

# Ambiguity and Controversy

An Introduction

**James C. Coyne**

*Essential Papers on Depression*

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## Ambiguity and Controversy: An Introduction

Discussions of depression often start with a statement that it is the common cold of psychopathology, a ubiquitous affliction to which most all of us are subject from time to time. Such discussions may note that at any one time, one fifth of the adult population will have significant depressive symptoms, and that most of this depression goes untreated (Weissman & Meyers, 1981). It may also be suggested that whoever is most likely to become depressed is largely a matter of psychological background and social conditions; depression is a “curse of civilization,” and its occurrence is linked to stress and deprivation, the disintegration of relationships, and depressing life circumstances. Thus, Pearlin (1975) has stated that depression is “intertwined with the values and aspirations that people

acquire; with the nature of the situation in which they are performing major roles, such as in occupation and family; with the location of people in broader social structures, such as age and class; and the coping devices that they use . . .” (p. 206).

At the other extreme, discussions of depression may begin with an assertion that it is one of the most serious of mental-health problems. The discussion may then go on to emphasize that it is primarily a biological disturbance, an illness, the predisposition to which lies in genes and biochemistry. While people may indeed react to their circumstances with happiness and unhappiness, this is of questionable relevance to the clinical phenomena of depression.

Advocates of each of the positions discussed above can marshal impressive evidence; yet, taken together, they present a basic contradiction. They

differ not only in their view of the causes of depression but its very definition. Beck (1967) has noted, “there are few psychiatric syndromes whose clinical descriptions are so constant through successive eras of history” (p.5). However, as these opposing positions demonstrate, definitional problems continue to plague the study of depression, and they are not going to be readily resolved. There remains considerable disagreement as to what extent and for what purposes a depressed mood in relatively normal persons can be seen as one end of a continuum with the mood disturbance seen in hospitalized psychiatric patients and to what extent the clinical phenomena is distinct and discontinuous with normal sadness and unhappiness.

Should we limit the term “depression” to those people who are most distressed and seeking

treatment? And what do we make of the “merely miserable” that we have defined out of the “depressed” category? If we agree to make a sharp distinction, where is it to be drawn? What of the differences *among* depressed persons? The positions on these questions that one takes have major implications for who one studies and who one treats and how, what data are going to be considered relevant, and how one organizes that data. Many of the differences in the theoretical positions to be discussed in this volume start with a fundamental difference in how depression is defined. We cannot pretend to resolve these controversies, but we can at least identify them and note some of the definitions and distinctions that are being employed currently.

One purpose of this introductory chapter is to provide an overview of the phenomena of depression and to note some of the diagnostic

distinctions that are currently being made. It should become apparent that there is a tremendous heterogeneity to what falls under the broad rubric of depression and that there is an arbitrariness to any boundaries that are drawn on these phenomena. There are striking differences *among* depressed persons that invite some form of subtyping. As will be seen, however, efforts to derive such subtypes are generally controversial, and any scheme is likely to be more satisfactory for some purposes than for others. Confronted with all of this ambiguity and confusion, one must be cautious and not seek more precision than the phenomena of depression afford, and one should probably be skeptical about any decisive statement about the nature of depression.

This chapter is also intended to prepare the reader for the wide diversity of theoretical perspectives that will be presented in this volume.

Contemplating the phenomena of depression, one can readily detect patterns and come to a conclusion that some aspects of depression are more central than others; some are primary and causal, and others are secondary. One observer may be struck with the frequency of complaints about appetite and sleep disturbance by depressed persons and infer that some sort of biological disturbance must be the key to understanding depression. Another might find their self-derogation and pessimism irrational in a way that suggests that there must be some kind of fundamental deficit in self-esteem or cognitive distortion occurring. Still another may listen to the incessant complaining of a depressed person, get annoyed and frustrated, and yet feel guilty in a way that makes it easier to encourage the depressed person to continue to talk in this way than to verbalize these negative feelings.

Cognizant of this, the observer might conclude that there is some sort of interpersonal process going on that is critical to any understanding of depression.

### **DEPRESSION AS MOOD**

A major source of confusion is due to the fact that the term “depression” variously refers to a mood state, a set of symptoms, and a clinical syndrome. As a reference to mood, depression identifies a universal human experience. Adjectives from a standard measure of mood (*The Multiple Affect Adjective Checklist*; Zuckerman & Lubin, 1965) point to subjective feelings associated with a depressed mood: sad, unhappy, blue, low, discouraged, bored, hopeless, dejected, and lonely. Similarities between everyday depressed mood and the complaints of depressed patients have encouraged the view that clinical

depression is simply an exaggeration of a normal depressed mood. However, patients sometimes indicate that their experience of depression is quite distinct from normal feelings of sadness, even in its extreme form. A patient once remarked to me that her sadness was overwhelming when her husband died but that it did not compare with her sense of emptiness and her loss of any ability to experience pleasure at the time that she entered the hospital.

The view that depressed mood in otherwise normal persons is quantitatively but not qualitatively different than the depression found in hospitalized patients has been termed the *continuity hypothesis*. Beck (1967) has provided a useful analogy to suggest the alternative to the continuity hypothesis. He notes that everyday fluctuations in body temperature can be measured on the same thermometer as the changes

associated with a fever. Yet the conditions giving rise to a fever are distinct from those causing fluctuations in temperature in healthy individuals. Similarly, the conditions giving rise to clinical depression may be distinct from those producing fluctuations in normal mood.

Studies have compared the subjective mood of persons who are distressed but not seeking help to those who are seeking treatment for depression or a review, see Depue & Monroe, 1978a). The two groups may be similar in subjective mood, but they differ in other ways. Those persons who are not seeking treatment for depression tend to lack the anxiety and the physical complaints, including loss of appetite, sleep disturbance, and fatigue shown by the group seeking treatment. Still, it could be argued that there is a continuum between the two groups, with these additional features arising when a normal depressed mood becomes

more prolonged or intensified. The controversy is likely to continue until either questions about the etiology of depression are resolved or unambiguous markers for depression are identified.

Advocates of biomedical approaches to depression tend to assume that there is a discontinuity between a normal depressed mood and clinical depression, and that appropriate biological markers will be found. Yet, as the article by Winokur in this volume suggests, even if that proves to be the case, there are likely to be many individuals suffering from extremes of depressed mood who do not have these markers.

Advocates of psychoanalytic, cognitive and behavioral, and interpersonal and social perspectives on depression have generally assumed a continuum between a normal

depressed mood and clinical depression. They tend to exclude psychotic and bipolar depressed persons from treatment, but, beyond that, they have tended to disregard classification issues (Gilbert, 1984). For unipolar depression, at least, they have assumed that whatever discontinuities in the biology of mild and severe moods there might be are not necessarily relevant to the psychological and social processes in which they are most interested.

### **SYMPTOMS OF DEPRESSION**

Writers since antiquity have noted the core symptoms of depression: besides a sad or low mood, reduced ability to experience pleasure, pessimism, inhibition and retardation of action, and a variety of physical complaints. For the purposes of discussion, we can distinguish among the emotional, cognitive, motivational, and

vegetative symptoms of depression, although these features are not always so neatly divisible. Beyond these symptoms, there are some characteristic interpersonal aspects of depression that are not usually considered as formal symptoms. But they are frequent, distinctive, and troublesome enough to warrant attention.

### Emotional aspects of depression

Sadness and dejection are not the only emotional manifestations of depression, although about half of all depressed patient report these feelings as their principal complaint. Most depressed persons are also anxious and irritable. Classical descriptions of depression tend to emphasize that depressed persons' feelings of distress, disappointment, and frustration are focused primarily on themselves, yet a number of studies suggest that their negative feelings,

including overt hostility, are also directed at the people around them. Depressed persons are often intensely angry persons (Kahn, Coyne, & Margolin, in press; Weissman, Klerman, & Paykel, 1971).

Perhaps 10 or 15 percent of severely depressed patients deny feelings of sadness, reporting instead that all emotional experience, including sadness, has been blunted or inhibited (Whybrow, Akiskal, & McKinney, 1984). The identification of these persons as depressed depends upon the presence of other symptoms. The inhibition of emotional expression in severely depressed persons may extend to crying. Whereas mild and moderately depressed persons may readily and frequently cry, as they become more depressed, they may continue to feel like crying, but complain that no tears come.

Mildly and moderately depressed persons may

feel that every activity is a burden, yet they still derive some satisfaction from their accomplishments. Despite their low mood, they may still crack a smile at a joke. Yet, as depression intensifies, a person may report both a loss of any ability to get gratification from activities that had previously been satisfying—family, work, and social life—and a loss of any sense of humor. Life becomes stale, flat, and not at all amusing. The loss of gratification may extend to the depressed persons' involvement in close relationships. Often, a loss of affection for the spouse and children, a feeling of not being able to care anymore, a sense of a wall being erected between the depressed person and others are the major reasons for seeking treatment.

### Cognitive aspects of depression

In the past decade, a number of theorists,

notably Beck and Abramson, Seligman, and Teasdale have given particular attention to the cognitive manifestations of depression and have assumed that these features are causal of the other aspects of the disorder. Depressed persons characteristically view themselves, their situations, and their future possibilities in negative and pessimistic terms. They voice discouragement, hopelessness, and helplessness. They see themselves as inadequate and deficient in some crucial way. There may be thoughts of death, wishing to be dead, and suicide attempts.

Depressed persons' involvement in their daily lives are interpreted by them in terms of loss, defeat, and deprivation, and they expect failure when they undertake an activity. They may criticize themselves for minor shortcomings and seemingly search for evidence that confirms their negative view of themselves. Beck (see Kovacs &

Beck, this volume) suggests that they will tailor the facts to fit these interpretations and hold to them in the face of contradictory evidence. Depressed persons overgeneralize from negative experiences, selectively abstract negative details out of context, ignore more positive features of their situations, and negatively characterize themselves in absolutist and dichotomous terms. The revised learned-helplessness model (see Abramson, Seligman, & Teasdale, this volume) emphasizes that depressed persons are particularly prone to blame themselves for their difficulties and to see their defects as stable and global attributes.

Aside from these content aspects of their thinking, depressed persons frequently complain that their thinking processes have slowed down, that they are distracted, and they cannot concentrate. Decisions pose a particular problem.

Depressed persons are uncertain, feel in need of more information, and are afraid of making the wrong decision. They may simply feel paralyzed, and that the work of making a choice and a commitment is an overwhelming task to be avoided at any cost.

### Motivational aspects of depression

Perhaps one of the most frustrating aspects of depressed persons for those around them is their difficulty in mobilizing themselves to perform even the most simple tasks. Encouragement, expressions of support, even threats and coercion seem only to increase their inertia, leading others to make attributions of laziness, stubbornness, and malingering. Despite their obvious distress and discomfort, depressed persons frequently fail to take a minimal initiative to remedy their situations or do so only halfheartedly. To observers,

depressed persons may seem to have a callous indifference to what happens to them.

Depressed persons often procrastinate. They are avoidant and escapist in their longing for a refuge from demands and responsibilities. In severe depression, the person may experience an abulia or paralysis of will, extending even to getting out of bed, washing, and dressing.

In more severe depression, there may be psychomotor retardation, expressed in slowed body movements, slowed and monotonous speech, or even muteness. Alternatively, psychomotor agitation may be seen in an inability to sit still, pacing, and outbursts of shouting.

### [Vegetative aspects of depression](#)

The presence of physical or vegetative symptoms are sometimes taken as the dividing line between normal sadness and clinical

depression. One of the most common and prominent vegetative symptoms is fatigue. That someone is depressed may be first recognized by the family physician who cannot readily trace the person's complaints of tiredness to other causes.

Depressed persons also often suffer sleep disturbance, and it is tempting to link their tiredness to this, but in a sample of depressed patients, the two complaints are only modestly correlated (Beck, 1967). Depressed persons generally have trouble falling asleep, they sleep restlessly, and awaken easily. Yet some depressed persons actually sleep considerably more than usual, up to 12 hours a night.

When mildly or moderately depressed, some people eat compulsively and gain considerable weight, but depression is more characteristically associated with loss of appetite and a decrease in

weight. Indeed, for many depressed persons, a loss of appetite is the first sign of an incipient depression, and its return marks the beginning of recovery. Some depressed persons maintain their normal eating habits and weight, but complain that food is tasteless and eating an unsatisfying matter of habit. Besides a loss of appetite, depression is often associated with gastrointestinal disturbance, notably nausea and constipation.

Mild depression heightens sexual interest in some people, but generally depression is associated with a loss of interest in sex. In severe depression, there may be an aversion to sex. Overall, though, women who are depressed do not have sex less frequently, but they initiate it less, enjoy it less, and are less responsive (Weissman & Paykel, 1974).

Finally, depressed persons report diffuse aches and pains. They have frequent headache, and they are more sensitive to existing sources of pain, such as dental problems.

### Interpersonal aspects of depression

A brief interaction with a depressed person can have a marked impact on one's own mood. Uninformed strangers may react to a conversation with a depressed person with depression, anxiety, hostility, and may be rejecting of further contact (Coyne, 1976; see Gurtman, in press, for a review). Jacobson (1968) has noted that depressed persons often unwittingly succeed in making everyone in their environment feel guilty and responsible and that others may react to the depressed person with hostility and even cruelty. Despite this visible impact of depression on others, there is a persistent tendency in the literature to ignore it

and to concentrate instead on the symptoms and complaints of depressed persons out of their interpersonal context. Depressed persons can be difficult, but they may also be facing difficult interpersonal situations within which their distress and behavior makes more sense (see Coyne, this volume).

Depressed persons tend to withdraw from social activities, and their close relationships tend to be strained and conflictual. Depressed women have been more intensely studied than depressed men, in part because women are approximately twice as likely to be depressed (see Radloff, this volume). Depressed women are dependent, acquiescent, and inhibited in their communication in close relationships, and prone to interpersonal tension, friction and open conflict (Weissman & Paykel, 1974). Interestingly, the interpersonal difficulties of depressed persons are less

pronounced when they are interacting with strangers than with intimates (Hinchcliffe, Hooper, & Roberts, 1975).

About half of all depressed persons report marital turmoil (Rousanville, Weissman, Prusoff, & Heraey-Baron, 1979). There is considerable hostility between depressed persons and their spouses, but often there is more between depressed persons and their children. Being depressed makes it more difficult to be a warm, affectionate, consistent parent (McLean, 1976). The children of depressed parents are more likely to have a full range of psychological and social difficulties than the children of normal or even schizophrenic parents (Emery, Weintraub, & Neale, 1982), yet one must be cautious in making causal inferences. There is evidence that the child problems are more related to a conflictful marital relationship and a stressful homelife than

depression of the parent per se (Sameroff, Barocas, & Siefer, in press).

Depression thus tends to be indicative of an interpersonal situation fraught with difficulties, and this needs to be given more attention in both theorizing and planning treatment. Although depression is associated with interpersonal problems, *within* a sample of depressed persons the correlation between severity of depression and the extent of interpersonal problems tends to be modest. This may suggest that these problems are a matter not only of how depressed persons are functioning, but of the response of key people around them as well (Coyne, Kahn, & Gotlib, 1985).

### **THE DIAGNOSIS OF DEPRESSION**

One can make a list of the symptoms of depression, and assign any person a depression

score on the basis of the number of symptoms present. A number of standard self-report inventories such as the *Beck Depression Inventory* (Beck, et al., 1961), the *Center for Epidemiologic Studies Depression Scale* (Radloff, 1977), and the *Self-Rating Depression Scale* (Zung, 1965) have been validated and are widely used as research tools, screening devices, and measures of the changes associated with treatment.

Even if one assumes a continuity between normal depressed mood and clinical depression, it may still prove useful to make a distinction between the presence or absence of significant depression. One may wish to insure that a research study does not include a preponderance of persons whose depression is only mild or transient. Virtually no signs or symptoms are specific to depression, and yet in many contexts, one may need to distinguish depression from

other descriptors or explanations for a person's distress and behavior. In working with the elderly, for instance, it is important to distinguish between depression and dementia. In medical patients in general, there is a high prevalence of symptoms associated with depression, both because of physical illness and the stress of hospitalization (Cavanaugh, 1984), and, whether for research or practical purposes, one may wish to establish criteria for who is to be considered depressed and who is not. Finally persons who are labeled schizophrenic or alcoholic may show considerable depression, but it would be undesirable for many purposes to lump them with those persons whose primary problem is depression. Thus, for the purposes of research, treatment, and professional communication, it proves useful to have some means of specifying some boundary conditions for the term "depression," in terms of some minimal

level of severity as well as some coherence and specificity to what is included in the concept—even if one rejects the notion that it is a discrete entity, discontinuous with normal mood.

The problem of diagnosis is most critical in biomedical approaches to depression. The assumption is generally made that depression is a matter of one or more disease entities with specific etiologies and treatments. The statement, “Nosology precedes etiology” conveys the idea that the ability to identify the causes of depression depends upon the existence of an adequate diagnostic and classificatory system. For instance, to take a simplified hypothetical example, suppose that a particular biological abnormality occurs in 60 percent of all depressed persons and is specific to depression. Suppose also that, with the accepted diagnostic criteria, only 60 percent of the persons identified as such are “actually

depressed.” If these conditions occurred, then research might indicate that only 36 percent of depressed persons possess the abnormality.

An effective treatment for depression may also be misjudged or misapplied in the absence of an adequate diagnostic system. This was made apparent recently after a drug company had undertaken a large study to compare the effectiveness of a new drug to that of both an established drug treatment for depression and a placebo (Carroll, 1984). At five of the six research sites, the new drug proved to be no more effective than a placebo, but interpretation of this was limited by the additional finding that the established treatment proved no better. Patients identified as depressed by current criteria did not respond to drug treatment that had proven efficacious in a large body of past research. Either the past research was misleading, the current

diagnostic criteria are invalid, or, most likely, they were misapplied by reputable investigators.

Contemporary diagnostic systems owe much to the work of Kraepelin at the turn of the century. He divided major psychopathology into two broad syndromes: dementia praecox (schizophrenia) and manic-depressive illness. The latter category included almost all serious mood disturbance, including depression in the absence of an episode of mania. As retained today, the term generally is a synonym for bipolar disorder (see below). It is also still sometimes used as a generic term for severe depression. Kraepelin considered manic-depressive illness a biological derangement. Although it might in some cases be precipitated by psychological factors, “the real cause for the malady must be sought in *permanent internal changes* which are very often, perhaps always innate” (Kraepelin, 1921, p. 180). Once started, the

illness runs its course autonomously, independent of changes in the person's situation. Kraepelin also identified a group of psychogenic depressions, which were precipitated by life circumstances, but that were milder than manic-depressive illness and reactive to changes in these circumstances.

For over 30 years, the dominant diagnostic system in the United States has been the *Diagnostic and Statistical Manual of Mental Disorders* of the American Psychiatric Association, which is currently in its third edition (DSM-III). In its first edition it integrated the ideas of Kraepelin with those of Adolph Meyer and Sigmund Freud. While accepting Kraepelin's basic distinction between affective disturbance and schizophrenia, it also reflected Meyer's psychobiological view that mental disturbance represented not a simple disease entity, but the reaction of the personality to the a matrix of psychological, social, and

biological factors. By its second edition, the Meyerian term “reaction” was no longer used throughout, but Meyer’s influence remained. Freud’s ideas about the etiology of psychopathology were built into the criteria for specific disorders. Thus, the chief defining characteristic of neuroses was anxiety, but for purpose of diagnostic decisions, it could be manifest and observable or inferred to be operating “unconsciously and automatically” in someone who was not visibly anxious.

The authors of DSM-III attempted to avoid past controversies and answer many of the criticisms of its two predecessors. A decision was made to define diagnostic categories as precisely as possible, using descriptive data, rather than inferences about etiology. From a biomedical perspective, the ideal diagnostic and classificatory system would integrate knowledge about etiology

with overt symptomatology. However, it was concluded that the present understanding of the causes of most disorders is too limited for this purpose. Furthermore, the sense was that “the inclusion of etiological theories would be an obstacle to use of the manual by clinicians of varying theoretical orientations, since it would not be possible to present all reasonable etiological theories for each disorder” (American Psychiatric Association, 1980, p. 7).

In considering depression, the authors of DSM-III attempted to sidestep a number of longstanding controversies, including that of whether there is a continuum or a discontinuity between normal mood and clinical depression, as well as that of the role of precipitating life circumstances in distinguishing among types of depression. Depressive neurosis disappeared, along with the other neuroses. Depression is now encompassed

in two main categories. The first category, major affective disorder, involves the presence of a full affective syndrome, with the subcategories of bipolar and major depression distinguished by whether there has ever been a manic episode. The second category, other specific affective disorders, includes conditions in which the depression is not severe enough to warrant a diagnosis of major affective disorder, but the mood disturbance has been intermittent or chronic for at least two years.

The criteria for major depression are presented in Table 1. Major depression is subclassified as to whether it is a single episode or recurrent and also as to whether *melancholia* is present. Melancholia involves a complaint of a loss of pleasure in all or almost all activities, a lack of reactivity to pleasant events, and at least three of six symptoms: a quality of depressed mood that is distinct from grief or sadness; depression worse in

the morning; early morning wakening; marked psychomotor agitation or retardation; significant weight loss; and excessive guilt. The designation was intended as an acknowledgment that some more severe depressions were characterized by a particular constellation of symptoms and might be more responsive to treatment with drugs or electroshock. This issue will be discussed further below. It should be noted, however, that there is considerable consensus that such a distinction should be made, but the exact nature of it remains controversial.

*Table 1. DSM-III Criteria for Major Depressive Episode*

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A. Dysphoria or loss of interest or pleasure in all or almost all usual activities and pastimes. The dysphoric mood is characterized by symptoms such as the following: depressed, sad, blue, hopeless, down in the dumps, irritable. The mood disturbance must be prominent and relatively persistent, but not necessarily the most dominant symptom, and does not include momentary shifts from one dysphoric mood to another dysphoric mood, e.g., anxiety to depression to anger, such as are seen in states of acute psychotic turmoil. (For children under six, dysphoric mood may have to be inferred from a persistently sad facial expression.)

B. At least four of the following symptoms must have been present nearly every day for a period of at least two weeks (in children under six, at least three of the first four).

1) poor appetite or significant weight loss (when not dieting) or increased

appetite or significant weight gain (in children under six, consider failure to make expected weight gains)

- 2) insomnia or hypersomnia
- 3) psychomotor agitation or retardation (but not merely subjective feelings of restlessness or being slowed down) (in children under six, hypoactivity)
- 4) loss of interest or pleasure in usual activities, or decrease in sexual drive not limited to a period when delusional or hallucinating (in children under six, signs of apathy)
- 5) loss of energy; fatigue
- 6) feelings of worthlessness, self-reproach, or excessive or inappropriate guilt (either may be delusional)
- 7) complaints or evidence of diminished ability to think or concentrate, such as slowed thinking, or indecisiveness not associated with marked lessening of associations or incoherence

8) recurrent thoughts of death, suicidal ideation, wishes to be dead, or suicide attempt

C. Neither of the following dominate the clinical picture when an affective syndrome (criteria A and B above) is not present, that is, before it developed or after it has remitted:

1) preoccupation with a mood-incongruent delusion or hallucination

2) bizarre behavior

D. Not superimposed on either schizophrenia, schizophreniform disorder, or a paranoid disorder.

E. Not due to any organic mental disorder or uncomplicated bereavement

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As can be seen in Table 1, a diagnosis of major depressive disorder requires evidence of mood disturbance and at least four other symptoms lasting at least every day for two weeks. There are also exclusion criteria, including schizophrenia

and what is judged to be normal or uncomplicated grief.

The criteria for depression are somewhat arbitrary. An alternative set of diagnostic criteria that is widely used in research (Spitzer, Endicott, & Robins, 1978) requires a mood disturbance lasting at least one week and at least three symptoms. Still another (Feighner, Robins, & Guze, 1972) requires four symptoms and a one month duration. While there is a consensus that such disagreement is deplorable, there is not at present a way of resolving it satisfactorily that is not itself arbitrary. In general, these diagnostic systems are viewed as significant improvements over past efforts, but there is widespread dissatisfaction with them. A prominent biologically oriented researcher has lamented

An astute observer will find little that is intellectually satisfying about the DSM-III

diagnostic criteria for major depressive disorder. These criteria amount to a catalogue of symptoms, and they are in no way linked by coherent underlying constructs. They also suffer from the problem of being cast as disjunctive criteria. This means that in section B, for example, patients need to satisfy only 4 from a total of 20 possible symptoms. Therefore (and this occurs in practice), several patients may be assigned the same diagnosis without having any symptoms in common (Carroll, 1984, p. 16).

Carroll goes on to note that as the result of an inadequate diagnostic system, research studies are limited by the flaws in the diagnoses used as independent variables, and drug treatment of an individual patient tends to remain a matter of trial and error.

We are far from an adequate diagnostic system for depression. If one is to be achieved, it will have to come to terms with the enormous

heterogeneity in the signs and symptoms, level of severity, causal factors, and clinical course that has been subsumed under the term “depression.” In the past half century, there have many efforts to bring order to this heterogeneity with a variety of classificatory systems. Kendall (1976) has suggested that almost every classificatory system that is logically possible has been proposed at some point in this period, but he notes that little consensus has been achieved. Winokur (see this volume) will review some of the current controversies, but it would be useful to identify a few of the distinctions that have been made before we turn to the major theoretical perspectives on the disorder.

### **SUBTYPES OF DEPRESSION**

Of all the distinctions that have been proposed, the most widely accepted and least controversial

is that between unipolar and bipolar disorder. In its simplest form—and as it has been recognized in the DSM-III—the differential diagnosis is based on whether the patient has a personal history of mania. However, recent genetic studies have led to a familial definition of the distinction: Depressed patients who do not have a personal history of mania may still be diagnosed as being bipolar if there has been mania among first-degree relatives.

Work by Perris (1966) first established that bipolar disorder starts on the average of 15 years earlier than unipolar depression and recurs more frequently. Individual episodes are shorter, and there is a greater risk of disorder among the first-degree relatives of bipolar patients. Furthermore, there was a tendency for unipolar and bipolar disorders to breed true, with first-degree relatives of bipolar patients tending toward bipolar disorder, and first-degree relatives of unipolar

patients tending to have little more risk of mania than the general population. The unipolar-bipolar distinction has proven to be clinically useful; depressed bipolar patients respond significantly better to lithium than unipolar depressed patients.

Valid though the distinction appears to be, it has some important limitations. As yet, no consistent differences in the symptomatology of bipolar and unipolar depression have been identified. Although a bipolar diagnosis predicts a greater likelihood of response to lithium, as many as 40 percent of unipolar patients nonetheless respond positively (Depue & Monroe, 1978b). By itself, the distinction does not do justice to the heterogeneity among either bipolar or unipolar patients. Currently, persons with bipolar disorder are often subclassified as to whether either manic or depressive symptoms or both have been severe to require hospitalization (see Dunner, this

volume). Unipolar depressed persons remain a large and tremendously heterogeneous group. Nonetheless, in the continuing controversies as to how best to distinguish among depressed persons, the unipolar-bipolar distinction stands out in its usefulness for both clinical and research purposes.

Many issues in the study of unipolar depression have coalesced in the concept of endogenous versus nonendogenous depression. The differentiation is most often identified as being between endogenous and reactive depressions, although this has been used interchangeably with the endogenous-neurotic and psychotic-neurotic distinctions. The hope for the distinction has often been that it would prove to be the boundary between biological versus psychological and social concerns. Traditionally, the term “endogenous” has been invoked to differentiate depressions that are purportedly

biological in etiology, without environmental precipitants, and that are less amenable to psychotherapy. Also, endogenous depressions are expected to be more responsive to somatically oriented interventions, notably electroconvulsive shock therapy and antidepressant medication. “Reactive” has referred to depressions that are viewed as understandable reactions to some precipitating stress and that are both more suitable for psychotherapy and less responsive to somatic therapies. The distinction was originally based on the supposition that some depressions are related to precipitating events and others seem to appear without them and that this would predict response to treatment and clinical course.

Controlled studies have not found that the endogenous-reactive distinction predicts response to psychotherapy (Blackburn, et al., 1981; Kovacs, 1980; Rush, 1984). The presence or absence of

precipitating stress has not proved to be a good predictor of response to treatment (Leff, Roatch, & Bunney, 1970), and the endogenous-reactive distinction has been found to be deficient in a number of ways. Yet it retains considerable utility. Reactivity to changes in life circumstances *during* a depressive episode have been found to predict response to electroconvulsive shock and antidepressant medication (Fowles & Gersh, 1979). Other symptoms that have been associated with a positive response to somatic treatment include quality of mood and whether there has been a loss of the ability to experience pleasure; psychomotor retardation; feeling worse in the morning after than the evening; and sleep and appetite disturbance. Such symptoms are now more accepted as criteria for endogenous depression than is the absence of precipitating stress.

This consensus about the features of endogenous depression still leaves questions about its polar opposite, reactive or neurotic depression. In clinical practice, it tends to be defined in terms of milder mood disturbance, a preponderance of psychological rather than vegetative symptoms, and the presence of a precipitating stress, although there are particular doubts about the validity of this last feature. Akiskal et al. (1978) found that reactive or neurotic depression was the single most common diagnosis in inpatient and outpatient settings, but they raised the issue of whether it was useful to consider it a unified entity or type. In about a quarter of all the cases of such depression studied, it appeared to be truly reactive, in the sense that it developed in the face of overwhelming stress in persons who had previously seemed reasonably well functioning. In another quarter of the cases, it

seemed to reflect a more or less chronic tendency to respond to normative stress with depressed mood and to experience social difficulties. Many of these patients were described as dependent, manipulative, hostile, and unstable. Follow-up revealed overall that only 40 percent of the total sample was considered to be have been suffering primarily from an affective disturbance in the absence of some of other condition. Some of the subsample who had faced a clear precipitating stress developed endogenous features. In 10 percent of the sample, the depression seemed secondary to a medical-surgical illness. In 38 percent of the sample, the depression was secondary to some nonaffective disorder, ranging from an anxiety disorder to schizophrenia. In these patients with medical-surgical or nonaffective psychiatric conditions, intermittent depression seemed to follow the course of the

other difficulties. A final 10 percent of the sample remained undiagnosed, but depression was considered the probable diagnosis. The work of Akiskal et al. (1978) is further evidence of the problems in attempting to draw any sharp distinctions in the classification and diagnosis of depression. Beyond this, it suggests both the utility and the difficulty of distinguishing between depression that is primary and that which is secondary to other conditions. Furthermore, the work suggests the usefulness of attempting to understand depression in terms of the presence or absence of characterological or life-style difficulties. (See Winokur, this volume, for further development of this point.)

Thus, the endogenous pole of the endogenous-reactive distinction is more clearly defined than its counterpart. After a long history of debate and controversy, there is a growing consensus that the

differentiation of endogenous and reactive depression is useful but that they represent points along a continuum, rather than two distinct forms of disorder. It is sometimes suggested that endogenous depressions are simply more severe, but this leaves unanswered questions about differences in etiology or the determinants of one depressive episode progressing to an endogenous course and another not. Biomedically oriented researchers look to the identification of familial patterns of affective disturbance, the development of biological markers, and the refinement of diagnostic laboratory tests as the solution to the ambiguity and confusion. Baldessarini (see this volume) notes the promise of recent developments such as the dexamethasone suppression test, but he cautions that

While there has been considerable progress toward a biologically and clinically robust diagnostic scheme, and in understanding

some characteristics that can help to guide treatment, search for primary causes has been unsuccessful so far. Indeed, virtually all of the biological characteristics of [severely depressed] patients that have been identified are “state-dependent” (that is, they disappear with recovery) and are not stable biological traits or markers of a possible heritable defect.

### **DUALISM AND REDUCTIONISM**

In discussions of the diagnosis and subtyping of depression, it is easy to detect the suggestion that biology plays an obvious or central role in some depressions more than others, in bipolar more than unipolar, and endogenous more than nonendogenous. Useful though this insight is, it tends to be accepted too rigidly and simplistically. Too often it becomes a way of summarily resolving complex issues in the study and treatment of depression, namely, that there are some depressions that are biological and others that are

psychological and social in nature or some that are illnesses and some that are not. Indeed, the goal of being able to make such a clean distinction has often been behind efforts to develop classificatory systems.

The acceptance of a such a mind-body dualism and reductionism is widespread, but it is a distortion of available data and a barrier to both effective treatment and the development of a model that does justice to the complexities of depression. The biological dysfunctions associated with depression are well recorded and can no longer be ignored. Yet, even where biological vulnerability factors are well established, as in the case of bipolar disturbance, psychological, and social factors may determine whether an episode actually occurs; its severity, course, and outcome; and its costs to the individual, the immediate family, and the larger society. Whether we choose

to focus on the biological, the psychological, or the social, we are isolating only one of a set of factors in a complex matrix. Gilbert (1984) has suggested further:

Moreover, the interdependence of the structure of the matrix makes selection of one group of factors as etiological agents arbitrary. In other words, it is not particularly helpful, at the macro-level, to view the causes of depression as due only [for example] to cognitive changes, or only to biological changes. Rather, these factors are locked together in complex relationships, and it is the change of the whole person, determined by the relationship of factors within the person, which provides the most useful conceptualization (p. 105).

Increasingly, theoretical statements about the nature of depression start with an acknowledgment of its heterogeneity and the complexity and interdependence of causal factors now presumed to play a role in it. Yet beyond that,

authors tend to lapse into a singular frame of reference that is predictable from their discipline and their indoctrination.

The study of depression is thoroughly fragmented and efforts at integration have been few and generally feeble and unsatisfactory. Investigators in genetics, biochemistry, experimental psychopathology, and epidemiology generally do not stay abreast of developments in other fields that have direct bearing on their own work. Dualistic thinking about the relationship between biological and psychosocial variables has tended to leave psychologists phobic about possible advances in the understanding of the biology of depression, while biologically oriented psychiatrists remain ignorant about the necessity of considering the psychological background and current interpersonal circumstances of depressed persons.

Any successful effort at integration has to confront enormous differences in terminology, interpretation, and emphasis. In a manner that was anticipated by Thomas Kuhn (1970), proponents of the various perspectives on depression are always somewhat at cross-purposes when they attempt to discuss their differences. As will be seen, methods and data are not detachable from theory. To take a simple and basic difference as an example, the cognitive theorist has a commitment to accept the self-report of depressed persons as indicating what these persons are experiencing and wish to convey. The psychodynamic theorist, however, is likely to find such interpretations superficial and would instead be interested in underlying meanings and processes. To the cognitive theorist, “I feel unlovable” is taken at face value, whereas for some psychodynamic theorists, it is probably

best understood as a thinly veiled accusation directed at somebody else.

What is considered most crucial is always determined in light of some theoretical interpretation, and the facts themselves must be reconstituted in terms of this. There is no neutral language in which theoretical differences can be discussed to the satisfaction of proponents of differing viewpoints. These viewpoints are not distinct from the data their proponents muster; they are the way in which the data are seen. One might insist that the perspectives be compared in terms of accuracy, consistency, scope, simplicity, and fruitfulness. These criteria are vital; however, there are often disagreements about how they are to be applied, and the criteria themselves are often in conflict with each other (Kuhn, 1977).

It may be that it is currently too much to

expect a successful wholesale integration of the perspectives that we are going to consider. Dyrud (1974) has cautioned that if we attempt a premature smoothing of differences in terms and concepts, we will lose whatever precision has been achieved. Perhaps what will prove most fruitful for now is development and refinement *within* these perspectives.

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