Yet these pathological conditions involve the same sorts of symptoms—such as sadness, insomnia, social withdrawal, loss of appetite, lack of interest in usual activities, and so on—associated with intense normal sadness.

This separation of normal sadness and depressive disorder is a sensible and legitimate, indeed a crucial, one. It is consistent not only with the general distinction between normality and disorder used in medicine and traditional psychiatry but also with common sense, and it has both clinical and scientific importance. Yet contemporary psychiatry has come to largely ignore this distinction.

We argue that the recent explosion of putative depressive disorder, in fact, does not stem primarily from a real rise in this condition. Instead, it is largely a product of conflating the two conceptually distinct categories of normal sadness and depressive disorder and thus classifying many instances of normal sadness as mental disorders. The current "epidemic," although the result of many social factors, has been made possible by a changed psychiatric definition of depressive disorder that often allows the classification of sadness as disease, even when it is not.

The "Age of Depression" Results From a Faulty Definition of Depressive Disorder

Unlike Auden's "age of anxiety," which resulted from identifiable social conditions, there are no obvious circumstances that would explain a recent upsurge in depressive disorder. The most commonly heard suggestions—such as that modern life is less socially anchored and involves more alienation or that media constantly expose us to extremes of wealth and beauty that cause us to feel inadequate by comparison—would tend to explain only normal sadness reactions (analogous to the normal anxiety responses to which Auden pointed), not the massive growth of a mental disorder. No environmental pathogen that might

have resulted in a real increase in physiologically, psychologically, or socially induced brain malfunctions has been identified or even theorized. Certainly, progress in effectively treating depressive disorder with psychotropic medication has resulted in increased treatment of a condition about which physicians believe they can at least do some good, and perhaps it has motivated diagnosis of formerly ambiguous cases as depression in the hope of offering effective treatment. But that does not by itself explain the vast growth in the numbers of people who seemingly have and are treated for this disorder; better treatments do not usually lead to a substantial increase in disease prevalence. Nor would improved treatments explain the results of epidemiological studies that bypass patients and directly interview community members not in treatment. Thus the seeming explosion of cases of depressive disorder is puzzling. What has happened to create the appearance of this epidemic?

What has happened, we argue, is largely diagnostic inflation based on a relatively new definition of depressive disorder that is flawed and that, combined with other developments in society, has dramatically expanded the domain of presumed disorder. To understand how this phenomenon has occurred, it is useful to place current psychiatric practices in historical context and to consider how odd current diagnostic definitions of depressive disorder are by historical standards. One must also confront the esoterica of modern psychiatric classification presented in successive editions of the American Psychiatric Association's *DSM.* Often called the "bible of psychiatry," the *DSM* provides diagnostic definitions for all mental disorders.

But how can something as simple and limited as a definition have substantial consequences for such a field as psychiatry and thus for the media that popularize its claims and findings and for the thinking of society at large that relies on its expertise? In response to criticisms during the 1960s and 1970s that different psychiatrists would not diagnose the same person with the same symptoms in the same way (this problem was known as the "unreliability" of diagnosis), in 1980 the *DSM* began to use lists of symptoms to establish clear definitions for each disorder.[23] Almost all mental health professionals across a variety of settings, from hospital clinics to private practices, now use these formal definitions for clinical diagnosis. Moreover, these definitions have percolated out of the mental health clinical arena and are used in epidemiological studies of disorder in the community, in research studies of treatment outcomes, in marketing of antidepressant medications, in preventive efforts in schools, in screening in general medical practice, in court proceedings, and in many other settings. In effect, these *DSM* definitions have become the authoritative arbiter of what is and is not considered mental disorder throughout our society. What might seem like abstract, distant, technical issues concerning these definitions in fact have important consequences for individuals and how their suffering is understood and addressed.

The fact that these symptom-based definitions are the foundation of the entire mental health research and treatment enterprise makes their validity critically

important. Psychiatric research and treatment are like an upside-down pyramid, and the *DSM* definitions of mental disorders that determine who is counted as disordered are the one small point on which the soundness of the entire pyramid rests. Even the best clinical history taking and diagnostic interviewing or the best research sample selection, experimental design, and statistical analysis of data will not produce meaningful results if they use an invalid definition of disorder that mixes normal and abnormal features. Archimedes famously boasted, "Give me a lever long enough, and a pivot on which to rest it, and I will move the earth." In modern psychiatry, definitions move the treatment and research firmament, and modern clinicians with an invalidly broad definition can move diagnosed disorder to virtually whatever level they desire, especially when they deal with a disorder such as depression that features such symptoms as sadness, insomnia, and fatigue, which are widespread among nondisordered people. Thus the recent focus in psychiatry on reliability of diagnosis based on symptoms has been pursued at some cost to validity—that is, whether the diagnosis represents a correct attribution of disorder.[24] The *DSM's* criteria for Major Depressive Disorder are one instance in which increased reliability has had the inadvertent side effect of creating substantial new validity problems.

The *DSM*'s Definition of Major Depression

The current official psychiatric definition of depressive disorder that is the basis for clinical diagnosis and research studies is found in the most recent edition (4th ed., text revision) of the *DSM.*[25] The *DSM* definition of Major Depressive Disorder (MDD), the category under which most depressive disorders fall, is lengthy and has several qualifiers and exceptions. We defer full analysis and critique of the *DSM*'s approach to depressive disorder until chapter 5. For purposes of this initial discussion, we consider the most important features of the definition, which consists of symptom and duration requirements and a bereavement exclusion.

DSM diagnosis of MDD requires that five symptoms out of the following nine be present during a 2-week period (the five must include either depressed mood or diminished interest or pleasure): (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 or inappropriate guilt; (8) diminished ability to think or concentrate or indecisiveness; and (9) recurrent thoughts of death or suicidal ideation or suicide attempt.[26]

These symptom criteria form the heart of the definition of MDD, but there is one further important clause in the definition: "The symptoms are not better accounted for by Bereavement, i.e., 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."[27] In other words, patients are exempt from diagnosis if their symptoms are due to what the *DSM* defines as a normal period of bereavement after the death of a loved one, lasting no more than 2 months and not including especially serious symptoms, such as psychosis or thoughts about suicide. This limited "bereavement exclusion" is the definition's only acknowledgment that some instances of normal intense sadness might satisfy the symptomatic criteria.

The *DSM* definition of depressive disorder is reasonable in many ways. Its criteria might give rise to disagreements about particular symptoms, but each is widely and consensually believed to be an indicator of depressive disorder, and pre-*DSM* psychiatry recognized them as such. One might also debate the exact number of symptoms needed for diagnosis; some would argue for laxer requirements with fewer symptoms, others for more stringent symptom requirements to ensure that disorder exists, and still others would insist that there should be no sharp cutoff but rather a dimensional continuum of severity.[28] One might also dispute whether the required duration of 2 weeks is sufficient, but it is sometimes clear within 2 weeks of onset that someone has a depressive disorder, and clinicians should not be kept from diagnosing such cases even if the typical duration of depressive disorders is much longer. Likewise, it seems reasonable to exclude people who are recently bereaved. The criteria are also fairly clear and, in most cases, not more difficult to measure than typical psychiatric symptoms in other disorders. The reasonableness, clarity, and efficiency of its use help account for the nearly universal adoption of the *DSM* definition of MDD.

What, then, is the problem with this definition? The essence of the definition is that, aside from a few exceptions, the presence of a particular group of symptoms is sufficient to diagnose the presence of the disorder. Yet symptoms such as depressed mood, loss of interest in usual activities, insomnia, lessened appetite, inability to concentrate, and so on might naturally occur for a period of 2 weeks in the absence of any disorder after any of a wide range of negative events, such as betrayal by romantic partners, being passed over for an anticipated promotion, failing a major test that has serious implications for one's career, discovering a life-threatening illness in oneself or a loved one, or enduring the humiliation that follows revelations of disgraceful behavior. Such reactions, even when quite intense due to the severity of the experience, are surely part of normal human nature. Just as it is obvious why the *DSM* excludes bereavement from diagnosis, by parity of reasoning it seems obvious that it should also exclude these other sorts of reactions to negative circumstances. The diagnosis, however, does not exclude such nongrief responses. Because of the symptom-based nature of the criteria, any sadness response involving enough of the specified symptoms for at least 2 weeks will be misclassified as a disorder, along with genuine psychiatric disturbances. In attempting to characterize the kinds of symptoms suffered in depressive disorders without reference to the context in which the symptoms occur,

contemporary psychiatry has also inadvertently characterized intense normal suffering as disease.

Consider, for example, the following cases:

Case 1: Ending of a Passionate Romantic Relationship

A 35-year-old single female professor has sought psychiatric consultation to obtain medicine for insomnia. She must present a paper as part of a job interview and is afraid that she is not able to function adequately enough to do so. She reports that for the past 3 weeks she has experienced depressed mood and extreme feelings of sadness and emptiness, as well as lack of interest in her usual activities (in fact, she has spent much of her time lying in bed or watching television). Her appetite has diminished, and she lies awake long into the night unable to fall asleep due to the pain of her sadness. She is fatigued and lacking in energy during the day and cannot concentrate on her work. Because her painful feelings distract her during work, she is barely able to meet minimal occupational obligations (e.g., she shows up at her classes poorly prepared, has not attended faculty meetings, and has difficulty concentrating on her research). She has also avoided social obligations.

When asked about what might have precipitated these distressing feelings, she reports that about a month before, a married man with whom she had had a passionate 5-year love affair decided he could not leave his wife and ended the affair. The woman had perceived this relationship as a unique, once-in-a-lifetime romance that had an extraordinary combination of emotional and intellectual intimacy.

The woman agrees to check in with the psychiatrist periodically. Subsequently, as weeks go by, her sense of loss subsides gradually and is replaced by a feeling of loneliness and the need to get on with her life and find a partner. Eventually, she starts dating again, and after several more months the woman meets a new love interest, and any residual symptoms disappear.

Case 2: Loss of a Valued Job

A 64-year-old married man has developed feelings of sadness and emptiness, lack of pleasure in activities, insomnia, fatigue and lack of energy, and feelings of worthlessness. He is not interested in seeing friends and seems unable to concentrate on anything. He yells at his wife when she attempts to console him and rejects her efforts to comfort him.

The feelings were triggered 2 weeks before when the company the man worked for unexpectedly fired him as part of a corporate downsizing. The firing came just 6 months before he would have qualified for the company's retirement plan. One of the major reasons the man chose to work for the

company and then spend two decades with it had been the prospect of generous retirement benefits. The loss of these benefits means that he and his wife have very little retirement income other than Social Security to look forward to.

Subsequently, the couple is forced to sell their house and move to a small apartment. The man finds part-time work that, along with Social Security, provides barely enough resources to sustain him and his wife. He remains bitter about how he was treated, but his symptoms gradually subside over time.

Case 3: Reaction to a Life-Threatening Medical Diagnosis in a Loved One

A 60-year-old divorced woman who is visiting a medical center distant from her home asks a physician at the center for medication to help her sleep. The woman's daughter, an attorney and her only child, to whom she is very close and who is the pride of her life, was diagnosed just 3 weeks before with a rare and potentially fatal blood disease. After receiving the news of her daughter's diagnosis, the mother was devastated by feelings of sadness and despair and was unable to function at work or socially. Although she kept up a brave front for her daughter and was able to help her child with the arrangements for the medical consultation, the mother has been in a state of great distress since the diagnosis; she cries intermittently, is unable to sleep, lacks the ability to concentrate, and feels fatigued and uninterested in her usual activities as she attempts to come to terms with the news of her daughter's condition.

These symptoms gradually abate over a number of months during her daughter's treatment and struggle with the illness, which eventually stabilizes but remains a threat. The woman continues to periodically feel sad about her daughter's situation, but her other symptoms subside as she adjusts to the new circumstances and limits of her daughter's life.

Each of these people easily meets the symptomatic requirements for MDD and the *DSM* would thus classify them as psychiatrically disordered. Their symptoms persist beyond the 2-week duration criterion, they are experiencing significant role impairment or distress, and the bereavement exclusion does not apply to them. Yet these reactions seem to fall within the normal range for persons who have suffered from the sudden end of a passionate romantic relationship, the loss of a valued job, or the diagnosis of a serious illness in a beloved child. The symptoms that these people report are neither abnormal nor inappropriate in light of their particular situations.

What characteristics tend to suggest that these conditions are not disorders? In each case, symptoms emerge only after the occurrence of a discrete life event involving great loss. Further, the severity of these loss responses, although very

intense, is reasonably proportional to the nature of the losses that have been experienced. Finally, the symptoms either end when circumstances change for the better, endure because the stressful situation persists, or eventually go away with the passage of time. We do not believe that most thoughtful clinicians today, if making an independent judgment outside the sway of the *DSM*, would classify such reactions as disorders, any more than their predecessors did.

Stating that the *DSM*'s definition of depressive disorder mistakenly encompasses some normal emotional reactions in no way implies that there are not genuine depressive disorders. Such disorders do exist, they can be devastating, and the *DSM*'s definition does encompass them. However, they look very different from the kinds of normal reactions described here. Popular portrayals of depression uniformly present a picture of profound, immense, and immobilizing suffering that is bewilderingly disengaged from actual life circumstances, and this sort of experience does imply genuine disorder.

For example, consider the case of Deanna Cole-Benjamin, featured in a *New York Times Magazine* story about a new treatment for depression:

> Her youth contained no traumas; her adult life, as she describes it, was blessed. At 22 she joined Gary Benjamin, a career financial officer in the Canadian Army, in a marriage that brought her happiness and, in the 1990's, three children. They lived in a comfortable house in Kingston, a pleasant university town on Lake Ontario's north shore, and Deanna, a public-health nurse, loved her work. But in the last months of 2000, apropos of nothing—no life changes, no losses—she slid into a depression of extraordinary depth and duration.
>
> "It began with a feeling of not really feeling as connected to things as usual," she told me one evening at the family's dining-room table. "Then it was like this wall fell around me. I felt sadder and sadder and then just numb."
>
> Her doctor prescribed progressively stronger antidepressants, but they scarcely touched her. A couple of weeks before Christmas, she stopped going to work. The simplest acts—deciding what to wear, making breakfast—required immense will. Then one day, alone in the house after Gary had taken the kids to school and gone to work, she felt so desperate to escape her pain that she drove to her doctor's office and told him she didn't think she could go on anymore.
>
> "He took one look," she told me later, "and said that he wanted me to stay right there in the office. Then he called Gary, and Gary came to the office, and he told us he wanted Gary to take me straight to the hospital."[29]

Aside from the extraordinary severity and duration of the symptoms, it is noteworthy that the seriousness of this depressive condition has no relation to any events that normally might be expected to trigger such episodes.

Or consider Andrew Solomon's powerful depiction of his depressive illness:

> [My depression] had a life of its own that bit by bit asphyxiated all of my life out of me. At the worst stage of major depression, I had moods that I knew were not my moods: they belonged to the depression . . . I felt myself sagging under what was much stronger than I; first I could not use my ankles, and then I could not control my knees, and then my waist began to break under the strain, and then my shoulders turned in, and in the end I was compacted and fetal, depleted by this thing that was crushing me without holding me. Its tendrils threatened to pulverize my mind and my courage and my stomach, and crack my bones and desiccate my body. It went on glutting itself on me when there seemed nothing left to feed it.[30]

Again, Solomon's profound depression has "a life of its own," in the sense that its seriousness is not related to specific losses or other negative events that might normally lead to such feelings.

In perhaps the most elegant description of depression, *Darkness Visible,* William Styron describes his reaction to learning that he had won a prestigious literary prize:

> The pain persisted during my museum tour and reached a crescendo in the next few hours when, back at the hotel, I fell onto the bed and lay gazing at the ceiling, nearly immobilized and in a trance of supreme discomfort. Rational thought was usually absent from my mind at such times, hence *trance.* I can think of no more apposite word for this state of being, a condition of helpless stupor in which cognition is replaced by that "positive and active anguish."

Styron's condition persists independently of any social context: "In depression . . . the pain is unrelenting, and what makes the condition intolerable is the foreknowledge that no remedy will come—not in a day, an hour, a month, or a minute. If there is mild relief, one knows that it is only temporary; more pain will follow."[31] Styron's debilitating symptoms did not emerge after any stressful experience but actually arose after what would ordinarily be a cause for celebration.

The sociologist David Karp's *Speaking of Sadness* offers another typical depiction:

> By any objective standard I should have been feeling pretty good. I had a solid academic job at Boston College, had just signed my first book contract, and I had a great wife, beautiful son, and a new baby daughter at home. . . . Each sleepless night my head was filled with disturbing ruminations and during the day I felt a sense of intolerable grief as though somebody close to me had died. I was agitated and sensed a melancholy

> qualitatively different from anything in the past. . . . I thought for sure that my depression was rooted in these situational demands and that once I got tenure it would go away. I was promoted in 1977 and found that the depression actually deepened.[32]

Just as Styron's depression developed after a positive experience, Karp's dangerously severe condition was isolated from the actual circumstances of his life.

As these examples illustrate, the cases that both popular media and psychiatric texts typically depict are clearly genuine disorders. Yet these descriptions also illustrate that symptoms in themselves do not distinguish depressive disorders from normal sadness; the symptoms are not qualitatively different from what an individual might naturally experience after a devastating loss, as in our earlier case illustrations of normal reactions to major life disruptions. Instead, it is the absence of an appropriate *context* for symptoms that indicates a disorder. These cases either emerged in the absence of any loss event or developed after the occurrence of a positive event, such as winning a prestigious award or obtaining tenure. Their severity was of grossly disproportionate intensity to the sufferer's actual circumstances. Finally, symptoms persisted independently of any stressful contexts, took on a life of their own, and were immune to changes in external conditions. The fact that the literature emphasizes such examples can mislead us into overlooking the fact that the *DSM* diagnostic criteria themselves are not limited to such conditions and invalidly encompass a great range of intense normal reactions.

The basic flaw, then, of the *DSM* definition of MDD, as well as of all efforts that rely on it, is simply that it *fails to take into account the context of the symptoms and thus fails to exclude from the disorder category intense sadness, other than in reaction to the death of a loved one, that arises from the way human beings naturally respond to major losses.* The resultant lumping of nondisordered with dysfunction-caused symptoms of depression, and the classification of both as *disorders,* is a fundamental problem for current research, treatment, and social policy regarding depression. Moreover, as we show, the problem has been getting much worse in recent years, with growing pressure to use a lower number of symptoms, sometimes as few as two, as sufficient criteria for diagnosing a disorder. The potential for false-positive diagnoses—that is, people who meet the *DSM's* diagnostic criteria but do not in fact have a mental disorder—increases exponentially as the number of symptoms required for a diagnosis decreases.

The *DSM*'s overinclusive criteria for depressive disorder ultimately compromise psychiatry's own goals and concepts. The *DSM* aims to identify psychological conditions that can be considered genuine medical disorders and to distinguish them from problematic but nondisordered conditions.[33] Thus the error we are pointing to in disorder categories such as MDD is an error in terms of the *DSM*'s own stated aspirations.

The Distinction Between Normality and Disorder

Our core argument about the *DSM*'s definition of depressive disorder depends on the assumption that normal sadness can be intense; can be accompanied by sleeplessness, lack of concentration, changed appetite, and so on; can be impairing or distressful; and can last for 2 weeks, as the criteria demand. But what is the implicit understanding of normality and disorder by which one can distinguish painful sadness that is normal from that which is disordered?

Normal functioning is not mere statistical commonality. Some disorders can be statistically "normal" in a population, as are gum disease and atherosclerosis in ours, yet they are disorders nonetheless; and some normal variations can be quite rare. We must also distinguish disorder from social desirability and social values. Even the *DSM* acknowledges that an individual who is socially deviant or whose nature is in conflict with the values of society is not thereby necessarily disordered.[34] Adequate accounts must not only distinguish disorder from social values but also explain in what ways disorders are real medical ailments that represent, at least in part, some objective problem in individual functioning.

The most plausible demarcation point between human normality and disorder in the medical sense is, we believe, that between biologically "designed" functioning (i.e., the result of natural selection) and the failure of such functioning, that is, *dysfunction.*[35] This view comports well with commonsense intuitions and is probably the most widely accepted and defensible view among those who are concerned with the conceptual foundations of psychiatry, as well as medicine more generally.[36] For example, the criterion for normal functioning of bodily organs is what they are biologically designed to do and how they are designed to do it. Thus the heart serves to pump blood, the kidneys to eliminate waste, and the lungs to enable us to breathe, and if these functions are accomplished by the structures designed to accomplish them, functioning is normal. Disorder exists when the organ is unable to accomplish the function for which it is biologically designed.

Similarly, psychological processes that were selected as part of human nature have natural functions, that is, the effects for which they were naturally selected. Considerable neurobiological and psychological research suggests that the mind is made up of many specific modules or mechanisms that are designed to respond to specific environmental challenges.[37] Thus *contextuality* is an inherent aspect of many psychological mechanisms; they are designed to activate in particular contexts and not to activate in others. Fear responses, for example, are biologically designed to arise in dangerous situations but not in safe situations. Likewise, innate mechanisms that regulate reactions of sadness, despair, and withdrawal naturally come into play after humans suffer particular kinds of losses.[38] Conversely, dysfunctions in which sadness mechanisms do not operate as designed constitute disorders. The implication is that only in the light of some account, however provisional or sketchy, of how loss response mechanisms are designed to work and thus

of their normal functioning do we have grounds for calling some responses to loss *disordered.*

As with all human traits, there is much variation among individuals in the sensitivity with which they respond to loss with sadness. Culture also influences designed tendencies in various ways, so evaluating whether a response fits within the naturally selected range is sometimes no easy task. Nevertheless, under appropriate conditions, virtually all humans have the capacity to develop nondisordered sadness as a biologically selected adaptation to handling loss. In principle, this biological capacity provides a baseline for judging some cases as clear examples of normality and disorder.

An important caveat: because of our primitive state of knowledge about mental functioning, our understanding of how normal emotions, including sadness, are designed to work remains speculative and open to revision. Yet some fundamental principles, at least in a broad provisional form, seem overwhelmingly plausible and offer a sufficient basis for critically examining the validity of criteria for depressive disorder. These principles allow us to make some general distinctions between cases that clearly seem to indicate normal sadness and cases of depressive disorders, while acknowledging a large domain of borderline, ambiguous, and fuzzy cases. We emphasize three essential features of nondisordered loss in chapter 2: they emerge because of specific kinds of environmental triggers, especially loss; they are roughly proportionate in intensity to the provoking loss; and they end about when the loss situation ends or gradually cease as natural coping mechanisms allow an individual to adjust to the new circumstances and return to psychological and social equilibrium.

Important questions arise because we do not yet know precisely which internal mechanisms produce loss responses or what these mechanisms are actually like. If the mechanisms are inferred to exist but their specific nature is unknown, how can one tell that normal loss responses are indeed part of our biological heritage? And, without knowing the mechanisms, how can we tell what is normal and what is disordered?

The fact is that, although the distinction cannot yet be determined precisely, in the history of medicine and biology scientists have routinely drawn such inferences about normal and disordered functioning from circumstantial evidence without knowing the underlying mechanisms. So, for example, Hippocrates knew that blindness and paralysis are disorders and that there are mechanisms that are designed to allow human beings to see via their eyes and move via muscular effort, but he knew little of the mechanisms themselves and thus little of the specific causes of most cases of blindness and paralysis (other than gross injury). It took thousands of years to figure out those mechanisms, but during that time it was universally understood from circumstantial evidence that sight and movement are parts of human biological design. It is no different in principle with human mental capacities that are part of our biological nature, such as basic emotions.

Another concern might be that, because we do not understand the loss response mechanisms, we cannot with confidence state the function of the loss response and cannot therefore know what is normal and abnormal. Indeed, unlike the functions of the eyes and muscles, the functions of loss responses are not apparent and are subject to dispute.[39] Fortunately, it is often possible from available evidence to infer roughly what responses of a mechanism are normal, even without knowing the reason for the responses. For example, everyone agrees that sleep is an elaborately designed response and that some sleep conditions are normal whereas others are sleep disorders, but there is little scientific consensus on the functions that explain why we sleep. Without an adequate understanding of the function of loss responses to guide us, we too must engage in such admittedly presumptive but, we believe, still plausible inferences.

By depressive disorders, then, we mean sadness that is caused by a harmful dysfunction (HD) of loss response mechanisms.[40] According to the HD definition, a collection of symptoms indicates a mental disorder only when it meets both of two criteria. The first is dysfunction: something has gone wrong with some internal mechanism's ability to perform one of its biologically designed functions. Second, the dysfunction must be harmful. Cultural values inevitably play the primary role in defining what sorts of dysfunctions are considered harmful. In sum, a mental disorder exists when the failure of a person's internal mechanisms to perform their functions as designed by nature impinges harmfully on the person's well-being as defined by social values and meanings.

The HD analysis of disorder does not attempt to yield a precise conceptual boundary because the concepts of normality and disorder, like most concepts, do not themselves have precise boundaries and are subject to indeterminacy, ambiguity, fuzziness, and vagueness and so yield many unclear cases. Despite such fuzzy boundaries, the HD concept of disorder is useful and coherent because it enables us to adequately distinguish a range of clear normal cases from a range of clear disorders. Analogously, there are real distinctions between red and blue, child and adult, and life and death, although there are no sharp boundaries between these pairs. By contrast, current diagnostic criteria for depressive disorder, we argue, substantially fail to distinguish even many clear cases of normal sadness from disorder.

Mechanisms that are biologically designed to generate loss responses may fail to perform their functions in the appropriate contexts in a variety of ways.[41] Loss responses can emerge in situations for which they are not designed, they can be of disproportionate intensity and duration to the situations that evoke them, and in extreme cases they can occur spontaneously, with no trigger at all. For example, depressions such as William Styron's, which arose after the reception of a prestigious award, or David Karp's, which emerged after a successful tenure decision, indicate that loss response mechanisms have gone awry. Dysfunctions of loss responses may also involve distorted cognitive perceptions of the self, the world, and the future that trigger inappropriate sadness.[42] Such distortions may yield inappropriately sensitive mechanisms that magnify the meanings of minor losses beyond

the normal range of culturally appropriate stimuli. Someone who becomes deeply depressed after the death of a pet goldfish or a minor perceived slight, for example, displays such overly sensitive, disproportionate loss response mechanisms, unless special circumstances make the loss of much greater importance than is usual.

Yet it is not just the emergence of depression in the absence of appropriate causes that defines a dysfunction. Disorders might arise after an initially normal-level response to actual losses, but the response might then become disengaged from the circumstances of the loss and persist with disproportionate intensity long after the initial provoking conditions have ended. Or, among susceptible individuals, the experience of loss events can sometimes produce biochemical and anatomical vulnerabilities that make recurrences of depressive episodes more likely with less and less provocation.[43] Even if they begin as normal responses, emotional reactions that become detached from a specific time, place, and circumstance indicate dysfunctional loss response mechanisms.

Finally, dysfunctions in loss responses can sometimes cause symptoms that are so extreme that they indicate dysfunction in themselves. Depressive reactions that feature prolonged complete immobilization or loss of contact with reality, such as hallucinations, delusions, and the like, would not stem from appropriately functioning loss mechanisms and have always been recognized as disorders. Such inappropriate and excessive responses are analogous to dangerously high fevers or uncontrolled vomiting, which are design failures of otherwise adaptive responses.

Note that our distinction between sadness due to internal dysfunction versus sadness that is a biologically designed response to external events differs in important respects from the traditional distinction within psychiatry between depressions that are *endogenous* (i.e., spontaneously caused by internal processes, having no external trigger) and *reactive* (i.e., triggered by some external event).[44] Endogenous depressions by definition arise in the absence of real loss, and so are almost always due to internal dysfunctions. In contrast, many reactive depressions are proportionate to environmental events and so are normal responses.

However, not all reactive depressions are normal. External events can so deeply affect individuals that they trigger internal dysfunctions. For example, environmental traumas such as the sudden death of a loved one, forced relocation from one's home, or being the victim of a violent crime can cause designed loss response mechanisms to break down and an enduring disorder to occur.[45] As noted, emotional reactions can be so disproportionate to their triggering events as to suggest dysfunction, or the symptoms can take on a life of their own and fail to extinguish when the stressor ends. Thus, among reactive depressions to losses, some are disorders and some are not. The *presence* of an internal dysfunction, not the *cause* of this dysfunction (which may be endogenous or reactive), defines depressive disorders. Consequently, there is no simple one-to-one relationship between the traditional endogenous-reactive distinction and our distinction between internal dysfunction and biologically designed response.

To address a final point of possible confusion: the evolutionary design approach to distinguishing normal from disordered conditions does not imply that all depressive disorders have physiological causes or that there is always a brain problem when there is a depressive disorder. Although physiological causes do often produce disorders, psychological or social factors can also lead to dysfunctions. Biological design includes the design of various mental mechanisms (e.g., belief, desire, emotion, perception) that work via meanings humans assign to represent reality, and physiological descriptions may not capture how such meanings operate. There might be mental disorders that cannot be described as malfunctions in the underlying physiological machinery but as malfunctions at the mental level of meanings. This is not as mysterious as it might sound; think of the fact that computer software can malfunction in hardware that itself is working properly. The processing of meanings that recent cognitive science envisions as analogous to the "software" of the mind can perhaps similarly go awry without any underlying physiological malfunction. Our discussion is neutral on such issues of etiology, although we generally believe that there can be an array of biological, psychological, and social causes of both normal sadness and depressive disorder; research that resolves the clash of theories about the causes of depression must decide this issue.

One great advantage of our critique based on the HD approach is that it acknowledges that depressive disorders do exist and provides defensible grounds for improving psychiatry's diagnostic criteria. There are other, more radical, critiques of psychiatric diagnosis that dismiss diagnosis in general and that leave no room for constructive engagement with psychiatry. For example, psychiatrist Thomas Szasz's argument that there are no mental disorders because disorders require physical lesions; sociologist Thomas Scheff's labeling theory that reduces diagnosis to social control; behaviorist claims that all behavior is the outcome of learning processes and therefore that no mental disorder can exist; or anthropologists' assertions that distinctions between normal and abnormal functioning are purely cultural and therefore arbitrary all deny the possibility of making a conceptually coherent diagnostic distinction between depressive disorders and normal intense sadness responses.[46] They thus understate the real and distinct problems that genuine depressive disorders pose while at the same time they preclude the prospect of effectively critiquing overexpansive psychiatric definitions of disorder.

The Advantages of Distinguishing Normal Sadness From Depressive Disorder

Even if the *DSM* criteria are flawed in a way that allows the creation of an inflated amount of depressive disorder, why is correcting this error so important? There are some considerable advantages to doing so:

Pathologization of normal conditions may cause harm, and avoidance of such pathologization may decrease such harm. Not only may patients be misled to consider

themselves disordered and undertake unnecessary treatment, but also the social responses to nondisordered and to dysfunctional conditions usually differ. Social networks typically respond with social support and sympathy to sadness that appears after stressful life events.[47] Dysfunctional depressions, in contrast, typically elicit hostility, stigma, and rejection and lead to the loss of social support.[48] To subject those with normal sadness to the social prejudice faced by those with mental illness does not serve to combat that prejudice. It remains true, however, that for those who do have a genuine mental disorder, the disadvantages of diagnostic stigma must be balanced against the fact that diagnosis of their abnormal conditions with an official label that offers access to services may come as a welcome relief.

The distinction between disordered and normal sadness should improve assessments of prognosis. An essential purpose of diagnosis is prognosis: predicting the future course of a disorder.[49] The prognoses for people whose symptoms stem from nondisordered sadness usually differ from those for people with disorders. Symptoms of nondisordered conditions are likely to abate over time without intervention, to disappear if precipitating circumstances change, and to be responsive to generic social support. In contrast, symptoms that stem from internal dysfunctions are likely to be chronic and recurrent and to persist independently of stressful life circumstances.[50] An adequate distinction between disorder and nondisorder should provide better prognostic predictions.

Accurate diagnoses point to appropriate treatments. Although medication or therapy can help ease the pain that arises from both normal sadness and depressive disorder, they are often unnecessary in cases of nondisordered sadness, which do not involve internal dysfunctions. In some cases, such as bereavement, treating normal loss responses as diseases can even be counterproductive because it may exacerbate and prolong symptoms.[51] Conversely, the treatment of depressive disorders often involves pharmacotherapies, cognitive and other psychotherapies, or a combination of modalities to overcome dysfunctional conditions. An adequate conceptual distinction between dysfunctions and normal responses may enable us better to specify what sorts of responses can work most effectively for symptomatically similar, but actually distinct, conditions.

Separating normal sadness from depressive disorders can help in recognizing the relationship of sadness to adverse social conditions and thus in identifying appropriate social interventions. Psychiatry now tends to view depression as a major cause of many social problems, including welfare dependence, drug addiction, and poverty.[52] The first course of action would be to treat the illness and then to help depressed individuals to overcome their other challenges. Normal sadness, however, is much more likely to be the result rather than the cause of social problems. Recognizing the impact of social problems on normal human emotions would suggest that correcting these problems would be an appropriate initial response.

The separation of depressive disorder from normal intense sadness should provide a basis for more accurate epidemiological estimates of the prevalence of depressive

disorders and the cost of treating it. The failure to distinguish normal from disordered sadness results in large overestimates of people who have mental disorders, misleading policy makers into formulating poor public policy. Prevalence estimates count as diseased all people who have sufficiently intense symptoms, whether disordered or not.[53] They direct the attention of policy makers and mental health professionals toward conditions that may not need intensive expert attention and away from problems that can most benefit from professional help. Conflating normal sadness with depressive disorder also results in greatly overinflated estimates of the economic costs of depression.[54] These overstatements, in turn, have potentially negative policy implications in that they increase the reluctance of elected officials, insurance companies, and other policy makers to develop cost-effective responses to depression. Although no one who is suffering should be denied access to services, separating nondisordered from dysfunctional conditions can focus the expertise of mental health professionals on true mental disorders and lead to a more efficient use of mental health resources.

Distinguishing disorder from normal sadness allows for a better estimate of unmet need for mental health services. Because they fail to distinguish normal sadness from depressive disorder, and because nondisordered individuals are less likely to seek treatment, population surveys make it seem as if only a minority of disordered people are treated for their conditions. This has led social policies to focus on the presumed vast amount of unmet need for treatment.[55] These policies now emphasize widespread screening for depression among people who have not voluntarily sought treatment. Screening instruments likely uncover more normal sadness than depressive disorder but treat both conditions as if they were disorders. The reduction of such overdiagnosis could reduce the needless and potentially harmful overprescription of medication.

Drawing a more careful distinction between disorder and normal sadness allows researchers to select samples that more accurately reflect true disorders. Meaningful research into the causes of depressive disorder and into the best treatments for either depressive disorder or intense normal sadness requires that the groups studied be reasonably homogeneous in nature so that the results can be appropriately understood and generalized. The causes of depressive symptoms that arise from dysfunction are generally different from the causes of normal sadness, so the entire field of depression research remains problematic until the appropriate distinction is made.

Distinguishing nondisordered from dysfunctional loss responses avoids medicalizing our thinking about normal sadness and thus maintains the conceptual integrity of psychiatry. Aside from any other advantages, the long-term credibility of psychiatry and psychiatric diagnosis depends on getting the disorder-nondisorder distinction right by labeling only genuine psychiatric conditions as disorders. The failure to adequately separate naturally designed sadness responses from internal dysfunctions misconstrues as a mental disorder a basic and universal

aspect of the human condition and thus inappropriately pathologizes a tremendous range of human behavior, undermining the credibility of psychiatry.

Understanding how psychiatry has blurred a crucial distinction and tends to misclassify intense sadness as a disorder is also useful to the ordinary person. Every time people enter physicians' offices or their children take routine screening instruments in school, the conceptual muddles we identify in this book could lead to unwarranted diagnoses and treatment. To be an effective consumer of medical services, prepared patients should understand both how professionals arrive at the diagnoses to which they are subject and what questions to ask about the problems these diagnoses may entail.

Finally, it should be kept in mind that the truth about whether someone is disordered or normally sad often matters in a practical way because diagnoses of mental disorders influence many consequential decisions. For example, such diagnoses can make it more difficult to obtain life or health insurance or increase the cost of such insurance; they can be negative considerations in divorce proceedings that consider custody of children; and they frequently disqualify individuals from participating in clinical trials for new medications for severe conditions such as cancer. Because of the role that diagnosis has in so many areas of our lives and the assumption that a diagnosis represents a genuine medical condition, confusing depressive disorder with normal sadness should not be taken lightly.

Some Caveats: What About the Disadvantages of Such a Distinction?

Some might object that drawing a distinction between normal and disordered sadness, whatever its intellectual merits, is potentially harmful for various reasons. We can't address all the potential concerns, but it is worth briefly considering a few.

Are we somehow dismissing the suffering of those with normal sadness? By calling certain responses "normal," we in no way intend to minimize, let alone demean, the level of suffering involved; indeed, the extreme pain of normal sadness can often match that of depressive disorder. But just as you would want to distinguish a normal intense pain that results from, say, childbirth or a broken bone (which you would want to manage and treat) from an equally intense pain that results from a pain disorder in which the pain is not a normal response to a bodily lesion (which would have important implications for treatment), these intense forms of sadness need to be distinguished so that they can be understood and optimally managed and treated.

Could our analysis result in insurance barriers that would deny treatment to people who seek it even though they are not truly disordered? Yes, that is possible, but unlikely. The reality is that clinicians always have found and always will find

ways of responding to patient needs and of classifying these needs consistent with diagnostic definitions so that they receive reimbursement for treatment. Moreover, as in other medical domains, a strong argument can be made for reimbursement of treatment for nondisordered intense emotional responses because of the disabling effects they can have and as a preventive measure. An accepted distinction between normal and disordered intense sadness might even facilitate a discussion of such changes in the reimbursement system.

Are we moralists who think that people should not generally rely on treatment, and specifically on medication, but should somehow be forced to muddle through their difficulties? We are not arguing for or against using medication to treat normal feelings of sadness. That is a matter for individuals and their physicians to decide. Rather, we are arguing that conceptually illegitimate diagnoses of normal responses to events as disorders could prejudice such decisions by making it seem as though there is an internal malfunction for which medication is the optimal treatment when evidence suggests that other interventions may offer equal or better relief and avoid the possible negative side effects of medication. We are simply saying that such treatment decisions should be based on a correct understanding of the condition. Indeed, once an adequate conceptual distinction is made, clearer research on optimal treatment of intense normal sadness might move forward more effectively.

Doesn't diagnosis with a medical disorder reduce blame, and aren't we therefore encouraging the blaming of sad individuals for being emotionally weak? For example, if intense sadness is not diagnosed as a disorder, won't it then be assumed that experiencing such feelings is a character flaw, and won't people be urged to "shape up" and be strong rather than indulging such feelings? There is in fact scant scientific evidence on whether diagnosis does lead to beneficial relief from personal blame, in contrast to the vast evidence that it leads to harmful stigma. But it must be acknowledged that diagnosing individuals with a medical disorder, even if unjustified, may sometimes protect them from misplaced blame by family members and others for weakness of character. We argue, however, that there are ways to respond to such misplaced blame other than the extreme misuse of medical categories. In particular, our analysis emphasizes that intense sadness is a natural human capacity and not a character weakness; indeed, normal sadness probably has healing and reparative functions that are still not understood.[56] Oddly enough, many cultures actually blame individuals if they are inadequately sad when loss occurs (such as displaying too few or too briefly the signs of mourning after the death of a relative) because it seems to show lack of commitment or caring. The fact that intense sadness may be related characterologically to depth of feeling and may thus be more important for coping with loss in some individuals than in others is hardly a weakness. Our analysis suggests that blaming can easily be addressed without labeling the individual as disordered. It should also be recognized that diagnosis with a disorder is no redoubt against personal contempt and blame.

Isn't it cold-blooded that we focus on conceptual issues and don't spend much time exploring the painful experience of depression itself? There are rafts of books detailing the great impact of depression and vividly documenting the experience of it. Our focus is different because our goal is different, namely, to provide an understanding and critical perspective on how that experience is conceptualized, how it has come to be exploited by a variety of groups, and how its classification has changed in questionable ways over time.

A Note on Terminology

A few terminological clarifications at this point might prevent needless confusion later on. First, although we use *sadness* to describe both the normal human emotion and the experiences described in depressive disorders, the normal responses we consider in fact go beyond sadness and include certain episodes of emotional emptiness, shame, humiliation, and related responses to losses of various kinds, such as loss of self-esteem or loss of standing in a group. So we sometimes use broader, more abstract language such as *loss responses* to refer to such experiences. Even when we do use the word *sadness,* it should be understood that this is shorthand for a broader domain.

Second, when we write of "normal" sadness, we do not mean that everything is statistically normal, or normal in the sense of "okay." Rather, we mean that the sadness is functionally normal or nondisordered, that is, it is the result of the relevant mental processes working as they were biologically designed to work in response to loss. Such "normal" responses can be "abnormal" in a variety of ways: they may be much more intense in some people than in others due to human variation in temperament or different cultural meaning systems; they may be statistically highly unusual because they are responses to a highly unusual environmental circumstance (e.g., someone experiencing extreme grief due to the deaths of several family members within a short time); and they may be abnormal in the sense that they constitute a severe deviation from the individual's usual functioning. None of these forms of statistical abnormality implies disorder.

Third, many of the same kinds of psychological (e.g., blue mood) and physical (e.g., fatigue) phenomena occur in disordered and nondisordered sadness responses. There is no convenient neutral term to describe these phenomena, so we accept the general convention and refer to them as *symptoms.* But it should be kept in mind that this is potentially misleading because of the association of "symptoms" with a medical diagnosis of disorder. The use of the word *symptom* here is intended to be neutral as to whether the phenomenon is a manifestation of disorder or a normal response.

Fourth, as a convenient way to refer to whatever as yet unknown structures in the mind are designed to produce loss responses, we use the phrase *loss response*

mechanisms. The term *mechanism* is common in evolutionary discussions and should not be construed as implying anything reductionistic or literally "mechanistic" about the mind. We assume, for example, that complex individual and cultural meanings enter into loss responses. The term *mechanism* simply indicates that, given that loss responses are part of our biological heritage, there are some structures in the person that are biologically designed to produce such responses at appropriate times.

Finally, because we often refer to similar phenomena when we discuss disorders and normal responses, we use several conventions to try to remain clear about what is under discussion at a given time. When we discuss a specific *DSM* category of disorder (which may, according to our argument, actually mistakenly include some normal as well as disordered conditions), we adopt the *DSM* convention of capitalizing a category, and so write, for example, of Major Depressive Disorder or, more simply, Major Depression. In addition, because the term "depression" is ambiguous as to normal or disordered conditions, we specifically use the term *disorder* when discussing depressive pathology—referring to it usually as "depressive disorder." We do not capitalize this phrase because we are not referring to a *DSM* category—which, we argue, confuses disorder and normality—but to just those conditions that are genuine disorders. (We refrain from using the common phrase *clinical depression* to refer specifically to disorders because many instances of normal sadness are now seen in clinics.) When we want to refer to all sadness conditions, whether normal or disordered, we use generic phrases such as "depressive condition" or, simply, "depression."

What We Hope to Accomplish

Depression has gained an iconic status in both the contemporary mental health professions and the culture at large. Many experts claim it is a dire public health problem that afflicts a large proportion of the population. The seeming massiveness of the problem simultaneously calls for urgent policy responses and yet paralyzes the will to respond.

While recognizing the reality of depressive disorder and the enormous suffering it causes, this book strives to bring perspective into discussions of depression. It shows how an inadequate conceptual distinction between disorder and nondisorder is a crucial weakness in the entire clinical and research industry devoted to this condition and demonstrates that the problems amplify as the erroneous definition echoes through various social institutions. In particular, virtually all discussions of this condition ignore the critical question of when depressive symptoms indicate a mental disorder and when they are nondisordered responses to loss. Answers to this question would affect our understanding of how many people have mental disorders, to what degree we can prevent

depression, whom we should treat for this condition, and what sort of policies we should develop. Exploring the current misdefinition of depression is a way of showing how seemingly esoteric technical issues can inadvertently influence broader social movements and how various constituencies are motivated to exploit and perpetuate conceptual errors once they are made.