Major Depressive Disorder

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Major depressive disorder (MDD) is a diagnosis outlined in the American Psychiatric Association's *DSM-5*. Classified as a depressive disorder, MDD denotes persistent sadness, hopelessness, lack of energy, irritability, sleep problems, and other symptoms associated with depression. According to *DSM-5,* a diagnosis of MDD is applied when an individual has experienced one or more major depressive episodes. Estimates based on 2004 data from the National Comorbidity Survey Replication indicate that roughly 16.6% of adults, or 30 million adults, in the United States meet the criteria for a diagnosis of MDD at some point in their lives. In a given 12-month period, roughly 6.6% of adults, or 13 million adults, in the United States will experience a major depressive episode, with women twice as likely to be diagnosed as men. Depression is the leading cause of disability for people aged 15–44, with an annual public health cost estimated at between U.S.$44 and U.S.$53 billion (Smith & Smith, 2010). MDD is often associated with very poor outcome, with up to 86% of those who commit suicide having had a depressive episode within 6 months of death (Coryell & Young, 2005).

Diagnostic Criteria and Specifiers

A major depressive episode denotes the presence of at least five of the following symptoms nearly every day for a period of at least 2 weeks: depressed mood for most of the day; loss of interest in or inability to derive as much pleasure as usual from previously enjoyable activities (anhedonia); significant, unintentional change in appetite and/or body weight; difficulty falling or staying asleep, or sleeping too much; psychomotor retardation or agitation; feelings of guilt or worthlessness; trouble concentrating and/or making decisions; lack of energy; irritability; recurring thoughts of death or suicide. Either depressed mood, anhedonia, or both must be present in order for a major depressive episode to be diagnosed, and symptoms must not be better accounted for by a general medical condition, the direct effects of a substance, or a psychotic disorder or manic episode.

Specifiers can be added which describe specific features of MDD, such as whether symptoms are part of a single or recurrent episode. A recurrent episode requires that, after an initial major depressive episode, symptoms remitted for at least a 2-month interval before the current episode began. Episodes may also be specified as mild, moderate, or severe. *DSM-5* provides only loose guidelines for how each term should be applied, so MDD severity specifiers are often determined via clinical judgment. Unless formal assessment is administered, these may lack reliability, however.

If an episode of MDD is specified as severe, then it must be designated as occurring with or without psychotic features, which are defined as hallucinations or delusions. If a case of MDD is specified as severe with psychotic features, then those psychotic features can be specified as mood-congruent or mood-incongruent. Mood-congruent psychotic features are hallucinations or delusions that fit with negative, self-loathing, or morbid themes associated with depression. Psychotic features involving themes not associated with depression are considered mood-incongruent.

Subtype specifiers include MDD with catatonic features, with melancholic features, with

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atypical features, and with postpartum onset. The catatonic features specifier indicates the presence of two symptoms from a list including pronounced psychomotor peculiarities such as physical immobility, stupor, or waxy flexibility, holding one's body in bizarre postures or engaging in stereotyped movements, purposeless movements, or echopraxia. Catatonic features may also include mutism or echolalia. The melancholic features specifier is applied if a point of complete or near complete anhedonia is reached in the course of a major depressive episode. This means that the individual loses the ability to take pleasure or interest in anything, or that the individual's mood does not brighten or brightens only very slightly, when something good happens. In addition, the melancholic features specifier requires the presence of at least three signs or symptoms from the following list: depression that feels qualitatively different from the grief felt when a loved one dies; tendency for symptoms to be worse in the morning; routinely awakening too early in the morning; psychomotor retardation or agitation; excessive feelings of guilt. The atypical features specifier designates MDD in which the current or most recent depressive episode included weight gain, excessive sleeping, a feeling of leaden paralysis in the limbs lasting up to a few hours at a time, and a long-term pattern of hypersensitivity to interpersonal rejection that causes impairment. Finally, a postpartum onset specifier is applied to a major depressive episode in MDD if it begins within 4 weeks of childbirth.

Other specifiers deal with the course and chronicity of MDD. If symptoms of the current major depressive episode persist such that full criteria are met for a period of 2 years or more, then the chronic specifier is used. If, after meeting criteria for a major depressive episode, symptoms improve and no longer meet full criteria, then the specifier "in partial remission" is applied. If symptoms lift entirely and do not recur for a period of 2 months, then the specifier "in full remission" is applied. If symptoms ever return to supradiagnostic threshold levels, then MDD can be specified as recurrent, either with or without full interepisode recovery. If major depressive episodes occur during the winter months and remit during spring and summer for 2 years in a row, then the seasonal pattern specifier can be applied.

In previous editions of *DSM* (*DSM-IV-TR*, *DSM-IV*, *DSM-III-R*, *DSM-III*), MDD could not be diagnosed within 2 months of the death of a loved one. This bereavement exclusion was intended to prevent normal grief from being pathologized. In the current *DSM-5*, the bereavement exclusion is no longer part of diagnostic guidelines for a major depressive episode or MDD. Opponents of the change have argued that removal of the bereavement exclusion will lead to overdiagnosis of MDD and unnecessary treatment. Those in favor of the change argue that symptoms that initially occur as part of the grieving process can linger and develop into MDD.

Comorbidity

Comorbidity, when one meets diagnostic criteria for two or more psychological disorders, occurs in the majority of cases of MDD. Over half of people diagnosed with MDD (those included in statistics on lifetime prevalence) will also meet the *DSM-5* criteria for an anxiety disorder at some point in their lives, approximately 31% will meet criteria for an impulse control disorder, and roughly a quarter will meet criteria for a substance abuse disorder. When all diagnoses are included, comorbidity occurs in close to three-quarters of individuals diagnosed with MDD. For most of these individuals, the first major depressive episode occurs at some time after the onset of another disorder; MDD is only diagnosed first in 12.4% of cases in which it is comorbid with another diagnosis.

Comorbidity occurs because of our system of classifying psychological disorders. Comorbidity rates depend upon where categorical boundaries are placed. Diagnostic categories can group signs and symptoms on the basis of descriptive features, long-term course and outcomes, causal theories, treatments to which

they respond, or statistical analyses measuring the frequency with which they co-occur, among other ways.

Debates Concerning Definition and Description of MDD

MDD is clearly bounded and specific in the *DSM-5*. However, the relationship between MDD as a diagnostic category and psychological problems as they occur in a population is considerably less clear. It is often assumed that MDD is a diagnostic category because it represents a naturally occurring syndrome that is distinct from other syndromes. That is, that MDD as a diagnostic label is to MDD as a disorder just as blue as a label used to categorize light is to blue as it appears in a rainbow. This assumption is problematic, given the question of whether MDD exists in reality or represents our best estimate at defining a chronically depressed state. Indeed, many aspects of the *DSM-5* diagnostic criteria for MDD were included in an effort to enhance simplicity and clarity, not because they correspond to clear-cut boundaries in nature. Such aspects include the distinctions separating MDD from related diagnoses and the diagnostic cutoffs that divide MDD from the normal range of behavior and experiences.

A broader concern is that MDD may fail to capture the context of symptoms. This descriptive approach can also impact contemporary interpretations of psychological problems from previous historical periods. Proposed solutions include the adoption of dimensional systems, or other techniques that conceptualize psychological disorders as emerging from a personal network.

History

The symptoms that comprise diagnostic criteria for MDD have appeared throughout history, although not always in the forms we recognize today (Berrios, 1988, 1996; Hergenhahn, 2001; Radden, 2000; Shorter, 2005). The concept of melancholia, which appears in the writings of Greek physicians Hippocrates (460–370 BC) and Galen (130–200), is a condition resembling MDD. Hippocratic descriptions of melancholia included some symptoms that mirror contemporary diagnostic criteria for MDD.

Melancholia was considered a product of humoral theory, the belief that the body contained four fluids or humors and that mental health required a balance among them. Melancholia was believed to reflect an excess of black bile, and it could manifest in either the brain or the body. The symptoms associated with melancholia were thought to share a common cause. If there appeared to be an external reason for those symptoms (e.g. the death of a loved one) then they were not believed to reflect an excess of black bile, and thus were not melancholia. Furthermore, a distinction was made between an acute episode of excess black bile (often attributed to diet), and naturally high levels of black bile. The former resulted in melancholic disorder, while the latter resulted in a melancholic temperament or disposition and was not necessarily considered a problem. In fact, Aristotle associated the melancholic personality with wisdom and artistic genius.

Melancholia continued to appear in medical and philosophical literature after its emergence. For example, Persian physician and philosopher Avicenna, or Ibn Sina (980–1037), described obsessive fears, delusions, an "overflowing of thought," anxiety, and withdrawal from social interactions in his writing on melancholy. According to Timothy Bright (1550–1615), a physician in England, fearfulness, sadness, and despair were the primary features of melancholia.

Whereas the concept of melancholia was biologically based, the depressive state known as *acedia* was portrayed as a spiritual condition that primarily afflicted monks. In many ways acedia bears more similarity to MDD than do early depictions of melancholia, mainly because acedia was a state of dejection or even despair, apathy, weariness and lethargy, disdain for formerly valued activities, and also included shame and guilt related to the acedia itself. As

described by John Cassian (360–435), and later by Thomas Aquinas (1225–1274), acedia represented a strain or personal failure in one's relationship with God. It was also associated with the spiritual attacks of Satan or demons, an idea that was also applied to melancholia in the Medieval period.

Throughout the seventeenth and eighteenth centuries many depressive symptoms appeared as part of new diagnoses such as the vapors, the spleen, and hypochondria. Women with similar symptoms often received a diagnosis of hysteria. These conditions were termed nervous disorders or neuroses, reflecting beliefs of the time that they were due to dysfunction of the nerves. Similarly to contemporary MDD, the nervous disorders included symptoms such as prolonged sadness, irritability, lack of energy, and inability to concentrate. However, they also featured anxiety and somatic symptoms.

As the nineteenth century progressed, "depression," a word taken from general medicine where it meant disease-related lowering of activity or function, became a reference to sadness or low spirits. At first, depression was considered a symptom of other disorders such as melancholia and hypochondriasis. Gradually, the concept of depression as a disorder began to emerge. Emil Kraepelin (1856–1926) described depressive states or episodes within the category of manic-depressive insanity. These episodes were said to occur without sufficient external cause, and Kraepelin primarily focused on genetic origins and biological mechanisms. The 1906 *Nomenclature of Diseases* released by the Royal College of Physicians in London followed a Kraepelinian biological model and included melancholia as a "nervous system disease."

In contrast to Kraepelin's medical model of psychological problems, Sigmund Freud (1856–1939) viewed melancholia as arising from loss in childhood, followed by unconscious psychodynamic conflict. Freud compared and contrasted melancholia and mourning, the latter of which he described as a normal human reaction to loss. The two experiences shared a symptom profile that included sadness and psychological pain, anhedonia, and loss of motivation. However, melancholia included the additional features of guilt and self-loathing, which Freud believed were due to unconscious anger or ambivalence toward the lost figure being turned back on the self. Melancholia (but not normal mourning) could also occur without an externally obvious loss, if an individual *perceived* that he or she had lost or been abandoned by a loved one. Therefore, while Freud primarily focused on unconscious psychodynamic processes as distinguishing normal psychological reactions from those that were disordered, he did take external causes into consideration.

The dominance of psychoanalytic theory in the early to middle part of the twentieth century resulted in greater emphasis being placed on situational or psychological explanations for depression. These themes are strongly featured in the *DSM-I*, in part because of the influence of Adolf Meyer. Meyer shared Freud's views, characterizing depression as primarily a reaction to experiences. However, Meyer was less interested in unconscious psychodynamic conflict and more focused on the impact of social and situational factors. Meyer also saw room for genes to play a role in depression. It was Meyer who developed the term "depressive reaction," which is included as a diagnostic label in *DSM-I* (1952). The term was replaced with "depressive neurosis" in the *DSM-II* (1968), although the diagnosis of "psychotic depressive reaction" still appeared in the second edition. The diagnostic category of MDD was not included until *DSM-III* (1980), which marked a major shift away from the psychodynamic view of depression and toward strictly descriptive diagnostic criteria.

Etiology

Biological, psychological, and environmental theories attempt to explain the etiology (cause) of MDD. The prevailing theory combines each of these prospective causes to form a biopsychosocial model.

Biological Explanations

Genetic vulnerability and heritability. People seem to be genetically predisposed toward MDD (Levinson, 2006). Specifically, family pedigree studies indicate that if one member of a family suffers from MDD, occurrence of depression among other members of that family is twice as high as in the general population. Although shared environment could be responsible for such findings, adoption studies (which control for environmental factors) point in the same direction. For example, the rates of depression are higher in biological parents of depressed adoptees compared with biological parents of nondepressed adoptees. In addition, twin studies show that the co-occurrence of depression between monozygotic (identical) twins is two to four times as high as it is between dizygotic (fraternal) twins. This seems to be the case even when monozygotic twins are raised separately. However, although monozygotic twins share identical genes, most commonly when one twin develops MDD, the other does not. This indicates that genes may predispose a person to MDD but do not determine whether one will develop the disorder.

Neurochemical explanations. Commonly used to treat MDD, antidepressants work by increasing levels of certain neurotransmitters (e.g., serotonin, norepinephrine) in the brain. For that reason, an early hypothesis (known as *catecholamine hypothesis*) suggested that depression is caused by neurochemical imbalance—specifically, low levels of serotonin and/or norepinephrine (Ingersoll, 2010). However, this does not solely explain why people develop MDD. Just because two phenomena co-occur does not prove that they are causally linked. For example, levels of serotonin and epinephrine increase within hours after taking antidepressants, but depressive symptoms only reduce after 4–6 weeks. This implies that increases in the levels of serotonin and/or norepinephrine may be only one step in the chain of changes that occur in the brain.

Cognitive Explanations

Biased processing of emotional information. According to cognitive models of depression, MDD is characterized by negative biases in attention, interpretations, and memory (Joormann, 2009). Importantly, because such negative biases are usually evident before the first onset of depression and during a remission phase, they are likely not mere markers of the disorder but instead constitute vulnerability factors responsible for the initiation and maintenance of MDD. For example, research consistently shows that, compared with nondepressed controls, individuals with MDD remember more negative than positive information during a free recall. In a couple of studies, this negative bias in recall was associated with lower rates of remission a year later. Similarly, individuals who are depressed, remitted, or at high risk for depression (due to familial history) pay more attention to depression-related stimuli (e.g., themes of sadness and loss) than do healthy individuals. Such negative attention biases predict future onset of depression, especially in individuals experiencing high levels of stress. Finally, depressed individuals interpret ambiguous stimuli more negatively than do nondepressed people; again, studies found that such negative bias in interpretation was related to increase in depressive symptoms a few weeks later.

Learned helplessness/hopelessness and attributional style. In the 1960s and 1970s, Martin Seligman conducted experiments in which he applied a series of electric shocks to dogs without allowing them to escape or prevent the shocks. In later trials, although the dogs were able to escape the shocks, they would actually give up and become inactive. Based on these experiments, Seligman developed the *learned helplessness* theory of depression. According to this theory, the belief that one is unable to control external events (especially negative ones) increases the likelihood of a depressive episode.

People may often ask themselves who or what is responsible for negative events. Consequently, depending on their attribution style,

they may attribute these events to various causes. For example, one may attribute the events to *internal* causes (e.g., "I'm not qualified enough to find a job") or *external* ones (e.g., "the recession makes it difficult to find a job"). In addition, a person can make a *global* attribution (e.g., "I'm not qualified enough to get any job") or a *specific* attribution (e.g., "I'm not qualified enough to get a job at NASA"). Finally, one can attribute negative events to *stable* causes (e.g., "I will never be able to get a job") or *unstable* causes (e.g., "once I increase my qualifications, I will have a better chance of finding a job"). Research provides strong evidence that depression is more likely when a person's attribution style is internal, global, and stable (rather than external, specific, and unstable). This is particularly true because a person with such an attribution style (e.g., "I will never be qualified enough to find any job") is more likely to feel helpless and hopeless about his or her ability to change the future.

Self-schema, identity, and rumination. Because MDD is so strongly associated with negative bias in cognition, Beck (1967) suggested that depression is caused by dysfunctional negative thinking styles (or *cognitive distortions*). Specifically, he proposed that people who are depressed have: (a) negative self-schemas (i.e., mental representations of themselves), (b) negative views about their future, and (c) negative views about the world. This is known as *cognitive triad of depression*. For example, compared with never-depressed individuals, depressed people are more likely to rate their performance on various tasks as poor, be pessimistic about their future, and think of the world as a threatening place. More importantly, a heightened negative cognitive style is predictive of more severe, longer lasting depression and lower rates of recovery; in addition, even those who do recover display more cognitive distortions than never-depressed individuals. Thus the negative thinking style seems to be a relatively stable vulnerability factor for depression rather than an outcome of depressed mood.

Environmental/Situational Explanations

Most cases of MDD are preceded by stressful life events, especially the ones related to loss such as relationship breakup, divorce, job loss, or death of a loved one. Specifically, the likelihood of developing depression increases and the chance of remission decreases with the number of stressors experienced in the last 12 months. Moreover, onset of MDD is more likely after experiencing one severe stressor rather than several minor stressors, and prolonged, chronic stress is more likely to cause onset of depression than stress lasting for a shorter period of time.

Stressors may be present in many different forms. One example may be low socioeconomic status (SES); in particular, there is a positive relationship between rates of depression and poverty level. Although causal links between depression and SES are difficult to examine, it is conceivable that those who live in poverty have fewer resources to cope with everyday stress, and consequently are more likely to become depressed. Moreover, data indicate that SES seems responsible for different rates of depression among ethnic groups. That is, although studies report higher prevalence of MDD among African Americans and Latinos compared with other ethnic groups living in the United States, such findings seem to be at least partially due to associations between ethnic groups and SES.

Other factors associated with minority group membership may be at play. For example, members of any group that is more likely to experience prejudice, social alienation, and/or violence are more prone to depression compared with the general population. Thus, research finds higher rates of depression not only among ethnic minorities but also, for example, among members of the lesbian, gay, bisexual, transgender, queer (LGBTQ) community. This is also consistent with the fact that social support buffers the effects of stress; that

is, prevalence of depression is higher among people who experience social exclusion or have poor family support.

The influence of family dynamics on depression is also strong. An individual who is separated from his or her caregivers during childhood is more likely to suffer from MDD later in life than a person who did not experience such a separation. This finding is predicted by *attachment theory*, which suggests that early childhood experiences with primary caregivers form internal representations that are used in relationships with others. Indeed, those with insecure (i.e., avoidant or anxious – ambivalent) attachment styles are more likely to become depressed than people who formed secure attachments with their caregivers. Particularly, parenting practices that are characterized by low care, overprotection, or harsh discipline lead to heightened vulnerability to depression in children. Similarly, children of parents with negative cognitive styles tend to acquire and embrace negative styles of thinking.

Unfortunately, although there are fairly clear predictors of MDD, predictors of suicide, which is closely associated with MDD, have proven much more difficult to identify. A current research focus in clinical psychology is on how to better identify biological, cognitive, and environmental factors that can predict negative behaviors as well as diagnoses.

Assessment of MDD

One of the most comprehensive assessment tools used to diagnose MDD is the Structured Clinical Interview for *DSM-IV-TR* (SCID). The SCID has been translated into a number of languages and is used throughout the world. Administration of the SCID requires a face-to-face assessment and may take any time between 30 min and 2 hr. Depression may also be assessed or screened using less comprehensive self-report measures. For example, the Beck Depression Inventory II (BDI-II) assesses emotional (e.g., loss of pleasure), cognitive (e.g., concentration), and physical (e.g., tiredness) symptoms of depression including suicidal thoughts. In addition, the Inventory of Depression and Anxiety Symptoms (IDAS) is a recently developed 64-item scale which includes subscales of general depression (20 items) and dysphoria (10 items). Similarly to the BDI, the IDAS is characterized by excellent validity and reliability; however, compared with BDI, IDAS has better discriminant validity—that is, ability to differentiate between depression and other disorders such as anxiety.

Treatment of MDD

The first-line treatments for MDD are psychotherapy and psychopharmacology (medication). In addition, electroconvulsive therapy (ECT) is used to treat severe MDD that is unresponsive to psychotherapy or medication.

The most empirically supported psychotherapeutic treatments for MDD are cognitive behavioral therapy (CBT), behavioral therapy (BT), interpersonal therapy (IPT), and psychodynamic therapy. Mindfulness approaches, including dialectical behavioral therapy (DBT) and acceptance and commitment therapy (ACT) have also received some support.

CBT urges clients to identify negative automatic thoughts that they are having regarding themselves, the world, and the future. These thoughts, once brought into awareness through daily journal writing, are used to determine what core beliefs or schemas are underlying the way the individual is screening, coding, and evaluating stimuli that are causing the client harm. Changing a core belief such as "I am unworthy" to the belief "Everyone has worth, even people who have made mistakes," allows for information to be processed in a healthier and less depressogenic fashion, and reduces symptoms of MDD.

BT (with behavioral avoidance being the form of BT that has received the greatest amount of empirical support) places less emphasis on the cognitive elements associated with MDD, and focuses primarily on changing behavioral avoidance tendencies that predict

MDD. Using principles of behavioral reinforcement, BT urges the client to approach functional aspects of his or her life that are being avoided, and to positively reinforce these actions. This change in behavior can thus produce cognitive changes, and both lead to a decrease in MDD symptoms.

IPT and psychodynamic approaches differ from CBT and BT in that they place greater emphasis on affect and emotion, past interpersonal experiences, the therapeutic relationship, and in the case of psychodynamic therapy, on the wishes, dreams, and fantasies of the client. These therapies share a belief that examining past relationships is the best way to ensure that the negative patterns of those relationships are not repeated in the future.

Evidence supports the efficacy of all of these treatments for MDD. However, some controversy exists with regard to psychodynamic therapy, due to some studies using less rigorous methods than those pioneered by CBT researchers. No single type of treatment has shown itself to consistently outperform others in treating MDD, suggesting that the common factors shared by these approaches may be more important than their differences. This remains a hotly debated and researched topic, however.

First-line psychopharmacological treatments of MDD are antidepressant medications. These include serotonin-specific reuptake inhibitors (SSRIs), monoamine oxidase inhibitors (MAOIs), tricyclics, and atypical antidepressants. SSRIs and MAOIs function by inhibiting the removal of serotonin in and around neurons. Tricyclics enhance the activity of monoamine neurotransmitters in the brain by inhibiting their reuptake by the cells that secrete them. Atypical antidepressants function on multiple neurotransmitters, usually including serotonin, and are commonly given to supplement other antidepressants. Of these antidepressants, SSRIs are most commonly prescribed, because they do not cause potentially dangerous side effects of MAOIs and tricyclics. However, there has been a recent increase in the prescription of atypical antidepressants.

Psychopharmacological treatments are efficacious, with many findings showing that persons with MDD have better response to these drugs than to placebo treatments. However, controversy exists with regard to the severity of MDD needed to show a meaningful response. Irving Kirsch published a meta-analysis showing that when unpublished studies were included when examining the treatment efficacy of antidepressants, there was little evidence that antidepressants showed clinically significant symptom reduction in comparison to placebo for mildly or moderately depressed individuals. Only individuals who were severely depressed at baseline showed a meaningful difference, and Kirsch argued that this difference is due to a lack of response to placebo, not an exaggerated response to the drug. More research is needed to further assess this claim, but Kirsch makes a convincing argument that psychotherapy may provide a safer alternative to drugs for people with MDD.

Electroconvulsive therapy (ECT) is also still used in treating individuals with MDD who do not respond to other treatments. Despite being much maligned in popular culture, this treatment has produced meaningful change in severely disturbed patients. The treatment purportedly works by effectively resetting the system of the patient, thus allowing for a reboot of information-processing and affective structures. However, ECT's exact mechanism of change is unclear, and ECT is only advised in severe cases due to side effects that include memory and coordination loss.

SEE ALSO: Antidepressants; Antidepressant Research Controversies; Beck, Aaron T. (b. 1921); Behavior Therapies; Cognitive Therapies; Electroconvulsive Therapy (ECT) and Other Neuromodulation Therapies; Interpersonal Psychotherapy (IPT); Mood Disorders

References

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects.* New York: Harper and Row. Republished as: Beck, A. T.

(1970). *Depression: Causes and treatment*. Philadelphia: University of Pennsylvania Press.
Berrios, G. E. (1988). Melancholia and depression during the 19th century: A conceptual history. *The British Journal of Psychiatry, 153,* 298–304.
Berrios, G. E. (1996). The anhedonias. *The history of mental symptoms: Descriptive psychopathology since the nineteenth century* (pp. 332–348). New York: Cambridge University Press.
Coryell, W., & Young, E. A. (2005). Clinical predictors of suicide in primary Major Depressive Disorder. *Journal of Clinical Psychiatry, 66,* 412–417.
Hergenhahn, B. R. (2001). *An introduction to the history of psychology* (4th ed.). Belmont, CA: Wadsworth/Thomson Learning.
Ingersoll, E. (2010). An integral understanding of the etiology of depression. In S. Esbjorn-Hargens (Ed.), *Integral theory in action: Applied, theoretical, and constructive perspectives on the AQAL model* (pp. 149–171). New York: State University of New York Press.
Joormann, J. (2009). Cognitive aspects of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (2nd ed., pp. 298–321). New York: Guilford Press.
Levinson, D. F. (2006). The genetics of depression: A review. *Biological Psychiatry, 60,* 84–92.
Radden, J. (2000). *The nature of melancholy: From Aristotle to Kristeva.* New York: Oxford University Press.
Shorter, E. (2005). Melancholia. In *A historical dictionary of psychiatry* (pp. 174–175). New York: Oxford University Press.
Smith, J. P., & Smith, G. C. (2010). Long-term economic costs of psychological problems during childhood. *Social Science & Medicine, 71,* 110–115.

Further Reading

Dimidjian, S., Matell, C. R., Addis, M. E., & Herman-Dunn, R. (2008). Behavioral activation for depression. In D. H. Barlow (Ed.), *Clinical handbook of psychological disorders: A step-by-step treatment manual* (4th ed., pp. 328–364). New York: Guilford Press.
Gotlib, I. H., & Hammen, C. L. (2009). *Handbook of depression* (2nd ed.). New York: Guilford Press.
Seligman, M. E. P. (1991). *Helplessness: On depression, development, and death* (2nd ed.). New York: W. H. Freeman.