SCIENTIFIC FOUNDATIONS OF COGNITIVE THEORY AND THERAPY OF DEPRESSION

David A. Clark, Aaron T. Beck, (with Brad A. Alford)
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DEPRESSION IS the second most common psychological disorder, annually afflicting nearly 100 million people worldwide (Gotlib & Hammen, 1992). Depending on how depression is defined and assessed, lifetime prevalence rates for diagnosable major depressive disorder range from 2.6% to 12.7% in men, and 7% to 21% for women (Kessler et al., 1994; M. Weissman, Bruce, Leaf, Florio, & Holzer, 1991). The 1994–1995 National Population Health Survey revealed that 5.6% of Canadians over 18 years of age experienced a major depressive episode in a previous 12-month period (Beaudet, 1996). In their review of 20 studies conducted since 1980, Wittchen, Knauper, and Kessler (1994) concluded that the lifetime prevalence of major depression falls between 15% and 18%. Milder sub-clinical forms of depressive disorder involving too few symptoms to meet diagnostic criteria for caseness are even more common, with prevalence rates significantly higher than for major depressive disorder (Barrett, Barrett, Oxman, & Gerber, 1988; K. Wells et al., 1989). In fact, most people can attest to feelings of sadness or dysphoria that emerge as a normal human response to the frustrations, failures, and losses occurring in daily life. Because depressed mood or dysphoria is a universal experience that falls within the normal spectrum of emotion, most people fail to appreciate the serious personal, social and economic costs associated with the more severe clinical forms of depression. Presence of a depressive disorder or even a few depressive symptoms has been associated with significant reductions in physical and social functioning (Spitzer et al., 1994).

DEPRESSIVE SYMPTOMS AND DISORDERS (K. Wells et al., 1989)
As well, individuals with just a few depressive symptoms appear to be at higher risk for the development of a subsequent more severe depressive episode (Crum, Cooper-Patrick, & Ford, 1994; Horwath, Johnson, Klerman, & Weissman, 1992; K. Wells, Burnam, Rogers, Hays, & Camp, 1992). When one also considers that rates of depression appear to be on the rise especially among younger age groups 15–45 years of age (Cross-National Collaborative Group, 1992; Klerman & Weissman, 1989; Wittchen et al., 1994), it is little wonder that the diagnosis and treatment of this disorder represents one of the most important challenges facing mental health researchers and clinicians today. In this chapter, we deal with six issues that are crucial to our understanding of depressive phenomena. Although we will not be covering all the issues important in depression research, nevertheless these six topics deal with fundamental aspects of depression that must be addressed by any theoretical model of the phenomena. These issues all deal with depression at the descriptive level and have resulted in a considerable body of empirical research. The six areas of inquiry are (a) the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-*IV;* American Psychiatric Association [APA], 1994) and core symptoms of depression, (b) diagnostic heterogeneity and depressive subtypes, (c) the continuity versus discontinuity of disorders, (d) the co-morbidity of depression, (e) the course of clinical depression, and (f) negative consequences or outcomes associated with depression. This chapter is not intended to provide a comprehensive review of depression since this can be found in textbooks on psychopathology (e. g., Beckham & Leber, 1985; Maser & Cloninger, 1990; Paykel, 1992). Rather, we want to focus on important conceptual issues that must be addressed by any theory of depression. In subsequent chapters, we evaluate Beck's cognitive theory of depression in terms of its ability to adequately account for the issues and features of depression highlighted in this chapter. Let us begin then with a discussion of what is considered the cardinal manifestation of clinical depression, major depressive disorder.
CORE SYMPTOMS OF DEPRESSION DYSPHORIA AND LOSS OF INTEREST If there is one set of symptoms that psychologists and psychiatrists consider prototypical of clinical

depression, it would have to be that of major depressive episode. According to *DSM-IV* (American Psychiatric Association, 1994), a diagnosis of major depressive episode can be made only if five or more symptoms from Criterion A are present for at least two weeks. The list of possible symptoms includes depressed mood, loss of interest or pleasure, weight or appetite gain or loss, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or loss of energy, sense of worthlessness or excessive guilt, reduced concentration or indecisiveness, and recurrent thoughts of death or suicidal ideation. In addition, depressed mood or loss of interest or pleasure must be present in order to make a diagnosis of major depressive episode, and the symptom presentation must cause clinically significant distress or impairment in social, occupational, or other areas of daily functioning. Major depression would be ruled out if the symptoms were associated with a mixed episode of mania and depression, were caused by the direct physiological effects of drugs or a medical condition, or were better accounted for by bereavement. Several features of these symptom criteria are worth noting. First, *DSM-IV* considers depressed mood and loss of interest or pleasure to be core symptoms of major depression. To qualify for a diagnosis of major depression, individuals must present with one or both of these symptoms. Coyne (1994b) noted that the sustained mood disturbance in major depression can be met by either sad mood or apathy (i. e., loss of interest or pleasure in most activities). Studies have found that either of these two symptoms occur in practically all cases of clinically significant depression (A. Beck, 1967; Depression Guideline Panel, 1993). Analysis of 40,000 questionnaires collected during the 1992 and 1993 National Depression Screening Day revealed that "having difficulty doing things that one did in the past" and "no longer enjoying the things one used to do" were among the top five most prevalent symptoms in subjects reporting some level of depression (" Study Results," 1994). Coyne (1994b) noted that 10% to 15% of severely depressed patients may deny feelings of sadness and only meet mood criteria on the basis of loss of interest or pleasure. How-ever, Buchwald and Rudick-Davis (1993) found that loss of interest was present in only 75% of outpatients meeting criteria for *DSM-III* major depressive episode. Bech (1992) stated that the mood disturbance in major depression is not simply a feeling of sadness but includes feelings of apprehension, hopelessness, and helplessness. We can conclude from this that a sustained negative mood disturbance is a core defining characteristic of clinical depression and that it involves either severe dysphoria and/ or loss of pleasure or anhedonia.

SOMATIC VERSUS SUBJECTIVE SYMPTOMS IN DEPRESSION

A second characteristic of the *DSM-IV* criteria for major depression is the strong emphasis on the biological or somatic symptoms of depression and the relative neglect of the subjective experience of the disorder (Gotlib & Hammen, 1992). Seven of the Criterion A symptoms refer to the somatic, motivational, or behavioral features of depression diminished interest, weight loss, insomnia, psychomotor agitation, fatigue, inability to concentrate, and suicidality), whereas only two symptoms, depressed mood and feelings of worthlessness, refer to the subjective aspects of depression. But is this emphasis on somatic and behavioral symptoms supported by the empirical evidence? Numerous studies have found that the more subjective symptoms of depression such as depressed mood, hopelessness, and feelings of worthlessness or self-dislike are as critical or even more central to the definition of major depression than the biological symptoms such as weight loss and insomnia (A. Beck, 1967; Coulehan, Schulberg, Block, & Zettler-Segal, 1988; D. Goldberg, Bridges, Duncan-Jones, & Grayson, 1987; Lovibond & Lovibond, 1995; "Study Results," 1994). On the other hand, anhedonia, or loss of interest or pleasure, has consistently emerged as a defining symptom feature of depression (see reviews of L. A. Clark & Watson, 1989, 1991b). In our own research on a large sample of psychiatric outpatients (52% had a clinical depression) and nonclinical subjects, we found that the cognitive and subjective symptom items of the Beck Depression Inventory loaded uniquely on a depression factor; whereas the biological symptoms such as weight loss, loss of libido, and loss of appetite failed to produce significant factor loadings (D. A. Clark, Steer, & Beck, 1994; Steer, Clark, Beck, & Ranieri, 1995). However, in a study of symptom frequency in out-patients with major depressive episode, Buchwald and Rudick-Davis (1993) found that sleep disturbance, loss of energy, and appetite disturbance occurred in over 80%

of cases; whereas psychomotor disturbance and feelings of worthlessness were present in less than 70% of the depressed patients. Coryell, et al. (1995) recently reported 6-year follow-up data on individuals with an untreated major depressive disorder. For this group, anhedonia, fatigue, trouble concentrating, guilt, and insomnia were reported by at least 50% of the sample. Together, these findings indicate that there is some disagreement in the nosological literature on the centrality of the biological symptoms for a diagnosis of depression. Alternatively, somatic symptoms such as weight loss, insomnia, poor appetite, and psychomotor retardation may be indicative of an endogenous or melancholic subtype of depression (APA, 1994; Blazer et al., 1988; Grove et al., 1987; Rush & Weissenburger, 1994); or at least some of these symptoms, such as insomnia, may simply be indicative of depression severity (Casper et al., 1985; D. A. Clark, Beck, & Beck, 1994). At the very least, the strong emphasis on biological symptoms and the relative neglect of subjective symptoms in the *DSM-IV* diagnostic criteria for major depressive episode may represent an imbalance or bias that is not warranted by the findings from descriptive symptom studies of depression. As discussed in later chapters, the cognitive model considers the subjective symptoms such as a negative view of self, world, and future critical defining features of depression (A. Beck, 1963, 1967).

THE DURATION AND IMPAIRMENT CRITERIA

The mere presence of depressive symptoms is not sufficient for a diagnosis of major depression. There must also be evidence of a sustained mood disturbance (i. e., present every day or nearly every day for at least 2 weeks) which causes clinically significant distress or impairment. The impairment criteria was added to *DSM-IV* because of concerns that too many cases of *DSM-III-R* major depression were only mildly symptomatic and so not responsive to anti-depressant medication (Coyne, 1994b). Thus both the duration and impairment criteria are included to assist in distinguishing major depression from minor sub-clinical forms of depression. The importance of the duration criteria is supported by studies indicating that major depression tends to take a chronic, recurring course in a majority of patients seeking treatment (Coryell, Endicott, & Keller, 1991; Gotlib & Hammen, 1992; Keller, Shapiro, Lavori, & Wolfe, 1982; K. Wells et al., 1992). In the *DSM-IV* mood disorders field trial, 68% of the major depressive sample presented with a recurrent depressive condition (Keller et al., 1995). The recurrent nature of depression suggests that the course of the disorder may be important in differentiating clinical from nonclinical states. Coyne (1994b) has argued that duration is an important factor in diagnosable depression because sub-clinical depressive and dysphoric states tend to be briefer and more transient than major depressive episodes. Moreover, the presence of a depressive disorder, or even a few depressive symptoms, is associated with significant and persistent impairment in social and occupational functioning in both the acute and follow-up period as indicated by increased family dysfunction, reduced social activities, an increase in days lost from work, reduced annual income, decline in job status, poorer work performance, reduced educational attainment, and increased financial dependency (Broadhead, Blazer, George, & Tse, 1990; Hays, Wells, Sherbourne, Rogers, & Spritzer, 1995; Kessler, Foster, Saunders, & Stang, 1995; Mintz, Mintz, Arruda, & Hwang, 1992; M. Weissman et al., 1991; K. Wells et al., 1989). These findings suggest that the duration and impairment criteria of *DSM-IV* may be important characteristics of diagnosable major depression. However, two qualifications are in order. First, there is no evidence that assessment of impairment can be done reliably and accurately by clinicians who depend entirely on the patient's self-report. Individuals may find it more difficult to judge the degree of interference that depressive symptoms cause in their life compared with reporting on the mere presence or absence of symptoms. And second, it is still not clear that inclusion of the impairment criteria will eliminate cases of mild depression. It has been shown that even minor sub-clinical forms of depression can cause significant impairment in social and occupational functioning (Broadhead et al., 1990; Hays et al., 1995). Thus the clinical utility of the impairment criteria in *DSM-IV* major depressive episode remains to be seen. This brief discussion of the diagnostic criteria of *DSM-IV* major depressive episode leads us to the first characteristic of depression that must be addressed by any theory of the disorder:

ISSUE 1: *Clinically significant depression is characterized by a sustained disturbance in mood involving behavioral inhibition, subjective negativity and, in more severe cases, disturbance in somatic functioning.*

HETEROGENEITY OF DEPRESSION There is considerable symptom variability in the depressive experience, prompting depression researchers to acknowledge that diagnosable clinical depression consists of a heterogeneous set of disorders. Over the years, numerous classification schemes have been proposed in an effort to identify reliable and valid subtypes of depression. Some of these schemes have generated a considerable amount of research attention such as the endogenous-reactive, neurotic-psychotic, and primary-secondary distinctions as well as Winokur's Iowa classification system (see Gotlib & Hammen, 1992; Grove & Andreasen, 1992; Leber, Beckham, & Danker-Brown, 1985). In this chapter, however, we focus on four subtypes of depression that appear in *DSM-IV* as valid or provisional diagnostic categories; bipolar disorders, dysthymia, minor depressive disorder, and mixed anxiety-depressive disorder.

BIPOLAR DISORDER
One of the most widely accepted diagnostic distinctions in psychiatric nosology centers on whether depression occurs with or without a period of mania. *DSM-IV* follows this tradition, drawing a sharp distinction between unipolar depression or major depressive disorder which occurs without mania, and bipolar disorder which can take the form of alternating periods of depression and mania or hypomania. *DSM-IV* makes a further distinction between bipolar I disorder and bipolar II disorder. The primary focus in bipolar I disorder is on the manifestations of mania. It is diagnosed when there has been one or more manic or mixed episodes with or without a previous major depressive episode (*DSM-IV,* APA, 1994). Mania is described as a distinct period of abnormally elevated mood lasting at least one week and characterized by at least three of the following symptoms; inflated self-esteem, decreased need for sleep, more talkative than usual, flight of ideas, distractibility, increase in goal-directed activity, or excessive involvement in pleasurable activities. These symptoms must also be sufficiently severe to cause marked impairment in social and occupational functioning. In hypomania, these same symptom criteria are present except that the duration is shorter (at least 4 days) and the disturbance, though representing a change in functioning, is not so severe as to cause significant impairment in function. For a mixed episode, criteria are met for both a manic and major depressive episode nearly every day over at least a one-week period. A diagnosis of bipolar II disorder is made when one or more major depressive episodes occurs in the presence of at least one hypomanic episode. In bipolar II disorder, there has never been a full-blown manic or mixed episode. Considerable empirical evidence has accumulated over the years to support the diagnostic validity of the unipolar-bipolar distinction. For example, there is evidence that, compared with unipolar depression, bipolar disorder has a stronger and/or different genetic basis (Suinn, 1995), occurs at a younger age, is equally prevalent in men and women, is much less common in the general population, may have shorter acute episodes but a higher relapse rate, and has a worse overall outcome during the post-treatment follow-up period (Depue & Monroe, 1978a; J. F. Goldberg, Harrow, & Grossman, 1995; Leber et al., 1985; Perris, 1992; M. Weissman et al., 1991; Winokur, Coryell, Keller, Endicott, & Leon, 1995). However, it has been more difficult to find consistent differences in depressive symptomatology, personality, occurrence of life events, biological variables and response to treatment (Fabrega, Mezzich, Mezzich, & Coffman, 1986; Khouri & Akiskal, 1986; Perris, 1992). Thus the distinction between unipolar and bipolar disorder is based primarily on the differential course associated with each disorder rather than on symptomatic or etiologic distinctives. Although this represents a departure from the usual *DSM* approach to differential diagnosis, the weight of the empirical evidence remains with those advocating the unipolar-bipolar distinction.

DYSTHYMIC DISORDER
In *DSM-IV*, chronic, mild depressive states are represented under the category of dysthymia. The

essential characteristics of this disorder are (a) chronicity—depressed mood lasting more days than not for at least a 2-year period; (b) mild clinical presentation—presence of at least two symptoms comprising either poor appetite or overeating, insomnia or hypersomnia, low energy or fatigue, low self-esteem, poor concentration or difficulty making decisions, or feelings of hopelessness; (c) absence of recovery—in the preceding 2 years, no evidence of a symptom-free period for longer than 2 months; (d) major depressive episode not present during the first 2 years of the disturbance; and (e) clinically significant distress or impairment in social, occupational, or other important areas of functioning. Dysthymia as a chronic, subsyndromal state refers to a dispositional tendency toward dysphoria and so the concept has links to the European notion of the depressive personality (Akiskal, 1983) and the *DSM-II* concept of depressive neurosis (Phillips, Gunderson, Hirschfeld, & Smith, 1990). As noted by Akiskal, the person with dysthymia is characteristically "gloomy, introverted, brooding, overconscientious, incapable of fun and preoccupied with personal inadequacy" (p. 11). There can be little doubt that there is a subgroup of patients whose depression is not self-limiting but rather follows a chronic, sometimes lifelong course (Akiskal, 1983; Gold, 1990). Chronic depressions account for 25% to 40% of patients presenting for treatment of depression in psychiatric settings (Kocsis & Frances, 1987; M. Weissman, Leaf, Bruce, & Florio, 1988), although the lifetime prevalence rates of dysthymia (3%– 6%) tend to be lower than the 5% to 17% reported for major depressive disorder (Kessler et al., 1994; M. Weissman et al., 1988, 1991). There is also empirical evidence that compared with episodic major depression, dysthymia is characterized by lower levels of social adjustment, higher levels of chronic strain, poorer prognosis, more depressive personality traits, and more melancholic symptoms (D. N. Klein, Taylor, Dickstein, & Harding, 1988; K. Wells et al., 1992). In the *DSM-IV* mood disorders field trial, cognitive and social/motivational symptoms were more common in dysthymia than the vegetative and psychomotor symptoms (Keller et al., 1995). A number of problems are apparent with the diagnosis of dysthymia. First, the diagnostic boundaries between major depression and dysthymia are "fuzzy" making it difficult for the clinician to arrive at a differential diagnosis. Kocsis and Frances (1987) noted that the severity criteria for *DSM-III* dysthymia (3 out of 13 depressive symptoms) were too close to that of a major depression episode (4 out of 6 symptoms), thereby possibly inflating the number of double depressions (individuals with major depression superimposed on dysthymia). It is clear that major depression and dysthymia are related, and possibly overlapping, conditions given evidence of a common genetic basis, high co-morbidity rate (i. e., double depression), a substantially elevated risk for subsequent major depression among dysthymics, and the fact that the defining symptoms for each disorder are practically identical (Akiskal, 1983; Hirschfeld, 1994; D. N. Klein, Clark, Dansky, & Margolis, 1988; D. N. Klein, Taylor, et al., 1988; D. N. Klein et al., 1995; Lewinsohn, Rohde, Seeley, & Hops, 1991; K. Wells et al., 1992). Keller et al. (1995), for example, found that of the 190 individuals in the *DSM-IV* mood disorders field trial who met diagnostic criteria for dysthymia, 62% also met criteria for a current major depressive episode and 79% a lifetime diagnosis of major depression. A second problem noted for dysthymia is the probable heterogeneity of chronic depression. Akiskal (1983) distinguished between two types of chronic depression, the first labeled character-spectrum disorders and the second subaffective dysthymic disorders. The character-spectrum dysthymia is primarily a characterological pathology with poor response to medication treatment, absence of melancholic symptoms, substance abuse, and high rates of familial alcoholism. The subaffective dysthymic disorders are characterized by a greater emphasis on frequent episodes of sub-clinical endogenous depressions with personality disturbances playing a secondary role. These patients showed a better response to biological treatment, had symptoms characteristic of primary affective disorders, and had personalities that resembled the depressive personality typology. There is some empirical support for Akiskal's distinction between the two types of chronic depression (see Phillips et al., 1990). *DSM-IV* allows for a distinction between early-onset dysthymia (onset before 21 years of age) and late onset disorder (onset after 21 years of age) because early-onset dysthymia is considered to be most similar to a depressive personality disorder. A third problem centers on disagreement over the *DSM-III* decision to place dysthymia within the Axis I disorders rather than within Axis II as a depressive personality disorder (Kocsis & Frances, 1987). Whether one

considers dysthymia an Axis I or II disorder depends on which characteristics of the disorder are emphasized. Those who focus on the early-onset and chronic nature of the disorder are more likely to view dysthymia as a type of personality disorder, whereas those who emphasize the subaffective symptoms are more likely to view it as an Axis I disorder. Phillips et al. (1990) more recently argued for retaining dysthymia on Axis I and the creation of a category called depressive personality disorder on Axis II. Pepper et al. (1995) found that a significantly higher proportion of patients with early-onset dysthymia had a co-morbid Axis II personality disorder (60%) than did patients with episodic major depression (18%), again suggesting a strong link between dysthymia and characterological disorders. Kocsis and Frances (1987) have also argued that the *DSM* criteria for dysthymia may have poor content validity. They suggest that the somatic and vegetative symptoms are overrepresented and the cognitive and functional symptoms underrepresented. Descriptive cross-sectional studies have tended to support this criticism with evidence that cognitive, social, and motivational symptoms predominate in dysthymia more than the somatic and vegetative symptoms (Keller et al., 1995; Kocsis & Frances, 1987). *DSM-IV* has recognized this controversy by offering a list of alternative criteria that emphasizes the social, cognitive, and motivational symptoms. However, these are found in the provisional appendix and are intended for research purposes only. Despite the controversy over the nature, diagnosis, and relationship of dysthymia to other Axis I and II disorders, a significant number of individuals present with a chronic unremitting form of sub-clinical affective disturbance.

MINOR DEPRESSIVE DISORDER

A new diagnostic category, Minor Depressive Disorder, was introduced in *DSM-IV* as one of the provisional criteria sets that did not have sufficient empirical support for its inclusion as an official category but nonetheless deserved further study. The symptom features and duration are identical to that of major depressive episode with the exception that fewer symptoms are needed to meet diagnostic criteria (2 out of 9 with 1 of the 2 being depressed mood or loss of interest or pleasure). Exclusionary criteria include a past episode of major depression or dysthymia. In addition, the significant distress or impairment criterion found in major depression has been omitted. The importance of minor depression can be seen in the prevalence of depressive symptoms and their clinical significance. Some depressive symptoms, such as dysphoria, thoughts of death, and change in sleep and appetite, are quite common, with lifetime prevalence rates of 20% to 30% in the general population. Other symptoms, such as psychomotor slowness or loss of interest, are much less frequent (M. Weissman et al., 1991). Community studies indicate that the presence of a few depressive symptoms that are not sufficient to meet criteria for major depression can be found in 9% to 24% of the population, depending on whether interviews or screening questionnaires are utilized (Boyd & Weissman, 1981; Horwath et al., 1992). In primary care settings, the prevalence of subthreshold depression is even more common with rates ranging from 27% to 50% (Crum et al., 1994; Duer, Schwenk, & Coyne, 1988; Schulberg et al., 1985). In their review of depression research in adolescence, Compas, Ey, and Grant (1993) estimated point prevalence rates of 15% to 40% for adolescents with significant depressed mood, with 5% to 6% of these adolescents having anxious and/ or depressed symptoms in the clinical range but only 1% to 3% meeting diagnostic criteria for a depressive disorder. Minor depression or presence of a few depressive symptoms is associated with significant long term impairment in social and occupation functioning, decrease in well-being, increased utilization of health care services, and more disability days from work (Broadhead et al., 1990; Hayes et al., 1995; J. Johnson, Weissman, & Klerman, 1992; Spitzer et al., 1994). Duer et al. (1988) found that physical symptoms, chronic health conditions, recent life events, and deficiencies in social support were all significantly higher in family practice patients with elevated levels of self-reported depressive symptoms. Moreover, there is evidence that minor depression is a variant of affective disorder. Individuals with subthreshold or minor depression are at a substantially higher risk for developing a subsequent major depression (Crum et al., 1994; Horwath et al., 1992; K. Wells et al., 1992) and show a family history of depressive episode (Sherbourne et al., 1994). Zonderman, Herbst, Schmidt, Costa, and McCrae (1993) found that elevated self-reported depressive symptoms predicted the occurrence of clinical depression as well

as other psychiatric diagnoses as determined from hospitalization records taken during a 12-year follow-up period. However, Keller et al. (1995) found that only 6% of their sample of depressed patients met *DSM-IV* criteria for minor depression, calling into question the need for an additional diagnostic category. These results appear contrary to the findings of other studies, especially those conducted in primary care settings suggesting the need for a diagnostic category that captures the large number of individuals presenting with subthreshold depressive states and who are at higher risk for developing a subsequent major depressive episode.

MIXED ANXIETY-DEPRESSIVE DISORDER

A final depression category, mixed anxiety-depression, has again been introduced in the provisional diagnostic section of *DSM-IV*. This category is intended to identify individuals with only a few depressive and anxious symptoms that are not sufficient in frequency or severity to meet diagnostic criteria for an anxiety or depressive disorder. The symptoms must persist for at least one month and involve a dysphoric mood with four or more of the following symptoms:

- Difficulty concentrating.
- Sleep disturbance.
- Fatigue or low energy.
- Irritability.
- Worry.
- Being easily moved to tears.
- Hypervigilance.
- Anticipating the worst.
- Hopelessness.
- Low self-esteem or feelings of worthlessness.

In addition, the symptoms must cause significant distress or functional impairment, with exclusionary criteria being a previous episode of major depression, dysthymia, panic disorder, or generalized anxiety disorder. Empirical support for this diagnostic category is still in the preliminary stage. In their review of community, psychiatric, and primary care studies, Katon and Roy-Byrne (1991) concluded that a large subgroup of individuals have a mixture of anxious and depressive symptoms that are not sufficient to meet caseness for an anxiety or depressive disorder. Furthermore, this subthreshold anxiety and depression is associated with decrements in social and occupational functioning, and places individuals at higher risk for a more severe major depressive or anxiety disorder. A *DSM-IV* field trial for mixed anxiety-depression was recently conducted with 666 patients drawn from five primary care medical sites and two psychiatric outpatient facilities (Zinbarg et al., 1994). Analysis of self-report and interview symptom measures revealed a significant number of patients with subdefinitional threshold affective symptoms. These cases, diagnosed Anxiety NOS and Depression NOS under *DSM-III-R* criteria, showed a symptom profile of nonspecific anxious and depressive symptoms. They were best discriminated from subjects with no psychiatric diagnosis by general distress, or negative affect symptoms rather than more specific anxiety or depressive symptoms. Furthermore, there was some evidence that this milder, nonspecific mixed anxiety-depression took a chronic course and was associated with significant impairment in functioning. Based on these findings, the authors proposed the current *DSM-IV* diagnostic criteria for mixed anxiety-depression. The relation between this milder, mixed disturbance and syndromal anxiety or depression is currently unknown. We have discussed four subtypes of affective disorder that, along with major depressive disorder, have a varying amount of empirical support in the research literature. Regardless of which system of subclassifying depression is accepted, depression is not a unitary construct. The cognitive model recognizes this symptom and diagnostic diversity in depression, but considers a core cognitive dysfunction evident to a greater or less degree in all types of depression (Haaga et al., 1991). This, then, brings us to the second characteristic of depression that must be addressed by any theory of the disorder:

ISSUE 2: *Depression consists of a heterogeneous group of disorders that vary in severity, chronicity, and clinical presentation.*

DIMENSIONAL VERSUS CATEGORICAL PERSPECTIVES ON DEPRESSION

One debate at the core of any conceptualization of psychopathology is whether psychological disturbance varies in degree or in kind. Researchers in depression have been particularly divided on this issue, with some advocating a dimensional or continuum view of depression, and others advancing a categorical or discontinuous perspective on depressive states. The dimensional viewpoint argues that depression lies on a continuum of severity, with normal depressed mood and minor depressive states at one end of the pole and the more severe clinical states, such as major depressive episode, at the other end. Thus the nonclinical, milder states of depression form a continuum with clinical, diagnosable depression, differing only in the severity and persistence of the depressive symptomatology. The categorical perspective, on the other hand, argues that clinical or diagnosable depression is qualitatively different from subthreshold depression and normal dysphoria. Both of these views have far-reaching implications for how depression research is conducted. Those adopting a dimensional view argue that research on milder depressive states in nonclinical samples, or on subjects who report an increase in depressive symptoms on self-report depression measures, may be of relevance to clinical depression (A. Beck, 1991; Flett, Vredenburg, & Krames, 1997; Seligman, 1978; Vredenburg, Flett, & Krames, 1993). Researchers holding a categorical viewpoint argue that research on nonclinical subjects who produce elevated scores on self report depression measures is of questionable relevance or generalizability to clinical depression. A reason cited for the lack of relevance is the postulated discontinuity between clinical and nonclinical depression (Coyne, 1994b; Coyne & Downey, 1991; Depue & Monroe, 1978b; Gotlib & Hammen, 1992).

EVIDENCE FOR DISCONTINUITY

Several reasons are cited by those who hold the categorical position. One of the most compelling arguments for discontinuity is the apparent difference in incidence or prevalence rates between mild depression or dysphoria, as assessed by self-report depression measures like the Beck Depression Inventory, and the rates of diagnosable depression obtained with diagnostic interview schedules (Coyne, 1994b; Flett, Vredenburg, et al., 1997; Vredenburg et al., 1993). Coyne (1994b), in particular, has argued forcefully that high scores on self-report depression measures are not necessarily indicative of major depression (see also Compas et al., 1993). He contends that depression scores may also be elevated for other reasons such as the presence of physical symptoms, side effects of medication, high negative affectivity or neuroticism, or other psychopathological states such as anxiety or substance abuse. Coyne (1994b) cites a number of studies in which the majority of subjects having elevated scores on the Beck Depression Inventory (BDI) or Center for Epidemiologic Studies Depression Scale (CES-D) were found not to have a diagnosable depression when followed up with a clinical interview (Coyne, Schwenk, & Smolinski, 1991; Deardorff & Funabiki, 1985; Fechner-Bates, Coyne, & Schwenk, 1994; Myers & Weissman, 1980; Oliver & Simmons, 1984). Gotlib and Hammen (1992) noted that most people who show mild, transient symptoms of depression following a stressful experience do not become clinically depressed. Coyne (1994b) stated that one reason for the low positive predictive value found for measures like the BDI and the CES-D is the differential base rates for depressive symptoms and disorders in the normal population. Whereas some depressive symptoms, such as sadness or dysphoria, are relatively common, rates of diagnosable depression are comparatively quite rare. Thus self-report measures consistently overestimate depression in the normal population resulting in high sensitivity but low specificity. Furthermore, a number of correlational and factor analytic studies have found that depression measures correlate very highly with measures of negative affect and other psychological states (i. e., anxiety, anger) prompting some to conclude that self-report depression measures may be assessing general nonspecific distress or negative affectivity rather than specific depressive states (L. A. Clark & Watson, 1991b; Feldman, 1993; Gotlib, 1984;

Meites, Lovallo, & Pishkin, 1980). More recently Flett, Vredenburg, et al. (1997) questioned whether the high false positive rates for self-report depression measures can be taken as evidence for the underlying discontinuity of dysphoria and diagnosable depression. They noted that a high false positive rate is not unique to self-report depression measures but instead is a general feature of psychopathological self-report measures. High rates of false positive have been reported for questionnaire measures of eating disorders, panic disorder, psychosis proneness, obsessive-compulsive disorders, and personality disorders. In addition, Flett and his colleagues argue that the discrepancy between self-reports and diagnoses may be due to methodological problems with the instruments themselves. Thus they conclude that the high false positive rate for self-report depression measures may be due to limitations in self-report symptom methodology and so cannot be assumed to be a demonstration of discontinuities in the construct under investigation, in this case depression. A second source of evidence cited for discontinuity is the apparent distinctiveness in symptom presentation between diagnosable depression and milder states of nonclinical or "normal" depression. Depue and Monroe (1978b), for example, concluded from their review of the few studies available at that time, that behavioral, somatic anxiety, and the other somatic symptoms of depression best distinguished diagnosable depression from normal depression and unhappiness (see also Coyne & Gotlib, 1983). Nonclinical depression, or dysphoria, on the other hand, was best characterized by unhappiness, sadness, and loneliness. L. A. Clark and Watson (1991b) also concluded that milder nonclinical states of depression may be primarily characterized by the more general nonspecific symptoms of negative affectivity (i. e., a state of worry, unhappiness, distress, anger, tension, low self-worth) whereas diagnosable depression is distinguished by symptoms considered specific to depression such as anhedonia, loss of interest and pleasure, or low positive affect (i. e., loss of pleasurable engagement). In the Buchwald and Rudick-Davis (1993) study, patients with major depressive episode (MDE) were distinguished from the non-MDE patients by the vegetative symptoms of depression as well as loss of energy and thinking difficulties. The non-MDE subjects reported at least two weeks of sadness or loss of interest but failed to present with at least four other depressive symptoms in order to meet diagnostic criteria. As noted previously, Zinbarg et al. (1994) found that patients diagnosed with a *DSM-III-R* diagnosis of depression NOS (patients with subdefinitional threshold affective symptoms) were characterized by elevated scores on a negative affect scale but not on the anhedonia/ low positive affect, subjective anxiety, or physiological arousal scales. In a community study of adolescents, Gotlib, Lewinsohn, and Seeley (1995) found that individuals with diagnosable depression were distinguished from participants with high CES-D scores but no depressive disorder (false positives) by anhedonia, weight change, sleep difficulties, indecisiveness, and suicidal ideation. In their review paper, Compas et al. (1993) concluded that the presence of somatic and vegetative symptoms in adolescents with depressive disorders distinguished them from adolescents with merely depressive symptoms or a dysphoric mood state. Empirical evidence for a distinctive symptom profile for clinical versus nonclinical depression has been inconsistent at best. Vredenburg et al. (1993) highlighted a number of specific methodological problems with the early empirical studies cited by Depue and Monroe (1978b). Other studies like those of Zinbarg et al. (1994) and Buchwald and Rudick-Davis (1993) may not be particularly relevant to this issue because their symptom comparisons were made on clinical samples only rather than between clinical and nonclinical groups. Flett, Vredenburg, et al. (1997), in a detailed and thoughtful critique of this literature, concluded that the bulk of the evidence comparing symptom presentation at varying levels of depression favors the continuity position. For example, although Gotlib et al. (1995) did report symptom differences between the diagnosed depression and the false positive groups, Flett, Vredenburg, et al. reanalyzed the information provided in this study and found a substantial degree of similarity in the rank order of the nine depressive symptoms of the two groups. As described under "Evidence for Continuity," some studies have found considerable similarity in symptom expression between clinical and nonclinical depression. A third argument for discontinuity centers on whether the correlates or risk factors of dysphoria and diagnosable depression are different. The following factors appear to differ between nonclinical and clinical depression: (a) poverty increases the risk for dysphoria and depressive symptoms but not major depressive episode; (b) minor and major life events both appear related to

increases in depressive symptoms, but only severe, relatively infrequent life events that require long-term adjustment are associated with diagnosable depression; (c) chronic stressors such as unemployment or caring for a handicapped child are associated with depressive symptoms but not depressive disorder; and (d) gender differences are apparent with twice as many women suffering major depression as men, whereas elevated scores on self-report depression measures assessing dysphoria fail to show this gender difference (Coyne, 1994b; Coyne & Downey, 1991). The study by Lewinsohn, Hoberman and Rosenbaum (1988) deserves special mention because it has been frequently cited (i. e., Gotlib & Hammen, 1992) as providing strong empirical evidence for differential correlates or risk factors for depressive symptoms and disorders. In this study, 998 community adults returned a mail-out battery of self-report measures at Time 1 and then completed a diagnostic clinical interview 8 months later at Time 2. Individuals were considered to have a depressive disorder if they met Research Diagnostic Criteria for major, minor, or intermittent depressive disorder based on the SADS clinical interview at Time 2. Demographic, symptom, life event, social support, cognition, and attributional variables were correlated with Time 1 CES-D scores and a dummy variable indicating whether or not a subject had diagnosable depression at Time 1. Age, sex, and past history of depression had a stronger association with a diagnosis of depression at Time 2, whereas the cognition and attribution variables had a stronger relationship with CES-D scores. The authors concluded that although diagnosable depression and depressive symptoms as measured by elevated CES-D scores have a common core of nonspecific negative affect, each "also carries with it unique characteristics" (p. 261). There are, however, a number of problems with this study. First, the authors' conclusions are based on subjective judgments of differences in the magnitude of the correlations rather than on statistical tests to determine whether the differences in the correlates of CES-D scores and diagnosis are indeed significant. This subjectivity allows greater opportunity for bias to enter into the interpretation of the results. For example, four out of the five attribution correlations were below .30 indicating that these variables had little association with either depression scores or diagnosis. Age correlated -. 14 with depression diagnosis and -. 09 with CES-D scores. We question whether this difference is as meaningful as the authors seem to think. Second, the authors provided no adjustment for Type I error rate. With less than half of the correlations with CES-D and depression diagnosis different, it is difficult to know the extent to which the differences that did emerge were simply due to chance. At the very least, the authors have over interpreted the differences and underestimated the considerable degree of similarity in the social correlates of dysphoria and diagnosable depression. Flett, Vredenburg, et al. (1997) also raised statistical problems with studies that compare correlations with continuous (e. g., CES-D) versus dichotomous (e. g., presence versus absence of diagnosable depression) criterion variables. Differences between the correlates of continuous and dichotomous variables could be due to lower statistical power because of the restricted range of the dichotomous variable, measurement error (i. e., structured diagnostic interviews can have interrater variability), or the need to use different statistical procedures with dichotomous variables. They conclude, "These statistical differences make it exceedingly difficult to draw conclusions about the continuity issue in terms of the correlates of distress versus depression" (Flett, Vredenburg, et al., 1997, p. 407). Finally, a number of additional arguments have been made by advocates of discontinuity: (a) differential rates of duration exist, with dysphoria and depressive symptoms being more transient and less persistent than diagnosable depression; (b) diagnosable depression is associated with greater functional impairment in daily living than nonclinical and minor depression; and (c) depressive symptoms as measured by self-report measures predict other psychiatric disorders in addition to depression suggesting that nonclinical depressive states may be more accurately characterized as a general vulnerability factor (Coyne, 1994b; Tennen, Hall, & Affleck, 1995; Zonderman et al., 1993).

DEPRESSIVE SYMPTOMS AND DISORDERS EVIDENCE FOR CONTINUITY

Support for the continuity or dimensional perspective on psychological disorders such as depression comes from two general avenues. First, criticisms have been raised with categorically based classification schemes, such as *DSM,* which attempt to define the distinct attributes of

disorders so that diagnostic boundaries can be established between cases and non-cases, or between various types of disorders. These criticisms have led to proposals for a more dimensional approach to psychiatric classification. And second, empirical studies have investigated the relationship between levels of depression severity (mild, moderate, or severe) or have compared clinical and nonclinical depressive states. These studies, then, provide the empirical basis for the dimensional viewpoint. In a review and critique of current psychiatric classification, L. A. Clark, Watson, and Reynolds (1995) discuss three issues that challenge a categorical view on psychopathology. First, they state that the high rate of co-morbidity across Axis I disorders complicates the establishment of distinct categories of disorder. They note that in depression, 75% of cases involve some degree of co-morbidity making the occurrence of pure cases of depression quite rare. Second, the considerable degree of within-category heterogeneity again threatens categorical diagnoses. In major depression, patients must have either sad mood or loss of interest plus any four of the remaining symptoms. This means that individuals with different clinical presentation can nonetheless meet diagnostic criteria for major depression. L. A. Clark et al. (1995) note that the attempt to deal with this heterogeneity by creating subtypes ends up more closely resembling a dimensional rather than a categorical classification scheme. This is nowhere more apparent than with depression, where we see the introduction in *DSM-IV* of subtypes such as dysthymia, adjustment disorder with depressed mood, minor depressive disorder, recurrent brief depressive disorder, mixed anxiety-depressive disorder, and depressive personality disorder. And third, the authors point out that the large number of atypical and subdefinitional threshold cases calls into question the validity of a categorical classification (see also H. Eysenck, Wakefield, & Friedman, 1983). This was evident in the study by Zinbarg et al. (1994), which had no difficulty in identifying a large number of primary care and outpatients who were diagnosed with depression NOS (i. e., presence of a few depressive symptoms that falls short of diagnostic criteria). The dimensional perspective on depression also has support from a number of empirical studies. Flett, Vredenburg, et al. (1997) concluded from their review of this literature that there is considerable empirical support for phenomenological (i. e., similar symptom presentation) and etiologic (i. e., minor depression confers greater risk for subsequent major depression) continuity in depression. The difficulty in consistently finding distinct subtypes of depression is also consistent with the continuity viewpoint. However, Flett and colleagues acknowledge that recent psychometric studies of self-report depression measures indicate that the items may not assess the full range of depressive symptom expression nor do they all necessarily follow a continuous distribution with increasing levels of severity. This body of data may be the strongest empirical support for discontinuity in depression (see Santor, Ramsay, & Zuroff, 1994; Santor, Zuroff, Ramsay, Cervantes, & Palacios, 1995). In this section, we present a brief summary of the empirical evidence for continuity. The reader is referred to Flett, Vredenburg, et al. for a more detailed discussion. As noted, studies have found that individuals with minor depression or a few depressive symptoms are at higher risk for developing a subsequent major depressive episode (Crum et al., 1994; Gotlib et al., 1995; K. Wells et al., 1992) although they seem to be at higher risk for developing other psychiatric disorders as well (Gotlib et al., 1995; Zonderman et al., 1993). Horwath et al. (1992), for example, found that more than 50% of first-onset cases of major depression have had a prior history of depressive symptoms. G. W. Brown, Bifulco, Harris, and Bridge (1986) in a one-year follow-up study of a community sample of inner London working-class women with at least one child living at home, found that presence of sub-clinical anxious and/ or depressive symptoms did predict a subsequent depressive disorder. However, the authors also found that low self-esteem, ongoing life difficulties, and severe life events were significant predictors of depression onset and so raised the possibility that depressive symptoms predict depressive disorder mainly through their association with ongoing difficulties and severe matching life events. Flett, Vredenburg, et al. (1997) referred to this as etiologic continuity and concluded that it has substantial support in the empirical literature. Second, presence of a few depressive symptoms is associated with considerable functional impairment that appears to differ from major depression only in degree rather than kind (Broadhead et al., 1990; Spitzer et al., 1994; K. Wells et al., 1989). In a longitudinal community study of adolescents, Gotlib et al. (1995) found that subjects with elevated

self-report depression scores but no diagnosable depression (false positives) did not differ significantly from individuals with diagnosable depression on most variables of psychosocial functioning (i. e., on 14/ 20 variables). The two groups did differ on internalizing and externalizing psychopathology, suicidal ideation, physical health and illness, self-esteem, and social sup-port of friends. Third, in another study, individuals with subthreshold depression were found to have a 41% rate of family history for depression, which is only slightly lower than the 59% rate found for individuals with major depression (Sherbourne et al., 1994). The authors concluded that subthreshold depression appears to be a variant of affective disorder. Fourth, it is not at all clear that minor depressive states or depressive symptoms are more transient than major depressive disorders. Katz and Yelin (1993) assessed depressive symptoms in 822 rheumatoid arthritis patients over a 4-year period using the Geriatric Depression Scale. Presence of depressive symptoms in one year greatly increased the probability of depressive symptoms in future years, with a person with depressed symptoms in 1986 six times more likely to report depressive symptoms in 1990 than nondepressed individuals. As noted, the persistence of depressive symptoms can be seen in the Zonderman et al. (1993) study where the presence of depressive symptoms constituted a significant risk factor for depressive and other psychiatric disorders over a 12-year follow-up period. Also, Gotlib et al. (1995) found that adolescents with a diagnosable depression and those with an elevation in self-reported depressive symptoms but no diagnosable disorder failed to differ in the duration of their depressive symptoms. Coyne, Gallo, Klinkman, and Calarco (1998) found that their sample of distressed primary care patients had elevated scores on the Hamilton Rating Scale of Depression that remained relatively unchanged over a 4.5-month period. These findings support Flett, Vredenburg, et al. 's (1997) contention that mild sub-clinical depressive states are not always brief and transient, and that those associated with a persistent mood disturbance of at least 2 weeks' duration are more similar to major depression than sub-clinical depression without persistent sadness or dysphoria. A fifth consideration is the degree of similarity in symptom presentation between clinical and nonclinical depressive states. By definition, individuals with diagnosable depression will have a greater number of depressive symptoms at higher levels of severity or intensity than individuals with subthreshold depression. However, it has not been consistently shown that major depression has unique symptom characteristics that distinguish it from subthreshold depression. Many studies have found more similarities than differences in the symptoms reported by those with clinical and nonclinical depressive states (e. g., Fechner-Bates et al., 1994; M. Weissman, Prusoff, & Pincus, 1975). In the factor analytic studies of the BDI and Beck Anxiety Inventory (BAI) referred to earlier, we found that the structure of self-reported anxiety and depressive symptoms was very similar in a large sample of primarily anxious and/ or depressive disorder outpatients and a sample of nondepressed college students (D. A. Clark, Steer, et al., 1994; Steer et al., 1995). Cox, Borger, Enns, and Parker (1997) compared the covariance matrices of 101 adult outpatients with *DSM-IV* major depression and 175 dysphoric undergraduate students (BDI score greater than 9) on the BDI and the *DSM-IV* Based Depression Scale (DDS). The pattern of correlations for both the DDS and BDI were virtually identical in both samples. The clinical sample did report significantly higher endorsement of suicidal ideation items, although 57% of the dysphoric students reported at least moderate levels of distress and lifestyle interference. The authors concluded that the symptom and impairment differences between their samples was more quantitative than qualitative in nature. Finally, severity of depression has been found to be an important factor in determining the nature and course of depression (Goethe, Fischer, & Wright, 1993; Gold, 1990). Severity and number of depressive symptoms are significantly related to the onset of future depressive episodes (e. g., Lewinsohn et al., 1988; Zonderman et al., 1993) and to relapse rates (Coryell et al., 1991). In a study of depressed psychiatric inpatients, depressed and nondepressed medical patients, and nondepressed healthy controls, self-report and clinician rating scales of anxious and depressive symptoms showed a linear progression in symptomatology that depended on the level of severity (D. A. Clark, Cook, & Snow, 1998). Thus depressed psychiatric inpatients scored highest on all the symptom scales, followed by the depressed medical patients, then the nondepressed medical patients, and finally the nondepressed normal controls. This was found for the negative affect, depressotypic cognition,

anhedonia, neurovegetative, and low positive affect symptom measures. These results support the continuity model in postulating symptom intensity or severity as an important variable in depression. In fact, it is incumbent on the advocates of discontinuity to ensure that symptom differences between clinical and nonclinical states is not due simply to a continuous variation in symptom severity.

IMPLICATIONS OF THE CONTINUITY/ DISCONTINUITY DEBATE

We have provided an extended discussion of the continuity versus discontinuity debate in depression. Not only is this issue fundamental to our understanding of psychopathology in general and depression in particular, but it determines how we diagnosis, assess, and conduct research on depression. Those who assume the discontinuity of disorders will feel more comfortable with categorical classification systems such as *DSM-IV.* They will consider structured clinical interview schedules that require one to count the number of criterion symptoms present based on dichotomous ratings of the presence or absence of symptoms as the only true and valid method for assessing clinical disorders. And they will abhor research involving the administration of continuous symptom measures to nonclinical samples, claiming that it has no relevance to true clinical depression. But as we have seen, the case for the discontinuity of depression is not convincing. There is now a considerable amount of empirical support for the continuity or dimensional view of depression. No doubt, the moderate standpoint adopted by Flett, Vredenburg, et al. (1997) is more in tune with reality. They advocate a complex view in which depression may show continuity in some spheres but discontinuity in other domains. As discussed in Chapter 4, the cognitive model takes a strong dimensional perspective on depression (see also A. Beck, 1991). The model assumes that psychopathological states represent extreme or excessive forms of normal cognitive, emotional, and behavioral functioning. As is evident in our presentation of the empirical research on the cognitive model, we include studies on depressed mood in normals, sub-clinical or analogue depression samples, and clinically depressed patient groups because we concur with Flett, Vredenburg, et al. (1997) that there is substantial evidence in favor of the continuity of depression at least at the phenomenological and etiologic levels:

ISSUE 3: *Depressive states vary on a continuum of severity with milder, non-clinical affective symptoms at one end and the more severe clinical affective disorders at the other end.*

COMORBIDITY AND DEPRESSION

The importance of depression as a serious and pervasive psychological condition is also evident in its high rate of co-occurrence with other types of psychiatric and medical conditions. Referred to as co-morbidity, researchers are just beginning to understand the frequency and diagnostic significance of co-morbid conditions. Depressive disorders can be found in alcohol and drug abuse, anxiety disorders, eating disorders, somatization disorder, personality disorders, and chronic medical disorders to name but a few conditions (Canadian Mental Health Association, 1995; Depression Guideline Panel, 1993). Maser and Cloninger (1990) argued that the term *comorbidity* should be reserved for the co-occurrence of syndromes or disorders, and should not be applied to the co-occurrence of symptoms. Within the co-occurrence of disorders, however, there are different types of co-morbidity. There is pathogenic co-morbidity in which two disorders have a common underlying etiology, or prognostic co-morbidity in which one disorder can develop into a second disorder (Kendall & Clarkin, 1992; Maser & Cloninger, 1990). Our discussion in this section focuses on diagnostic co-morbidity or the cross-sectional co-occurrence of two disorders in the same individuals. Co-morbid rates among disorders are very influenced by the characteristics of the diagnostic classification system. Although anxiety and depression have a high co-morbidity rate, this was unrecognized in *DSM-III* because of the use of diagnostic hierarchical exclusionary criteria. In *DSM-III,* depression occupied a higher position than anxiety disorders. Consequently, patients who met diagnostic criteria for depression and anxiety were only diagnosed as depressed. With *DSM-III-R* and then *DSM-IV,* the rates of co-morbid conditions have steadily increased because of (a) decreased emphasis on hierarchical exclusionary criteria, (b) an increase in possible

diagnoses, (c) overlapping nonspecific symptom criteria across different disorders, (d) a tendency to lower the threshold for some diagnoses, and (e) defining different diagnostic categories from a single continuum of pathology (Frances, Widiger, & Fyer, 1990; Maser & Cloninger, 1990).

COMORBIDITY OF ANXIETY AND DEPRESSIVE DISORDERS

As noted, the existence of "pure" depressive states may be quite rare, with up to 75% of depressed patients showing co-morbidity with other psychiatric conditions (Canadian Mental Health Association, 1995; L. A. Clark et al., 1995). However, it is the co-morbidity between anxiety and depressive disorders that has received most of the empirical attention because roughly 50% of clinically depressed or anxious individuals may have a coexisting anxious or depressive disorder (L. A. Clark, 1989). Numerous studies have demonstrated a high co-morbidity rate for anxiety and depression depending on sample characteristics (i. e., community, outpatient, or inpatient samples) and whether co-morbidity estimates are based on current or lifetime diagnoses (for reviews, see Alloy, Kelly, Mineka, & Clements, 1990; Brady & Kendall, 1992; T. A. Brown & Barlow, 1992; L. A. Clark, 1989). In the Philadelphia Center for Cognitive Therapy outpatient sample, 37% to 42% of patients with a principal diagnosis of major depression and 29% to 47% with dysthymia met diagnostic criteria for a secondary anxiety disorder, the most common being generalized anxiety or social phobia (D. A. Clark, Beck, et al., 1994; Sanderson, Beck, & Beck, 1990). A co-morbid depressive disorder was evident in 58 (37%) patients with panic disorder and 40 (51%) individuals with generalized anxiety disorder (D. A. Clark, Beck, et al., 1994). However, T. A. Brown and Barlow (1992) reported much lower co-morbidity rates for depression among their outpatients with a principal diagnosis of panic disorder (16%), generalized anxiety (29%), or social phobia (20%), although 45% of a small sample of patients with major depression (n = 11) were co-morbid for generalized anxiety (Di Nardo & Barlow, 1990). Much lower co-morbidity rates for anxiety and depression are found when a different diagnostic classification of depression is used such as the Research Diagnostic Criteria (Rohde, Lewinsohn, & Seeley, 1991). Based on the NIMH Epidemiologic Catchment Area (ECA) study, Regier, Burke, and Burke (1990) reported lifetime prevalence rates for co-morbid anxiety of 43% for those with affective disorders, and a rate for co-morbid depression of 25% for individuals diagnosed with a principal anxiety disorder. Results from the more recent National Co-morbidity Survey revealed that 79% of individuals with lifetime disorders were co-morbid for two or more disorders (Kessler et al., 1994). Comparable high rates of co-morbid anxiety and depression have also been found among children and adolescent clinical samples (Brady & Kendall, 1992). In fact, anxiety and depression co-morbidity may be the highest among severely disturbed groups such as psychiatric inpatients. A number of studies have reported high correlations between measures of anxiety and depression, prompting some researchers to conclude that these symptom measures do not assess distinct anxiety and depression constructs but rather a single common general distress or negative affect dimension (e. g., L. A. Clark & Watson, 1991a; Dobson, 1985; Feldman, 1993; Gotlib, 1984; Ollendick & Yule, 1990). Other studies, though, have shown that self-report measures like the BDI and BAI can distinguish between anxiety and depression (D. A. Clark, Beck, & Stewart, 1990; D. A. Clark, Steer, et al., 1994; Cox, Swinson, Kuch, & Reichman, 1993; Lovibond & Lovibond, 1995; Steer et al., 1995). One reason posited for the high co-morbidity of anxiety and depression as well as the high correlation between anxiety and depression symptom measures is the overlap in many of the symptoms (and consequently item overlap) that define these two syndromes (Brady & Kendall, 1992; L. A. Clark, 1989; Gotlib & Cane, 1989). Thus research into the common and distinct symptoms of anxiety and depression has gained added significance for the diagnosis and assessment of these disorders.

COMMON AND SPECIFIC SYMPTOMS OF DEPRESSION

Symptoms such as irritability, restlessness, difficulty concentrating, fatigability, insomnia, crying, general distress, decreased activity, poor social skills, helplessness, worry, and low self-efficacy have been considered common to both anxiety and depression by at least some researchers (Alloy et al., 1990; L. A. Clark, 1989). More recently, L. A. Clark and Watson (1991a) proposed a

tripartite model of anxiety and depression. They described a state/ trait concept of high negative affect (NA) characterized by feelings of distress, fear, hostility, nervousness, or worry, (b) negativistic appraisals of self and others, (c) somatic complaints, (d) negative cognitions, and (e) diverse personality traits such as low self-esteem, pessimism, and trait anxiety. In their proposal, high NA was considered a common symptom feature of both anxiety and depression (see also L. A. Clark, Watson, & Mineka, 1994). According to the tripartite model, depression is distinguished by symptoms of anhedonia and low positive affect (PA), the latter being de-fined as a loss of pleasurable engagement with the environment, disinterest, low motivation, and social withdrawal (Tellegen, 1985; Watson & Kendall, 1989). Anxiety, on the other hand, is distinguished by symptoms of heightened physiological arousal such as heart palpitations, shortness of breath, dizziness, trembling, and the like (L. A. Clark et al., 1994). L. A. Clark and Watson (1991b) contend that symptom measures of anxiety and depression could improve on their low discriminant validity by including more items that assess either physiological hyperarousal or PA, respectively. Mineka, Watson, and Clark (1998) offered a revision to the tripartite model that recognizes that each psychological syndrome contains both common and specific symptom components. The common component in anxiety and depression is NA, a pervasive higher-order factor that accounts for the overlap among disorders. Anhedonia or low PA is a unique feature of depression; however, contrary to the original model, anxious arousal is no longer considered characteristic of all anxiety disorders but rather is a unique symptom feature of panic disorders. Every other anxiety disorder, with the exception of generalized anxiety disorder (GAD), will have unique symptom features that can be distinguished from anxious arousal. Two implications of this more integrative model is the recognition that the size of the common and specific symptom components of different anxiety disorders could vary considerably, and, that in the end, symptom specificity must be considered in relative rather than absolute terms. That is, given the problems inherent with *DSM* diagnostic classification, it is unlikely that any symptom will be unique to any particular disorder. Numerous studies have found support for the tripartite model with NA being the common factor to both anxiety and depression, low PA specific to depression, and physiological hyperarousal specific to anxiety (e. g., Jolly, Dyck, Kramer, & Wherry, 1994; Watson, Clark, & Carey, 1988; Watson et al., 1995). However, some inconsistent findings have also been reported. In some studies, a hierarchical model has been supported with NA a common higher-order factor and anhedonia (depression) and physiological hyperarousal (anxiety) as lower-order factors (D. A. Clark, Steer, et al., 1994; Steer et al., 1995), whereas other studies have supported a nonhierarchical model with NA, low PA (depression), and physiological hyperarousal (anxiety) distinct or orthogonal first-order dimensions (Joiner, 1996; Joiner, Catanzaro, & Laurent, 1996; Jolly & Dykman, 1994). Burns and Eidelson (1998) found that the tripartite model did not fit any of the covariance matrices derived from self-report symptom measures based on large samples of mood and anxiety disorder outpatients. Furthermore, physiological hyperarousal has not always been found to be specific to anxiety (D. A. Clark et al., 1998; Lonigan, Carey, & Finch, 1994), and low PA and anhedonia symptom items have emerged with significant loadings on a general NA dimension (D. A. Clark et al., 1998; Watson et al., 1995). This led to questions about the specificity of PA, whereas the inconsistent findings with physiological hyperarousal support the revision offered by Mineka et al. (1998). The co-morbidity of anxiety and depression is an important clinical issue because findings indicate that the co-occurrence of anxiety and depression may be associated with more severe psychological distress, increased chronicity, poorer response to treatment, and poorer psychosocial outcome (Bronisch & Hecht, 1990; D. A. Clark et al., 1990; Hecht, Von Zerssen, & Wittchen, 1990; Kessler et al., 1994). However, the issue of co-morbidity is also important to the cognitive model. Cognitive theory postulates that anxiety and depression can be distinguished by their cognitive content, with thoughts of personal loss and failure specific to depression, and cognitive content involving physical or psychological threat and danger specific to anxiety. As discussed in later chapters, empirical support for cognitive content-specificity has been found in a number of studies (for reviews, see D. A. Clark & Beck, 1989; Haaga et al., 1991; see also A. Beck, Brown, Steer, Eidelson, & Riskind, 1987; D. A. Clark, Beck, et al., 1994):

ISSUE 4: *Depressive symptoms and disorders show considerable covariation with other psychological conditions, especially anxiety and its disorders.*

THE COURSE OF CLINICAL DEPRESSION DURATION OF DEPRESSIVE EPISODES

Depressive disorders are best characterized as chronic disabling conditions that take a self-limiting episodic course. Most individuals recover from an acute episode of unipolar major depression, either with or with-out treatment (Coryell et al., 1995). The typical major depressive episode lasts from 3 to 11 months (Gotlib & Hammen, 1992), although 60% of cases recover within 6 months of the onset of depression (Coryell & Winokur, 1992). Kendler, Walters, and Kessler (1997) reported that the median *DSM-III-R* major depressive episode was 42 days for the 235 twins who met diagnostic criteria in their population-based twin registry study. Within 90 days, 75% of the sample had recovered, whereas only 2.2% had not recovered by the end of one year. In their 6-year follow-up of 313 treated and 234 untreated individuals with major depressive disorder, Coryell et al. (1995) found that median episode duration for the untreated individuals was 12 weeks compared with 42 weeks for the treated subjects. Although most individuals with major depression or other psychiatric disorders do not seek professional treatment (Diverty & Beaudet, 1997; Kessler et al., 1994), the results of the Coryell et al. study indicate that individuals with a more persistent and disabling depression may be overrepresented in clinical samples.

RELAPSE AND RECURRENCE RATES

Most individuals with an initial episode of major depression will experience a recurrence of the disorder even though they may have recovered from the initial episode. In fact, there is growing recognition that major depression takes a recurring course in most individuals who seek treatment for their disorder. In the *DSM-IV* mood disorders field trial, 68% of subjects had a recurrent depressive condition (Keller et al., 1995). In the 6-year follow-up study conducted as part of the National Institute of Mental Health Program on the Psychobiology of Depression–Clinical Studies, 234 individuals who had an untreated major depressive disorder were found to have a mean number of 2.2 depressive episodes (Coryell et al., 1995). However compared with the study's probands who sought treatment for their depression, the untreated depressed group had milder and shorter-lived depressions that selectively caused impairment in marital and interpersonal relationships but resulted in little impairment in occupational or educational attainment. Keller et al. (1982) found that 25% of patients treated for major depression relapsed in the first 12 weeks after recovery. More-over, a smaller group of patients (25%) will experience six or more episodes throughout their lifetime (Angst, 1986). In their retrospective study, Keller et al. (1995) found that 66% of treated patients with recurrent major depression experienced intermittent depressive symptoms between episodes, whereas the remainder reported full interepisode recovery. In a review article, Keller (1994) concluded that the predicted median length of time for being well after an episode of major depression is 20 months and the probability of maintaining a state of recovery for 5 years is only 22%. Thus multiple and recurring episodes of major depression are common, with the highest rate of relapse occurring in the first six months after recovery. Several variables significantly predict relapse in major depression: (a) increased number of previous depressive episodes, (b) age of onset under 40 years, (c) severity of depression at worst episode, (d) presence of dysthymia, (e) presence of minor depression, and (f) an episode of depression during adolescence (Compas et al., 1993; Coryell et al., 1991; Keller et al., 1982). Belsher and Costello (1988) concluded from their review of the literature that (a) within 2 years of recovery, 50% of unipolar depressed patients will relapse, (b) the longer you stay well, the less likely you are to relapse in the future, (c) recent stressors and absence of social support both increase risk of relapse, (d) history of depressive episodes increases relapse, (e) persistent neuroendocrine dysregulation after recovery increases probability of relapse, and (f) maintenance treatment with antidepressant medication can reduce risk of relapse. Early onset, persistent low-grade depression, stressful environment, and a history of recurrence increase one's risk for further episodes of depression.

CHRONIC DEPRESSION

Between 15% and 25% of individuals with major depression fail to recover within the usual time period, although most of these individuals (75%) were found to eventually recover during a 5-year follow-up (see Gotlib & Hammen, 1992). Factors that appear to be related to chronicity include (a) older age at onset, (b) presence of psychotic features, (c) a major depression superimposed on dysthymia, (d) early onset dysthymia, (e) possibly a variety of adverse stressful circumstances and reduced social resources, (f) greater severity and longer duration of episode, (g) higher neuroticism and poorer ego resilience, and (h) possibly the inadequate provision of treatment (Coryell & Winokur, 1992; Gotlib & Hammen, 1992; K. Wells et al., 1992). Although not all these factors have been investigated or been found significant across studies, each has some empirical support as a possible predictor of chronicity in depression.

SUMMARY

Major depression is a self-limiting condition that often takes a persistent and recurring course. A number of clinical, psychological and social factors have been proposed as having an impact on the course of major depression. Because research on maintenance factors in depression requires large samples and moderately long follow-up periods, most of the findings are based on the large-scale NIMH research projects. As discussed in subsequent chapters, cognitive theory proposes that some individuals may possess a cognitive vulnerability that increases their risk for unipolar depressive episodes. Unless this underlying vulnerability is dealt with effectively in the course of treatment, the theory proposes that a recurring depressive episode is even more likely because an initial depression may prime the prepotent depressogenic cognitive structures. In later chapters, we discuss possible cognitive processes that may influence the course of depression as well as the empirical support for cognitive predictors of relapse and recurrence in major depression:

ISSUE 5: *Depressive disorders are self-limiting conditions that often take a persistent and recurring course.*

OUTCOME IN MAJOR DEPRESSION

A final feature of depression that must be considered by any model or theory concerns the negative consequences or outcomes associated with having an episode of major depression. In this section, we consider two important outcomes that have been associated with depression—suicide and psychosocial functioning.

SUICIDE

Depression is a life-threatening condition because of its association with an increased risk for suicide. Suicide is the ninth leading cause of death in the United States, occurring in approximately 11 deaths per 100,000 (Resnik, 1980). Presence of a psychiatric illness is a significant risk factor for suicide, with 90% of individuals who commit suicide found to have a psychiatric condition at the time of death. Depression, alcoholism, and schizophrenia are the leading psychiatric disorders associated with risk for suicide (Barraclough, Bunch, Nelson, & Sainsbury, 1974). The link between depression and suicide is especially strong with 40% to 70% of suicide victims estimated to have an affective disorder (Brent, Kupfer, Bromet, & Dew, 1988). However, follow-up studies indicate that the lifetime probability of suicide in depressed patients appears to be approximately 15%. Based on the NIMH ECA study, J. Johnson et al. (1992) found that a significant number of suicide attempts were associated with presence of a depressive disorder (23%) or even sub-clinical depression (25.8%). Thus although presence of an affective disorder or even depressive symptoms is associated with a significant risk for suicide or suicide attempts, obviously it is a minority of individuals with depression who eventually commit suicide. The low base rate for suicide has made it difficult to study risk factors because even if predictors are found that yield low false-positive and false-negative rates, the extremely low base rate results in more false-positives than true-positives (Goldstein, Black, Nasrallah, & Winokur, 1991). With this caution in mind, a number of

variables have emerged in studies of risk factors for suicide among depressed patients. Possible risk factors in some of the studies include (a) male gender, (b) suicidal ideation at admission, (c) number of previous suicide attempts, (d) unipolar depressive disorder, (e) social isolation, (f) longer duration of depressive episode, and (g) hopelessness (Goldstein et al., 1991; Hawton, 1992). Of particular interest to the cognitive model of depression is the link found between hopelessness, suicidal intent, and committed suicide. Hopelessness has been defined as a set of cognitive schemas involving negative expectations about the future, and has been measured by a 20-item true/ false self-report inventory called the Hopelessness Scale (A. Beck, Weissman, Lester, & Trexler, 1974; Minkoff, Bergman, Beck, & Beck, 1973). Various studies have found a stronger link between intended and/ or committed suicide and hopelessness than between suicide and depression (A. Beck, Brown, Berchick, Stewart, & Steer, 1990; A. Beck, Kovacs, & Weissman, 1975; Dyer & Kreitman, 1984). In these studies, depressed inpatients and outpatients with elevated hopelessness were at much higher risk for suicide than depressed patients with low hopelessness. These results indicate that understanding how individuals think about themselves and their future is an important aspect of suicidal ideation and behavior.

PSYCHOSOCIAL FUNCTIONING

As noted, depressive disorders and even presence of a few depressive symptoms have been associated with both immediate and long-term reductions in psychosocial functioning and well-being. No doubt the most comprehensive study on functioning and well-being associated with depression comes from the large scale Medical Outcomes Study (MOS). In this observational study, 1,790 primary care patients with depression, myocardial infarction, congestive heart failure, hypertension, and diabetes were followed for 2 years. Functional status and well-being were assessed with a 36-item short-form MOS Health Form which assessed bodily pain, limitations in role functioning due to physical and emotional health, limitations in physical functioning due to health, general health perceptions, restrictions in usual social activities due to health, and level of energy or fatigue (Tarlov et al., 1989; K. Wells et al., 1992). At baseline, patients with a depressive disorder had worse physical, social, and role functioning than individuals with depressive symptoms alone or patients with a general medical condition. Moreover, when patients with any depressive symptoms (those with or without a depressive disorder) were compared with the general medical patients, the depressed individuals still had significantly worse physical, social, and role functioning, spent more recent days in bed, had worse current health and reported more bodily pain than most of the nondepressed general medical patients (K. Wells et al., 1989). At 2-year follow-up, the functioning and well-being of the depressed patients had improved, although patients with major depression and dysthymia were still worse off than those with a general medical condition (Hays et al., 1995). Even those with subthreshold depressive symptoms had similar or worse levels of functioning and well-being at the 2-year follow-up than the medical patients. Other studies have also reported significant reductions in outcome with depression. In a 1-year follow-up of 2,980 participants in the ECA Study, Broadhead and colleagues (1990) found that major depression and dysthymia were associated with the greatest risk of acquiring disability days and days lost at work, although minor depression was also associated with greater disability compared with asymptomatic individuals. Mintz et al. (1992) noted in their review that work impairment is a common feature of acute depressive episodes but concluded that treatment can lead to improvements in work functioning primarily by providing symptom relief. In the National Co-morbidity Survey, individuals with an early-onset psychiatric disorder had significantly lower educational attainment leading the authors to estimate that 7.2 million Americans prematurely terminate their education due to early-onset psychiatric illness (Kessler et al., 1995). A number of other studies have documented that the greatest reductions in psychosocial functioning are found with major depression or dysthymia, and that more moderate but significant decrements are also apparent in subthreshold depressive states (e. g., Gotlib et al., 1995; J. Johnson et al., 1992; Lyness, Caine, Conwell, King, & Cox, 1993; Spitzer et al., 1994). Keitner and Miller (1990) in their review noted that there is considerable empirical evidence that the acute phase of major depression is associated with increased family difficulties in the form of problematic

communication, impaired parenting, and poor problem-solving ability. Thus it should come as no surprise that the level of functional impairment associated with depression has resulted in a significant increase in health care utilization (J. Johnson et al., 1992; Spitzer et al., 1994) and significant economic burden (P. Greenberg et al., 1993; Simon, Ormel, Von Korff, & Barlow, 1995). The negative impact of depression on psychosocial outcome is apparent even after symptomatic recovery from an acute episode. In a 5-year follow-up of 148 patients with bipolar illness and 240 with unipolar depression, depressed patients compared with nondepressed first-degree relatives were (a) found to have lower annual incomes, (b) less likely to have been employed over the previous year, (c) less likely to have improved their occupational status or increased their income, (d) less likely to have improved their educational attainment, (e) more likely to have experienced a separation or divorce, (f) more likely to rate their current marital relationship as poor, and (g) more likely to report deficits in other areas of functioning such as interpersonal relationships, involvement in recreational activities, and overall life satisfaction (Coryell et al., 1993). Interestingly, when the researchers separated out the 110 unipolar probands who had recovered and showed no episodes of major, minor, or chronic intermittent depression throughout the final two years of the follow-up period, their psychosocial impairment was almost as great as for the total unipolar sample. In a more recent 6-year follow-up study based on data from the NIMH Psychobiology of Depression Clinical Studies, Coryell et al. (1995) found sustained impairment in marital and interpersonal relationships, and in enjoyment of recreational activities but not work functioning in individuals with an untreated depression. Coyne et al. (1998) recently investigated the perceived effects of depression in 60 depressed primary care patients, 82 nondistressed, nondepressed primary care patients, 40 distressed, nondepressed primary care individuals, and 48 depressed psychiatric outpatients. Based on a self-report questionnaire specifically constructed to assess perceived effects of depression, it was found that depressed patients identified as having recovered from their depressive episode scored significantly higher than the nondistressed groups on scales assessing lack of energy, perceived burden on others, need to work at not feeling depressed, need to work at maintaining relationships, imposition of limitations and sense of social stigma due to depression. Thus depression appears to have long-term repercussions on one's subjective sense of self and coping. These results indicate that depressive disorders and even depressive symptoms are associated with long-term impairment in social, occupational, and psychological functioning.

SUMMARY

This brief review of the outcome literature indicates that depressive disorders and even sub-clinical depressive symptoms are associated with significant short-and long-term impairment in psychosocial functioning and well-being. Depression can also be life-threatening in the form of increased risk for suicide. Moreover, the negative impact of a depressive disorder or symptoms may be compounded by the presence of another psychiatric disorder or a medical condition. Depressive disorders and symptoms are significantly more prevalent in patients with chronic medical conditions (e. g., K. Wells, Golding, & Burnam, 1988), and the presence of both a depressive condition and a medical illness can have a significant additive effect on reduced physical and social functioning (K. Wells et al., 1989). This leads us, then, to the sixth and final statement about depression that theories must take into account:

ISSUE 6: *Depressive symptoms and disorders can be life-threatening conditions that are associated with significant short-and long-term decrements in physical, psychological, social, and economic functioning.*

CONCLUSION

Depression is a complex, ubiquitous state that occurs with varying degrees of severity, can take a variable course, manifests itself in a variety of symptom forms and types, and is highly reactive to current life circumstances. Multiple social, psychological, and biological factors converge in an interactive fashion in the pathogenesis of depression. Because of space limitations and the focus of

this book, we have not dealt with many psychosocial and biological factors that are important correlates and features of the depressive experience. These factors need to be acknowledged, and any theory of depression must account for the role they play in the depressive experience. A prominent characteristic of depression that has been found in many studies is the gender difference in rates of depression with women approximately twice as likely to experience depression as men (Beaudet, 1996; Coryell, Endicott, & Keller, 1992; Kessler et al., 1994; Nolen-Hoeksema, 1987). Several explanations have been offered to account for these differential rates, and the conditions that are most likely associated with the higher risk for depression in women. A number of social factors are also associated with increased risk for depression such as severe adverse life events, lack of social support, and negative close interpersonal relationships (i. e., poor marital and family relations). Distal developmental antecedents to depression have also been reported such as childhood adversity (e. g., severe neglect, physical or sexual abuse, and to lesser extent, parental loss through death or divorce), previous depressive episodes in adolescence (Bifulco, Brown, Moran, Ball, & Campbell, 1998), history of parental depression (Hammen, Burge, Burney, & Adrian, 1990; Warner, Weissman, Fendrich, Wickramaratne, & Moreau, 1992), and poor parenting styles involving rejection, lack of affection, excessive criticalness or overprotectiveness that can lead to difficult interpersonal relationships in childhood (Blatt & Zuroff, 1992). Personality factors such as neuroticism, harm avoidance, dependency (sociotropy), self-criticalness, autonomy, obsessionality, and perfectionism have been implicated as vulnerability factors to depression in a variety of studies (see review by Enns & Cox, 1997). And psychological variables such as low self-worth, negative attributional style, pessimism, and poor coping responses, to name but a few, have been implicated in depression. These latter processes are discussed more thoroughly in later chapters. Age also appears to affect rates of depression, with the highest rates of major depression in young adults (aged 25 to 44 years) whereas rates for self-reported depressive symptoms show a curvilinear trend with the highest rates in the youngest (20s) and oldest (75+) age groups (Karel, 1997). Biological factors such as dysregulation in certain neurochemical pathways, particularly serotonin and possibly norepinephrine (McNeal & Cimbolic, 1986; Shelton, Hollon, Purdon, & Loosen, 1991), as well as reduced activity in particular regions of the brain, especially the prefrontal cortex (George et al., 1995), may be important in the pathogenesis of depression. Finally, considerable evidence now exists of a genetic vulnerability to unipolar depression that interacts with stressful life experiences to increase risk of onset in major depression (Kendler & Karkowski-Shuman, 1997; Kendler, Kessler, Neale, Heath, & Eaves, 1993; Kendler et al., 1995). The existence of these multiple, interactive etiological factors in depression requires the integration of biological, psychological, and social perspectives (Shelton et al., 1991). Almost 25 years ago, Akiskal and McKinney (1975) wrote their influential paper on the etiology of depression, arguing that psychological and biological events may be capable of setting in motion a final common biological pathway to depression. As shown in the chapters to come, the cognitive model of depression recognizes multiple determinants of depression and also postulates a "final common pathway" to depression, only this pathway is located within the meaning-making structures and processes of the information processing system. In this introductory chapter, we have focused on aspects of depressive phenomena that are considered important in the diagnosis, assessment, and treatment of affective disorders and symptoms. A considerable amount of research has been devoted to each of these topics in the past few years so that any psychological theory of depression must take them into account if it is to offer a viable understanding of depressive phenomena. The following six issues were discussed in this chapter: 1. Clinically significant depression is characterized by a sustained disturbance in mood involving behavioral inhibition, subjective negativity and, in more severe cases, disturbance in somatic functioning. 2. Depression consists of a heterogeneous group of disorders that vary in severity, chronicity, and clinical presentation. 3. Depressive states vary on a continuum of severity with milder, nonclinical affective symptoms at one end and the more severe clinical affective disorders at the other end. 4. Depressive symptoms and disorders show considerable covariation with other psychological conditions, especially anxiety and its disorders. 5. Depressive disorders are self-limiting conditions that often take a persistent and recurring course. 6. Depressive symptoms and disorders can be life-

threatening conditions that are associated with significant short-and long-term decrements in physical, psychological, social, and economic functioning. In subsequent chapters, our presentation of the cognitive model of depression focuses on how the model accounts for each of these important features of depression. The validity of any theoretical model is deter-mined not only by its empirical support but also by its ability to address crucial aspects of the target construct. Thus the construct validity of the cognitive model of depression depends in part on its ability to incorporate into its theoretical account what is known about the salient features of depression. The six issues reviewed in this chapter represent fundamental aspects of depression and so must be addressed by the cognitive model. This is the focus of our discussion in Chapter 4, where we present the cognitive model of depression. Before describing the model, however, we want to lay the groundwork by tracing the historical roots of cognitive therapy in Chapter 2 and discuss the philosophical and theoretical assumptions of the model in Chapter 3.