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### History of Depression

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### Abstract and Keywords

The symptoms that define depressive conditions have been recognized for millennia of medical history. The earliest Hippocratic writings not only define depression in similar ways as current works but also use context to differentiate ordinary sadness from depressive disorder. Sadness was understood as a natural reaction to loss; symptoms indicated a disorder only if they were not attributable to an identifiable trigger or if they displayed disproportionate intensity or duration to their triggers. The first serious approaches to subcategorize different types of depressive disorders developed in the seventeenth century. Despite agreement that a melancholic or psychotic form of depression existed, researchers debated the categorization of neurotic or nonpsychotic depressions until 1980 when the *DSM-III* introduced major depression as a unitary category. The *DSM's* diagnostic system was historically anomalous because its diagnoses did not consider the context in which symptoms arose. The only exception within the *DSM*, for uncomplicated symptoms that follow bereavement, was removed from the *DSM-5* in 2013 so that depressive diagnoses now thoroughly conflate adaptive responses to loss with pathological depressions.

Keywords: depression, Hippocrates, DSM-III, diagnosis, melancholia, melancholy, Emil Kraepelin, Sigmund Freud, major depressive disorder, symptoms

Depression, unlike many conditions in the current psychiatric canon, has a lengthy and readily identifiable history. Indeed, it is perhaps the most easily recognizable psychological disorder throughout history; similar symptomatic descriptions occur over a 2,500-year span, representing what historian Stanley Jackson (1986, p. ix) calls a "remarkable consistency." From the earliest medical texts in ancient Greece to the present *Diagnostic and Statistical Manual of Mental Disorders (DSM)*, deep sadness and

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its variants—hopelessness, sorrow, dejection, despondency, emptiness, despair, discouragement—have been mentioned as core features of depression. Related symptoms have included aversion to food, sleeplessness, fatigue, irritability, restlessness, fear of death, repetitive focus on a few negative ideas, lack of pleasure or interest in usual activities, and social detachment.

Yet from the advent of psychiatry until the introduction of the *DSM-III* in 1980, traditional psychiatry recognized that even intense sadness with its associated “symptoms” can be a normal emotional reaction to life circumstances, and took a *contextual* approach to the diagnosis of depressive disorder. Its various definitions did not focus on symptoms alone but emphasized that, to be considered as disorders, depressive reactions must be of disproportionate duration or severity to the situation in which they emerged. Whether a condition was diagnosed as disordered depended not just on the symptoms, which might be similar in normal sadness and in pathological depression. It was also not entirely defined by the condition’s severity, for normal sadness can be severe and disordered sadness can be moderate. Instead, the distinction between sadness and depressive disorder depended on the degree to which the symptoms were an understandable response to circumstances.

This chapter elaborates the history of this contextual approach to depression providing the background for how the definition of depression in the *DSM-III* (1980) overturned thousands of years of thinking, replacing the more nuanced contextual approach with relatively precise and communicable symptomatic criteria that largely ignored the complexities of context, with detrimental side effects for psychiatric diagnosis. Worse, the *DSM-5* (American Psychiatric Association, 2013) for the most part eliminated the slim remainder of the contextual tradition—the bereavement exclusion—so that the current psychiatric diagnosis of major depression is entirely symptom based. Indeed, the most recent *DSMs* represent a significant regression from the earliest efforts to define depressive disorder and separate it from normal sadness. Although a symptom-based definition of depression might prove to be relatively reliable, this reliability comes at the expense of discriminant validity in distinguishing depressive disorders from normal sadness reactions.

## The Classical Tradition

Writing in the fifth century BCE, Hippocrates (460–377 BCE) and the school of Hippocratic physicians that formed around him provided the first known description of melancholia (the Greek name for pathological states of depression), stated succinctly in Hippocrates’s

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*Aphorisms*: “Fear or sadness that last a long time mean melancholia” (Hippocrates, 1923–1931, Vol. IV, p. 185). In addition to fear and sadness, the Hippocratic writings mentioned as symptoms of melancholia “aversion to food, despondency, sleeplessness, irritability, restlessness” (Hippocrates, 1923–1931, Vol. I, p. 263). This description is remarkably similar to the current definition found in the *DSM-5* (American Psychiatric Association, 2013). Unlike *DSM-5*, however, the Hippocratics did not view depression as a free-standing condition but linked it with other conditions, especially anxiety (“fear”) and delusions. On the basis of the latter feature, melancholia was often characterized as “delirium without a fever.” A combination of anxious concerns and nameless fears, depressive symptoms such as blackness of mood and suicidal impulses, and paranoid tendencies such as sullen suspiciousness characterized melancholic conditions. Similarly, Galen (131–201 CE) indicated that although “each (melancholic) patient acts quite differently than the others, all of them exhibit fear or despondency” (Radden, 2000, p. 10). He went on to note: “Therefore, it seems correct that Hippocrates classified all their symptoms into two groups: fear and despondency” (Jackson, 1986, p. 42).

Although the Hippocratics agreed with contemporary accounts on the symptoms of melancholia, the Hippocratic definition in *Aphorisms* also specified the contextual constraint that the symptoms had to last an unusually long time to constitute melancholia. This indicates that it is not depressive symptoms alone but symptoms of unexpected duration that indicate disorder. This insistence that the sadness or fear must be prolonged is a first attempt to capture the notion that disproportion to circumstances is an essential aspect of depressive disorder.

Hippocratic writings rarely focused on distinct external causes of melancholic disorders. Rather, their foundational principle was that health is a state of equilibrium within the body and that disease is due to a disturbance of this balance (Porter, 1997, pp. 55–62). The Greeks viewed mental diseases, like disease in general, in terms of four basic humors: blood, phlegm, yellow bile, and black bile. Each humor possessed two of four properties: hot, cold, moist, or dry. When the humors were in balance with each other, a healthy state resulted. Diseases, both mental and physical, stemmed from too much or too little of one of these humors, a notion that would recur when theories of neurochemical imbalance were developed at the end of the twentieth century. For the Greeks, melancholia was connected to an excess of black bile. Yet mental disturbances that resulted from an excess of black bile were not localized but disrupted a holistic relationship between individuals and their surroundings. A variety of factors, including diet, lifestyles, living conditions, and atmospheric elements, could lead to humoral imbalances.

Traditional diagnostic treatises followed Hippocrates in distinguishing depression as a disorder from a nondisordered type of deep sadness or fear that could have many of the

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same symptoms but was instead a normal, proportionate reaction to serious losses. For example, Aristotle (384–322 BCE), or perhaps one of his followers, distinguished normal states of sadness from pathological disease states, stating that in depression black bile is “cold beyond due measure” so “it produces groundless despondency” (Jackson, 1986, p. 32). Here, “beyond due measure” refers to responses that are disproportionate to their circumstances, so the resulting sadness is “groundless.” In contrast, sadness stemming from losses such as the death of intimates, reversals in fortune, disappointments in attaining valued life goals, and romantic disappointments could be proportionate to their contexts and, therefore, not disordered.

Given that the symptoms of normal sadness and depressive illness could be the same, ancient physicians understood that differential diagnosis required careful exploration that went beyond the symptoms to the context of the symptoms. For example, Greek physician Aretaeus of Cappadocia (ca. 150–200 CE) explicitly separated melancholic patients who “are dull or stern, dejected or unreasonably torpid, without any manifest cause” from those who experience “mere anger and grief, and sad dejection of mind” (Jackson, 1986, pp. 39, 40). To illustrate the distinction, Aretaeus recounted his own version of the diagnostic triumph famous in ancient times of Erasistratus (304–250 BCE) physician to King Seleucus of Syria, in which Erasistratus discovered through shrewd observation that the king’s son, Antiochus, was not suffering from melancholia as his symptoms suggested, but was instead suffering from unrequited (and unexpressible) love—for his father’s young wife! As Aretaeus tells it:

A story is told, that a certain person, incurably affected, fell in love with a girl; and when the physician could bring him no relief, love cured him. But I think that he was originally in love, and that he was dejected and spiritless from being unsuccessful with the girl, and appeared to the common people to be melancholic. He then did not know that it was love; but when he imparted the love to the girl, he ceased from his dejection, and dispelled his passion and sorrow; and with joy he awoke from his lowness of spirits, and he became restored to understanding, love being his physician.

(Jackson, 1986, p. 40)

Similarly, during the same period, Galen draws this distinction in his case histories. For example, Galen describes a case in which he is unsure whether the problem lies in normal despair over some loss that is being hidden from the physician or the development of a depressive medical disorder:

I was called in to see a woman who was stated to be sleepless at night and to lie tossing about from one position into another. Finding she had no fever, I made a

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detailed inquiry into everything that had happened to her, especially considering such factors as we know to cause insomnia. But she either answered little or nothing at all, as if to show that it was useless to question her. Finally, she turned away, hiding herself completely by throwing the bedclothes over her whole body, and laying her head on another small pillow, as if desiring sleep. After leaving I came to the conclusion that she was suffering from one of two things: either from a melancholy dependent on black bile, or else trouble about something she was unwilling to confess. I therefore deferred till the next day a closer investigation of this.

(Galen, 1929, p. 213)

In addition, early diagnosticians acknowledged that variations in temperament predispose some people to more readily or intensely experience sadness or fear. However, these variations were thought to be within a normal range of reasonably proportionate responses that did not represent a disorder. Depressive disorders thus differed from normal reactions, according to tradition, because they either arose in the absence of situations that would normally produce sadness or were of disproportionate magnitude or duration relative to their provoking causes. Such conditions indicated that something was wrong in the individual, not in the environment.

## The Anatomy of Melancholy

Subsequent to early Greek and Roman medicine almost no new developments in medical thinking about melancholy occurred until the end of the eighteenth century. Robert Burton's *The anatomy of melancholy*, published in 1621, illustrates the persistence of the classical tradition. It is the most renowned of all classical discussions of melancholy and perhaps of any volume ever written about depression. Burton described three major components of depression—mood, cognition, and physical symptoms—that are still viewed as the distinguishing features of the condition. However, he insisted that melancholic symptoms are not in themselves sufficient evidence of disorder. According to Burton, only symptoms that are without cause provide evidence of disorder. As he explained in this codicil to his definition: "*without a cause* is lastly inserted, to specify it from all other ordinary passions of Fear and Sorrow." And, he noted, "signs in the mind" of melancholia included "Sorrow ... without any evident cause; grieving still, but why they cannot tell."

Burton emphasized that a propensity to melancholy was present in all men, and was a normal and ubiquitous aspect of the human condition:

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Melancholy ... is either in disposition or habit. In disposition, it is that transitory melancholy which goes and comes upon every small occasion of sorrow, need, sickness, trouble, fear, grief, passion, or perturbation of the mind, any manner of care, discontent, or thought, which causeth anguish, dullness, heaviness, and vexation of spirit.... And from these melancholy dispositions, no man living is free, no Stoic, none so wise, none so happy, none so patient, so generous, so godly, so divine, that can vindicate himself; so well composed, but more or less, some time or other, he feels the smart of it. Melancholy, in this sense is the character of mortality.

(Burton, 1621/2001, pp. 143–144)

In contrast to normal melancholy that arises naturally after people have suffered losses and disappointments and that is part of the “character of mortality,” Burton held that melancholic afflictions are “contrary to nature.” This latter condition, the disorder of melancholy, he defined as “a kind of dotage without a fever, having for his ordinary companions fear and sadness, *without any apparent occasion*” (Burton, 1621/2001, pp. 169–170).

Burton was sensitive to the wide individual variation in the nature of loss responses. He believed that a quite broad range of temperamental reactions to loss was nondisordered as long as the reactions did not become chronic or self-perpetuating. He noted: “For that which is but a flea-biting to one, causeth insufferable torment to another, & which one by his singular moderation, & well composed carriage can happily overcome, a second is no whit able to sustaine” (Burton, 1621/2001, p. 132). It is only when such normal reactions to specific events become established as an ongoing condition independent of events that Burton sees disorder:

(I)t falleth out oftentimes that these Dispositions become Habits, and ... make a disease. Even as one Distillation, not yet growne to custome, makes a cough; but continuall and inveterate causeth a consumption of the lungs: so doe these our Melancholy provocations.... This Melancholy of which we are to treat, ... a Chronicke or continue disease, settled humor ... not errant but fixed, ... growne to an habit, it will hardly be removed.

(Burton, 1621/2001, pp. 145–146)

In addition to noting normal variation in temperament, Burton was an astute observer of the extremes to which normal reactions to loss could go. He noted that the most intense painful losses included separation from friends and bereavement following loss of a loved one (“in this Labyrinth of accidental causes [of melancholy] ... loss and death of friends

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may challenge first place" Burton, 1621/2001, pp. 357–358) and compellingly described the extremes that nondisordered grief can reach:

If parting of friends, absence alone, can work such violent effects, what shall death do, when they must eternally be separated, never in this world to meet again? This is so grievous a torment for the time, that it takes away their appetite, desire of life, extinguisheth all delights, it causeth deep sighs and groans, tears, exclamations, ... howling, roaring, many bitter pangs, and by frequent mediation extends so far sometimes, they think they see their dead friends continually in their eyes, ... Still, still, still, that good father, that good son, that good wife, that dear friend runs in their minds; a single thought fills all their mind all year long, ... They that are most staid and patient are so furiously carried headlong by the passion of sorrow in this case, that brave discreet men otherwise oftentimes forget themselves, and weep like children many months together.

(Burton, 1621/2001, pp. 358–359)

Burton's magisterial work remains the most comprehensive description of depression ever compiled. Yet his rambling and disjointed style, not to mention the inconsistencies and lack of systemization that plagued his work, meant that his vast compendium was not a useful foundation for the more scientific studies of depression that would emerge in later centuries. It remained for future diagnosticians to build on Burton's work and to disentangle melancholia from other conditions.

Burton's work is clearly situated within the Hippocratic tradition. In effect, medical commentators throughout the eighteenth century relied primarily on Greek physicians, especially Galen, as authorities on depression and other mental illnesses (Simon, 1980). Likewise, the association of sadness and fear under the general melancholic umbrella persisted for centuries. The humoral theory of disease also endured in medical understandings and treatments of melancholy until the end of the seventeenth century and, sometimes, beyond then. Humoral thought was foundational not only in the culture of physicians but also in the medical lore of common people and the lay healers. Diseases resulted from imbalances between the various humors: treatments aimed to correct such imbalances and restore the body to appropriate equilibrium. Hippocratic preferences for altering lifestyles continued to prevail over more intensive medical interventions. Fresh air, exercise, good sleeping, eating, and elimination habits, and control of the passions remained prominent treatments for melancholy. Such treatments were typically intertwined with religious, magical, and folkloric methods (Shorter, 1992).

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## From the Seventeenth through the Nineteenth Centuries

A radical change in the Western intellectual tradition occurred in the seventeenth century as the inductive, empirical, and observational methods of Bacon and Newton overturned the more deductive, intuitive, and relational nature of Hippocratic methods. Notions of disease specificity also began to emerge, particularly in the work of English physician Thomas Sydenham (1624–1689). Sydenham proposed that each disease had natural forms with uniform presentations in different individuals, an idea distinct from holistic Hippocratic thought. After thousands of years of dominance by Hippocratic views of humoral imbalance, a new system arose that was based on disturbances of the brain and the nervous system (Porter, 1997).

Gradually, the capacious category of melancholia that spanned from the Hippocratics through Burton began to split into more specific manifestations. Depressive conditions were divided into two major types (Shorter, 2013). The first conditions were marked by deep mental anguish, hopelessness, complete joylessness, stupor, and suicidal thoughts and/or actions. These were likely to be chronic and recurrent and require the attention of specialized healers (then called “alienists”) who treated insane conditions.

In addition to this serious melancholia, a new category of “nervous disorders” began to emerge that viewed the nervous system as the source of health and illness, emphasizing the importance of nerves, fibers, and organs. Accordingly, the causes of nervous conditions were found in physiology, particularly brain lesions. Depressive symptoms were viewed as one component of a syndrome of “nervous disease,” “nervous illness,” “neurosis,” or, later, “neurasthenia” that referred to nonpsychotic conditions related to problems of the nervous system. The depressive component of such states was not seen as distinct from the variety of heterogeneous anxious and physiological symptoms that comprised this diagnosis. Nervous disorders encompassed anxiety, fatigue, somatic preoccupations, and obsessions (Shorter, 2013). Because these conditions were related to an organic system, they were not seen as mental problems. Nervous disorders fell under the domain of general physicians, neurologists, and spa doctors (Micale, 2008).

Melancholic and neurotic depressions were not two different points of the same continuum of severity. Instead, “There are two different kinds of depression, as different as tuberculosis and mumps; it makes no sense to lump both of them together under the general term ‘depression’” (Shorter, 2013, p. 80). By the nineteenth century these conditions were sharply distinguished by their symptoms, causes, and treatments. While alienists and psychiatrists treated melancholic patients, often within inpatient settings, nervous patients remained in the community under the purview of general physicians or specialized nerve doctors.

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The two leading diagnosticians of the late nineteenth century took sharply different approaches to depression. German psychiatrist Emil Kraepelin (1856–1926), who spent his entire career practicing in mental asylums, focused on the melancholic type of depression. He linked depression with mania under the general umbrella of manic-depressive conditions, sharply distinguishing it from his second psychotic state of dementia praecox (schizophrenia). Manic depression and dementia praecox were homogeneous and distinct entities that presumably had entirely different causes, prognoses, and outcomes (Kraepelin, 1921).

Early in his writings on classification and diagnosis, Kraepelin (1903) described melancholia as a separate disorder unrelated to manic-depressive psychosis. Subsequently, however, he was impressed by the fact that in some cases that initially looked like melancholia, eventually—often after long periods of time—there developed a manic episode. He was also persuaded by a study by Dreyfus (1907) that the nature of melancholia and of the depressive pole of manic-depressive illness is in fact qualitatively indistinguishable, and thus likely represent the same underlying etiology. Kraepelin thus eventually combined all depressive and manic-depressive mood disorders into one category that, although encompassing a variety of clinical presentations, had as its hypothesized source the same underlying pathophysiology: “Manic depressive insanity as it is to be described in this section, includes on the one hand the whole domain of so called periodic and circular insanity, on the other hand ... the greater part of the morbid states termed melancholia .... In the course of the years I have become more and more convinced that all the above-mentioned states only represent manifestations of a *single morbid process*” (Kraepelin, 1921/1976, pp. 1–2). However, with the subsequent development of treatments specific to bipolar versus unipolar illness, all the unipolar forms were united under the *DSM*’s major depressive disorder.

Kraepelin’s approach to psychiatric diagnosis is generally credited as the inspiration for the *DSM* system, so it is of interest that, unlike the *DSM* but like most of his predecessors, Kraepelin believed in the necessity of taking context into account when diagnosing depressive disorder in order to differentiate it from normal intense 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” (Kraepelin, 1915, p. 68). Kraepelin, like Aretaeus, offered case illustrations of the danger of false positives and emphasized the need to consider context—including history—to discriminate disorder from nondisorder:

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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.

(Kraepelin, 1917, pp. 199–200)

In contrast to Kraepelin, who was generally concerned with the conditions of severe, hospitalized patients, the second towering figure of the time, Sigmund Freud (1856–1939), had little concern with psychotic conditions. Instead, Freud was centrally involved with nervous conditions found in community practices. Yet Freud gave short shrift to depression, giving anxiety pride of place in his pantheon of neurotic symptoms. Freud's sole major essay on depression, "Mourning and Melancholia," focused on the distinction between the normality of grief and the disorder of melancholia:

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.

(Freud, 1917/1957)

Freud emphasized that symptoms associated with mourning are intense and are "grave departures from the normal," in the sense that grief is greatly different from usual functioning. Nevertheless, grief is not a "morbid" condition; that is, it is not a medical disorder that represents the breakdown of a biologically normal response and in fact does not require medical treatment. Medical intervention, he suggested, could actually harm the grieving person by interfering with this natural process.

By the early decades of the twentieth century, then, depression was sharply split into melancholic conditions marked by serious symptoms that were linked to psychoses and neurotic depression that was one of the psychoneuroses. Whereas melancholic depression was thought to be due to some as yet unknown brain dysfunction, nonmelancholic conditions were seen as products of various psychosocial adversities, especially the loss of a love object. The former usually required some form of inpatient treatment while the latter could be handled within outpatient settings.

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## Between Kraepelin and Freud and the *DSM-III*

Freud, along with American psychiatrist Adolf Meyer, was the major influence on the *DSM-I* (1952) and *DSM-II* (1968), the two manuals that preceded the *DSM-III*. Depression, however, bore a more Kraepelinian stamp. Much in Kraepelinian style, the *DSM-I* grouped affective disorders that were characterized by severe mood disturbances consistent with melancholia as one of the three major categories of psychotic disorders (the two others were schizophrenic and paranoid reactions) (American Psychiatric Association, 1952, p. 12). Persons receiving this diagnosis showed “evidence of gross misinterpretation of reality, including, at times, delusions and hallucinations” (American Psychiatric Association, 1952, p. 25). In contrast, these earlier manuals conceived of neurotic depression as an epiphenomenon of an underlying anxiety condition. The very first sentence of the *DSM-I* classification of psychoneurotic disorders stated: “The chief characteristic of these disorders is ‘anxiety’ which may be directly felt and expressed or which may be unconsciously and automatically controlled by the utilization of various psychological defense mechanisms (depression, conversion, displacement, etc.)” (American Psychiatric Association, 1952, p. 31). Psychoneurotic depression was thus understood as a psychological defense against anxiety.

The *DSM-II* also grouped psychotic depression with states of mania, much in Kraepelinian fashion. It defined the category of major affective disorders as follows: “This group of psychoses is characterized by a single disorder of mood, either extreme depression or elation ...” (American Psychiatric Association, 1968, p. 35). It continued to submerge depressive neurosis within the broader category of anxiety conditions, stating that “Anxiety is the chief characteristic of the neuroses” (American Psychiatric Association, 1968, p. 39). In contrast to the prominence these manuals accorded psychotic forms of depression, they viewed psychoneurotic depression as one type of defense mechanism against anxiety. During the 1950s and much of the 1960s, nonpsychotic forms of depression were largely submerged into the broader conception of psychoneuroses.

Although psychotic forms of depression were central to psychiatric theory, research, and practice before 1980, in the study of neurotic depression, numerous and often competing diagnostic systems existed and none had preeminence over the others (Grob & Horwitz, 2010). Most researchers agreed with the *DSM-I* and *DSM-II* that melancholic (or psychotic) depression—a particularly serious state marked by vegetative symptoms, delusions, and hallucinations—was a distinct type of disorder (e.g., Kiloh & Garside, 1963; Klein, 1974; Mendels & Cochrane, 1968; Paykel, 1971). Although researchers

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concurred that a separable, psychotic form of depression existed, they could not reach a consensus about the nature of nonpsychotic types of depression.

Researchers argued over whether these depressions were continuous or discontinuous with psychotic forms, on the one hand, or with normality, on the other. They disputed how many forms neurotic conditions took and even whether they had any distinct forms at all. Diagnosticians who argued for discrete types could not agree on how many types existed. Some concluded that in addition to a melancholic, psychotic type, depression had only a single neurotic type (Kiloh & Garside, 1963). Others suggested that three or more distinct, neurotic states of depression existed (Hamilton & White, 1959; Paykel, 1971; Raskin & Crook, 1976). Various classifications of depression embraced from a single to as many as nine or more separate categories (Kendell, 1976). Still others conceived of neurotic depression as more closely resembling a personality or temperament type than a disease condition (Eysenck, 1970). Nor was it known whether some milder forms of depression were early indicators of eventual psychotic forms. In addition, little consensus existed about the particular symptoms that were essential to definitions of nonpsychotic forms of depression and more disputes abounded over whether depression should be classified according to its symptoms, etiology, or response to treatments.

In 1976 the prominent psychiatric diagnostician R. E. Kendell published an article whose title accurately conveyed the situation at the time: "The Classification of Depressions: A Review of Contemporary Confusion." Kendell outlined 12 major systems of classification, most of which had little to do with the others. He concluded (1976, p. 25) that "there is no consensus of opinion about how depressions should be classified, or any body of agreed findings capable of providing the framework of a consensus." In 1979, just a year before the publication of the *DSM-III*, psychiatrists Nancy Andreasen and George Winokur (1979) likewise noted the presence of "a hodgepodge of competing and overlapping systems" in research about depression. Similar to the other major diagnoses in psychiatry at the time, opinions regarding the classification of depression at the end of the 1970s featured an extraordinarily broad range of unresolved conflicts on how best to measure this condition. Yet in 1980, responding to this period of confused debate characterized by the highly unsettled state of empirical findings and lack of definitive theory about the nature of nonpsychotic depression, psychiatry would nonetheless adopt a definitive set of symptomatic criteria for depression that has remained stable until the present.

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## Sources of the *DSM-III*

One of the 12 classifications of depression that Kendell reviewed in his 1976 article was “The St. Louis Classification” developed by a group of psychiatrists at Washington University (Horwitz, 2011). During the era in which psychodynamic perspectives dominated the psychiatric profession, the Washington University Department of Psychiatry was an outpost of traditional medically minded thinking. Led by Samuel Guze and Eli Robins, this group’s primary concern was to develop a reliable system of diagnosis that could differentiate the etiology, prognosis, and drug responses of various conditions. They developed operational criteria for 14 disorders, known as the “Feighner criteria” after the psychiatric resident who was the first author of the article that described them (Feighner, Robins, Guze, Woodruff, Winokur, & Munoz, 1972).

The Feighner criteria for depression required fulfillment of three conditions. First, patients must have a dysphoric mood marked by symptoms such as being depressed, sad, or hopeless. Second, the criteria required five additional symptoms from a list including loss of appetite, sleep difficulty, loss of energy, agitation, loss of interest in usual activities, guilt feelings, slow thinking, and recurrent suicidal thoughts. Finally, the condition must have lasted at least 1 month and not be due to another psychiatric or medical illness (which were classified as “secondary affective disorders”). Patients whose symptoms arose from preexisting mental or physical illnesses would receive a diagnosis of a secondary affective disorder.

What was the basis for the Feighner criteria of depression? In contrast to the widespread belief that a strong empirical research base underlay the diagnostic criteria leading to the *DSM-III* (e.g., Kendler, 1990; Sabshin, 1990), in fact, the evidence supporting its classification of depression was very limited. Only one of the five publications cited in the footnotes to the article provided empirical substantiation for the depression criteria (another citation refers to unpublished research by Robins and Guze). This was a study by psychiatrist Walter Cassidy and several colleagues that reported findings from a quantitative study of 100 patients called “manic-depressive” and 50 medically sick controls (Cassidy, Flanagan, Spellman, & Cohen, 1957).

The criteria of Cassidy et al. (1957) for depression required that patients “(a) had made at least one statement of mood change, including any of the following: blue, worried, discouraged, and 16 equivalent expressions and (b) had any 6 of the 10 of the following special symptoms: slow thinking, poor appetite, constipation, insomnia, feels tired, loss of concentration, suicidal ideas, weight loss, decreased sex interest, and wringing hands, pacing, over-talkativeness, or press of complaints” (Cassidy et al., 1957, p. 1535).

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Feighner himself noted that he “relied a lot on an article by Cassidy” (Kendler, Muñoz, & Murphy, 2010, p. 136) and his eponymous criteria made only four relatively small changes to these conditions, dropping constipation, adding feelings of self-reproach or guilt, expanding insomnia to encompass sleep difficulties, and combining weight loss with anorexia into one item (Kendler et al., 2010). In addition, the Cassidy diagnostic criteria did not mention any necessary duration of symptoms, perhaps because all patients in their study were hospitalized and most had symptoms that endured for more than 6 months. The Feighner criteria added the stipulation that the symptoms must last for at least 1 month, a far shorter duration than that typifying the Cassidy hospitalized sample.

Several aspects of the Cassidy criteria are noteworthy. First, all of the patients in the sample were “considered sick enough to require hospital observations, and in most cases the patients were admitted for electroconvulsive treatment” (Cassidy et al., 1957, p. 1535). The diagnosis was thus grounded in melancholic symptoms that characterized state hospital patients, which could differ substantially from psychoneurotic depression found in outpatient settings or acute psychiatric wards, not to mention untreated community populations. In addition, Cassidy et al. (1957, p. 1542) recognized the inexact nature of their criteria, stating that “The question immediately arises as to whether all these patients did, in fact, have manic-depressive disease. At present, one cannot go beyond saying that the patients had a psychiatric illness....” In particular, the Cassidy group noted the unresolved relationship of manic-depressive disease to patients with melancholia, manic-depressive psychoses, anxiety, alcoholism, and manic-depressive personality types. They clearly believed that their diagnostic criteria were highly exploratory and far from the last word on depressive diagnoses and their relationship to criteria for other diagnoses.

Likewise, the Feighner group presented their criteria as a tentative first step that awaited future validation and noted that they were “not intended as final for any illness” (Feighner et al., 1972, p. 57). Similarly, Kendell (1976, p. 25) did not place any special priority on the Feighner measurement of depression, noting that “no evidence has been offered to suggest that it is anything more than a convenient strategy.” Yet just 4 years after Kendell made this assessment, the Feighner classification of depression became almost the sole basis for the *DSM-III* diagnosis. Indeed, by 1989, the article in which the Feighner criteria first appeared was the single most cited article in the history of psychiatry (Feighner, 1989). In a remarkably short period of time the process of diagnosing depressive disorder was transformed from a contentious battle among many competing systems to the unchallenged dominance of a single classification, the *DSM-III* diagnosis of Major Depression. How did this hegemony come about?

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## The *DSM-III*

*DSM-III* formulated depression in a radically new way relative to the previous 2500 years of medical diagnosis. Its definition of major depressive disorder (MDD) required either a dysphoric mood or loss of interest or pleasure in usual activities. In addition, at least four of the following symptoms must be present nearly every day for a period of at least 2 weeks: (1) poor appetite or significant change in weight; (2) insomnia or hypersomnia; (3) psychomotor agitation or retardation; (4) decreased sexual drive; (5) fatigue or loss of energy; (6) feelings of worthlessness, self-reproach, or excessive or inappropriate guilt; (7) diminished ability to think or concentrate or indecisiveness; and (8) recurrent thoughts of death or suicidal ideation or suicide attempt (American Psychiatric Association, 1980, p. 213).

These criteria almost completely mirrored the Feighner criteria, which themselves closely resembled the original Cassidy diagnosis. The only major changes the *DSM-III* made to the Feighner criteria were to, first, exempt from diagnosis anyone who meets these symptom criteria if their symptoms are due to bereavement after the death of a loved one that lasts no more than 2 months and are not of extreme severity (American Psychiatric Association, 1980, p. 214). The earlier criteria, in contrast, contained no exceptions other than for symptoms that arose from a preexisting mental or physical condition. Second, the *DSM-III* lowered the necessary duration of symptoms from 1 month to 2 weeks and the necessary number of symptoms from six to five. Finally, the *DSM-III* abandoned the differentiation that Kendell had considered at the core of the Feighner criteria: “The most important feature of this classification is the distinction it draws between primary and secondary affective disorders” (Kendell, 1976, p. 23).

Departing from diagnostic conceptions about depression over the previous 250 years, the *DSM-III* unified depressive conditions into a single category. In contrast to the sharp split of depression into psychotic and psychoneurotic forms in the *DSM-I* and *DSM-II*, MDD embraced both unipolar psychotic and psychoneurotic forms of depression. Melancholic depression—the central depressive condition before the *DSM-III*—became a subtype of MDD (American Psychiatric Association, 1980, p. 215). Meeting criteria for the subtype required lack of pleasure or emotionally reactivity, as well as three symptoms from a list including either distinct quality of mood, symptoms of greater severity in the morning, early-morning awakening, marked psychomotor retardation, weight loss, or excessive guilt. There is an understanding that the melancholic subtype had some correspondence to the classical conceptualization of depression (Parker, 2000). However, people could qualify for a diagnosis of melancholy only if they already meet the criteria for MDD.

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Moreover, the current definition that emphasizes the presence of specific symptoms departs from the contextual tradition. The submersion of melancholia into the broader MDD category ensured its fall into obscurity (McPherson & Armstrong, 2006; Zimmerman & Spitzer, 1989). Likewise, the category of “Dysthymic Disorder (or Neurotic Depression),” which was inserted into the manual to mollify psychoanalysts, never gained traction as a well-established disorder (Bayer & Spitzer, 1985; McPherson & Armstrong, 2006). Indeed, because this diagnosis required a 2-year duration, it was inherently applicable only to persons with the most longstanding types of mood disorder. Major depression was the sole depressive diagnosis of any importance.

The MDD diagnosis that emerged in the *DSM-III* was in many ways a major achievement. It succeeded in establishing a single standard of measurement that has been almost universally adopted in psychiatric research on depression (McPherson & Armstrong, 2006). It thus facilitated communication and understanding among the research community and provided diagnostic criteria that clinicians and researchers from a variety of theoretical persuasions could use. In addition, it realized the major aim of Spitzer and his colleagues to create a reliable way of measuring depression.

Although the MDD diagnosis was a major accomplishment for research-oriented psychiatrists, it also entailed serious deficiencies. The emphasis on creating measurable and reliable diagnoses came at the expense of establishing validity. The *DSM-III* itself defined a valid mental disorder as a “behavioral, psychological, or biological” dysfunction in the individual, which the *DSM-IV* later specified “must not be merely an expectable and culturally sanctioned response to a particular event, for example, the death of a loved one” (American Psychiatric Association, 1994, p. xxi). In line with this definition, the MDD criteria excluded bereaved people from diagnoses unless they have particularly longstanding or severe symptoms (e.g., including marked psychomotor retardation or morbid feelings of worthlessness). They did not, however, exclude people whose symptoms arose from other life events such as the dissolution of a romantic relationship, loss of a valued job, or failure to achieve a long-desired goal, thus suggesting that these responses are not “expectable” or “culturally sanctioned.” This reasoning was contrary to the centuries-long understanding that such people do not have individual dysfunctions but are responding naturally to undesirable losses in their lives (Horwitz & Wakefield, 2007).

The failure to allow exclusions other than bereavement enhanced the reliability of the MDD diagnosis because clinicians and researchers might disagree on whether depressive symptoms represent appropriate contextual responses. However, as the diagnostic tradition from the Hippocratics through Freud emphasized, the use of symptoms, without regard to the context in which they develop and are maintained, combines nondisordered people whose symptoms result from some loss with those people whose symptoms either

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are inexplicable or are disproportionate to their social context. Likewise, the diagnosis encompasses conditions that are as brief as 2 weeks as well as those that persist for long periods of time. They also treat severe symptoms such as suicide attempts or feelings of worthlessness as comparable to common symptoms such as insomnia and fatigue. The result is that the MDD diagnosis encompassed an extraordinarily heterogeneous range of conditions under a single label. In effect, it ignored the pre-*DSM-III* consensus that melancholic depressions were distinct from psychoneurotic ones.

The many issues that the varied classifications of depression could not resolve before the publication of the *DSM-III* suddenly disappeared. Questions over how many distinct types of depression existed, the relationship between psychotic and neurotic forms of depression, and whether depression was best measured by dimensions or categories were all settled by fiat. Although the Feighner group framed their criteria as a tentative first step toward the eventual establishment of a reliable and valid classification scheme, the *DSM-III* adopted these criteria with few changes. Moreover, save for the removal of the bereavement exclusion in *DSM-5*, which moved even further away from the classical contextual approach, the MDD criteria remained almost intact in subsequent manuals, the *DSM-III-R*, *DSM-IV*, and *DSM-IV-TR* (American Psychiatric Association, 1987, 1994, 2000). The wholesale, and largely arbitrary, adoption of one among a number of competing ways of defining depression perhaps accounts for why—more than 30 years after its promulgation—research on depression has yet to yield any major breakthroughs in the understanding of the etiology, prognosis, or treatment of this condition (Blazer, 2005; Frances, 2013; Horwitz & Wakefield, 2007; Shorter, 2013).

## The Consequences of the *DSM-III* Diagnosis of Major Depression

The MDD category in the *DSM-III* encompassed all of the heterogeneous categories of endogenous, exogenous, neurotic, and even psychotic forms of depression that existed before 1980. MDD captured both amorphous and short-lived reactions to psychosocial problems as well as serious and chronic conditions that in the past had been associated with melancholic depression. This heterogeneous quality of the MDD diagnosis is especially consequential when the diagnosis is used outside of hospital settings.

An underlying assumption behind the use of the Feighner criteria as the foundation of the MDD diagnosis is that measures developed in hospitalized populations of severely impaired patients could be applied to all depressive conditions. Yet within hospitalized populations the symptoms that appeared in the diagnostic criteria can be assumed to be

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severe and, usually, long-lasting. Yet MDD is used not just in inpatient institutions but in all settings that require diagnoses, including general medical practice and private mental health practices and clinics. It is also the diagnosis used in epidemiological investigations among untreated community populations, in research studies, and in treatment outcome assessment. The *DSM-III* diagnosis of major depression in effect became the arbiter of what depression was in clinical and clinical research settings, community studies, and the culture at large.

The significance of the MDD criteria changed when they were applied to outpatient populations and, especially, to community populations in which their application can result in many false-positive diagnoses (Wakefield, Schmitz, First, & Horwitz, 2007). In these settings, low mood, poor appetite, insomnia, fatigue, lack of concentration, and the like are typically common responses to ubiquitous stressful experiences such as the loss of valued relationships, jobs, or goals that, as noted, even the *DSM* definition of mental disorder did not categorize as valid disorders. Indeed, Robert Spitzer, the head of the *DSM-III* task force, acknowledges that in regard to MDD: “the *DSM* is not consistent even in applying its own definition of mental disorder to the diagnostic criteria sets for specific disorders” (Horwitz & Wakefield 2007, p. viii). When diagnoses require just 2-week duration they can include many short-lived responses to stressors. Moreover, the lack of exclusion criteria other than bereavement virtually ensured that the criteria could not separate natural symptoms of sadness from dysfunctional depressive disorders.

Because the MDD diagnosis that emerged in the *DSM-III* encompassed symptoms that typified very severe and enduring symptoms as well as those that were short-lived and common signs of distress such as sadness, fatigue, sleep and appetite difficulties, or lack of concentration, it is no surprise that following the publication of the *DSM-III* depression became by far the most common diagnosis in outpatient mental health treatment. Indeed, by the beginning of the twenty-first century Major Depression constituted a full 38% of all diagnoses in outpatient settings out of the hundreds of possibilities (Olfson et al., 2002).

Major Depression also became the major target of a new class of drugs, the selective serotonin reuptake inhibitors, which came on the market in the late 1980s. Because the *DSM-III* depression criteria could encompass such a wide variety of everyday psychosocial problems, it made the most marketing sense to call them “antidepressants.” In fact, these capacious drugs were, and are still, used to treat an enormous variety of conditions including not only depression but also anxiety, obsessions, alcohol abuse, eating disorders, and a host of undifferentiated symptoms. The label “antidepressant” reinforced the popularity of the depression diagnosis because if some condition was treated with an antidepressant it must be depression.

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The overwhelming marketing success of the MDD diagnosis came at the cost of hindering scientific research. It left unresolved whether melancholic depression—the central depressive condition before the *DSM-III*—is a distinct condition or simply the most severe type of major depression (Taylor & Fink, 2006). The melding of serious melancholic conditions with transient stress-related sadness creates a roadblock in the way of finding a condition with a distinct etiology, prognosis, and treatment. It also thwarts progress in finding the possible biological roots of depression by combining distinct states that might represent brain dysfunctions with natural products of psychosocial stressors.

Pre-*DSM-III* controversies over whether depression is best viewed as a categorical or dimensional condition likewise remain unresolved. In addition, the question of how to distinguish depressive disorders from depressive personality types is unsettled. Whereas the *DSM-III* generated consensus about the operational definition of depression, questions about whether depression is continuous or categorical, how many categories it has, what its relationship to melancholic conditions is, and how it can be distinguished from normal sadness seem no closer to resolution now than they were when the *DSM-III* arose.

## Conclusions

What is striking from this brief overview of conceptualizations of depressive disorder from Hippocrates to the *DSM-III* is, first, the remarkable consistency of the symptoms that are mentioned—by and large the same kinds of symptoms that current diagnostic manuals emphasize. And, second, there is a remarkably solid and well-elaborated tradition of distinguishing disorder from normal emotion via various versions of the “with cause” versus “without cause” criterion that goes back to ancient times. Because stressful life events can precede serious and long-standing depressions, “disproportionate to cause” seems to characterize dysfunctional conditions better than the traditional term “without cause.” The entire 2,500-year record indicates an understanding that pathological depression is an exaggerated form of a normal human emotional response. Thus, diagnosticians understood that the first step in understanding a patient’s condition must be to use the relation of symptoms to their triggering causes to distinguish the normal from the disordered. The power, consistency, and rationale of the “disproportionate” medical understanding of depressive disorder form the backdrop for our own era’s radical departures in diagnostic approach. Finally, the historical tradition preceding the *DSM-III* sharply distinguished severe states of melancholic depression from less disabling psychoneurotic depression, which it often associated with cooccurring symptoms of anxiety.

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In the urgent quest for reliability, the adoption of the current depression criteria for the most part inadvertently rejected the previous 2,500 years of clinical diagnostic tradition that explored the context and meaning of symptoms in deciding whether someone is suffering from intense normal sadness or a depressive disorder. The *DSM-III* criteria, which persist into the present, also blurred the traditional distinction between melancholic and neurotic depression, calling both forms “Major Depression.” The unwitting result of this effort, especially as psychiatry turned from the serious conditions of inpatients to the far more heterogeneous conditions of outpatients and community members, was to be a massive pathologization of normal sadness. Ironically, this can be argued to have made depressive diagnosis less, rather than more, scientifically valid. The sole remnant of the “disproportionate to cause” tradition was the bereavement exclusion that remained in *DSM IV-TR*. The removal of this criterion from the diagnostic criteria in *DSM-5* indicates that far from making diagnostic progress, the recent history of the mood disorders shows significant regression in understanding the most basic of all distinctions—the difference between normal sadness and depressive disorder.

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