Structured Group Psychotherapy for Bipolar Disorder

The Life Goals Program

Second Edition

Mark S. Bauer
Linda McBride

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Dr. Bauer: To my pals, Beth, and Nick and Maggie.

Ms. McBride: For my children, Ryan and Kate, and for Bud.

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Contents

Foreword by Gregory E. Simon and Evette J. Ludman *ix*

Acknowledgments *xi*

Introduction *xiii*

Part I Overview of Manic-Depressive Disorder

1 Diagnosis of Manic-Depressive Disorder 3

2 Impact of Manic-Depressive Disorder 20

3 Pathological Basis of Manic-Depressive Disorder 35

4 Overview of Biological Treatments for Manic-Depressive Disorder 52

5 Psychosocial Treatments for Manic-Depressive Disorder 88

6 The Conceptual Framework for the Life Goals Program 115

Part II The Life Goals Program

7 Phase 1: Illness Management Skills 143

Session 1: Orientation 155
Session 2: Mania Part 1 169
Session 3: Mania Part 2 177
Session 4: Depression, Part 1 189
Session 5: Depression, Part 2 197
Session 6: Treatments for Manic-Depressive Disorder 209

8 The Life Goals Program Phase 2 233

9 Program Evaluation 299

References *329*

Index *369*

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Foreword

During the last 5 years, a growing body of research has demonstrated the benefits of several specific psychotherapy programs for people with manic-depressive disorder. Although these programs differ in length and format, common elements include structured education regarding manic-depressive disorder and its treatment, use of techniques from cognitive and behavioral psychotherapy, and training in specific self-management skills. This good news regarding structured and specific psychotherapies for manic-depressive disorder, however, has not reached most mental health providers. Bauer and McBride's guide to their Life Goals Program is an effort to spread the word.

Bauer and McBride's program is based on a series of core principles for effective, patient-centered care for manic-depressive disorder. First is the belief that educating and activating people with manic-depressive disorder is the key to long-term management. Effective treatment depends on collaboration between providers and educated, motivated patients. Developing collaborative treatment relationships inside and outside the group is an important focus of therapy. Second is the focus on structured problem-solving techniques. One might view the problem-solving method as a distillation of the essential elements of cognitive-behavioral therapy: breaking problems down into small pieces, identifying specific solutions, focusing on small and specific steps, and evaluating results. The problem-solving methods emphasized in the Life Goals Program are especially appropriate for people struggling with a chronic illness such as manic-depressive disorder. Participants are encouraged to recognize and celebrate positive steps toward long-term goals. Each action plan is seen as a trial or experiment, with the expectation that plans must be continuously revised and refined. The most important principle is that that people with manic-depressive disorder (even those who are severely ill or disabled) are doing their best to manage the symptoms of mood disorder. Even behaviors usually viewed as maladaptive or even criminal (such as suicide attempts or substance use) can be viewed as attempts to manage mood symptoms. The Life Goals Program attempts to support more effective self-management by examining positive and negative consequences

of various coping strategies. For example: rather than immediately condemning or forbidding substance use, the group leader asks members to carefully consider how well it works. Those involved in the care of people with manic-depressive disorder (both family members and health care providers) would probably welcome an alternative to scolding and ultimatums in the management of disruptive or self-destructive behaviors.

Over the last 2 years, we have used the first edition of *The Life Goals Program* as part of a population-based effort to improve care for people with manic-depressive disorder in our health care system. For the majority of people with manic-depressive disorder (even those in treatment for many years) Phase 1 of the program was a valuable basic course in effective medical management and self-management. Many of those continuing in the long-term Phase 2 program described remarkable growth in the skills and self-confidence needed for effective self-management.

The second edition maintains the philosophy and structure of the first while incorporating some important improvements. Didactic information for the initial group sessions includes more specifics regarding types of pharmacotherapy and types of psychotherapy. Information regarding medication management has been thoroughly updated since the first edition in 1996. The revised program includes increased attention to stigmatization of psychiatric disorders and how stigma can affect one's choices for self-management and collaboration with health care providers. More specific information is included regarding self-care strategies such as maintenance of regular sleep–wake cycles. A new session has been added to the Phase 1 portion of the program to focus on long-term treatment planning (including establishing collaborative relationships with mental health providers and use of peer support resources such as the Depression and Bipolar Support Alliance). All participants are encouraged to create a detailed care plan—to be regularly re-evaluated and updated during Phase 2 of the program. Group leaders should find the revised format more user-friendly than that of the first edition.

The second edition of Bauer and McBride's book should serve a variety of audiences. For mental health providers or systems hoping to establish an organized psychoeducational group program, this detailed manual will prove invaluable. In an ideal world, this program or a similar one would be available through every mental health center or clinic serving people with manic-depressive disorder. When a full-scale group program is not a realistic option, the Life Goals Program manual should still be a useful resource for clinicians, patients, and family members.

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Introduction

The dual purposes of the Life Goals Program have not changed since the publication of the first edition in 1996. First, the Program assists individuals with bipolar disorder to participate more effectively in the management of their illness. Second, since the substantial social and occupational morbidity caused by manic-depressive disorder does not necessarily, or even regularly, improve once the symptoms of the illness are under control, the Program also addresses directly the functional deficits caused by the illness.

However, since the publication of the first edition of this book in 1996 much has changed in the field of evaluation and treatment of manic-depressive disorder. For the Life Goals therapist to be able to treat individuals effectively, he or she must be current in these areas. Hence the need for updating the "primer" on manic-depressive disorder that comprises the first part of this book.

In addition, there have been substantive changes to the Life Goals procedures themselves. Though not changing either the focus or the content of the program, the additional years of experience at our site and with an increasing number of collaborators in North America and Europe have led us to make changes that improve the delivery of the intervention.

First, what has *not* changed in the area of manic-depressive disorder? It is clear that the disorder has not gone away. The basic diagnostic criteria have not changed. All available information indicates that the morbidity from the disorder has not lessened. There is no evidence that its prevalence is decreasing. In fact, although there is also no firm evidence that the prevalence of the disorder has increased since 1996, the frequency with which it is discussed in the media and socially makes one wonder whether it is being recognized more frequently than in the past.

In addition, we do not yet have breakthrough data on the underlying pathophysiology of the disorder. Although there are intriguing leads, particularly in the fields of genetics and imaging (summarized in chapter 3), we are at the stage of better understanding the complexity of the questions than at the stage of being able to provide sophisticated, definitive answers to the question, "What causes manic-depressive disorder?"

What, then, is sufficiently new to warrant publication of a revised, updated edition of this book? Although there have been no major changes in the diagnosis or outcome of manic-depressive disorder, a new awareness of the role of comorbid, or co-occurring, disorders has developed, as reviewed in chapter 1. In addition, we now have a better, though still evolving, understanding of what characteristics will predict better versus worse outcomes for this disorder. In particular, as outlined in chapter 2, there is an increased sensitivity to the importance of ongoing depressive symptoms, even at low levels, on social and occupational function and conceptually, we continue with regard to specific individuals to ask the unanswerable question "How much of the continued functional deficits are due to ongoing depression, and how much of the continued depression is due to ongoing social and occupational impoverishment?"

In terms of treatment, there has been substantial progress in the development of new interventions for manic-depressive disorder, both pharmacologically and psychotherapeutically. Pharmacologically, as reviewed in detail in chapter 4, we have seen the introduction of several new anticonvulsants that have been applied to treatment of this disorder; among anticonvulsants only carbamazepine and valproate were widely used at the time that the first edition was published. In addition, several new atypical neuroleptics have been introduced; at the time of the first edition, only clozapine and risperidone were available. The application of this expanding group of compounds to symptoms of mania and hypomania holds substantial promise both in terms of different side effect profiles and in terms of efficacy compared to older neuroleptics.

Moreover, we as a field have become more sophisticated in evaluating scientific evidence as the tools of *evidence-based medicine* are applied with increasing reach and sophistication to mental health therapeutics. As a result, in choosing among available interventions for those whom we treat, we can identify and take into account evidence deficits and bias both from scientific sources and from the increasingly virulent marketing strategies to which we all—providers and consumers alike—are subject. This evidence-based mental health approach is apparent in chapters 4 and 5, overviews of biological and psychosocial treatments of manic-depressive disorder, each of which has been completely rewritten for this edition.

Perhaps even more dramatic than the expansion of our pharmacologic armamentarium since 1996 is the explosion of data on psychosocial interventions for bipolar disorder. Although no intervention studies suggest that psychosocial interventions can be successfully used without a pharmacologic component to treatment, it is becoming increasingly obvious that optimal pharmacologic, medical-model treatment requires psychosocial management as well. Chapter 5 takes an evidence-based approach in reviewing these psychosocial interventions, both older studies analyzed together for the first time and many studies published since 1996.

Two aspects that derive from this review of psychosocial interventions deserve particular mention. First, when one compares the methods side by side, it becomes apparent that despite their diverse formats, many share a common core agenda. This agenda focuses on:

- education regarding the illness and options for treatment in general
- recognition of one's own pattern of illness
- identification of symptom triggers
- helpful versus harmful coping responses
- development of a personally tailored plan of action for response to various symptoms.

If it sounds like the Life Goals Program, it is because we and many others have converged on this core agenda. Whether given within an individual, group, or family framework, and whether provided from a predominantly cognitive, educative, or other approach, the consistency of the core agenda across interventions is striking.

Second, we purposely speak of psychosocial rather than psychotherapeutic interventions in chapter 5 because of our recognition that the manner in which care is organized can comprise a major help, or hindrance, to optimal treatment for individuals with serious mental illness, including manic-depressive disorder. Accordingly, there have developed several studies, including two federally funded randomized controlled trials currently under way, that provide information on how systems of care can best facilitate the delivery of treatment for individuals with manic-depressive disorder. These studies are reviewed in chapters 5 and 6 and represent a new dimension of thinking about treatment for such illnesses—and a particularly exciting area of future investigation because it may provide new levers for improving outcome in this difficult, chronic illness.

Although the conceptual bases of the Life Goals Program have not changed since its inception, chapter 6 has been extensively rewritten. This has been done both to simplify and clarify the conceptual sources enumerated in the first edition of this book and to give more explicit acknowledgment to commonalities between the Life Goals Program and the several disease management programs for chronic medical illnesses that have contributed to the current form of this program. The point is made more explicitly throughout the book, but especially in chapters 3 and 6, that manic-depressive disorder is best conceptualized as a *biopsychosocial* disorder (Engel, 1977)—and so therefore must be its treatment.

The major open conceptual issue at this point is the degree to which the success of the Life Goals Program in improving outcome is a function only of the intervention itself versus the degree to which its success depends on the context of care in which it is given. For instance, as outlined in chapter 5,

in the Veterans Affairs study all aspects of treatment for manic-depressive illness are brought into an integrated disease management program. In the Group Health Cooperative of Puget Sound study, the individual in treatment collaborates with a registered nurse, who then relays information and guideline-derived management recommendations to various psychiatrists not specially trained in managing the disorder. In a study funded by the Canadian government that is just getting under way at this time in Toronto under the direction of Sagar Parikh, MD, the Life Goals Program is being given as a stand-alone intervention.

In each of these cases, the individuals in treatment are being educated and supported by the Life Goals Program. Will they find their providers to be willing and capable collaborators? Can the Life Goals Program exert some beneficial effect even if they do not? In perhaps the ultimate test of this question, we have recently begun work with a large health maintenance organization to incorporate many of the Life Goals Program components into an Internet-based package for education of individuals with manic-depressive disorder who inhabit that side of the "digital divide." If the principles used by the Life Goals Program and by similar interventions discussed in chapter 5 can exert a beneficial effect in such "supply side" or "pull marketing" interventions, it will provide a truly powerful and widely applicable method to improve outcome and quality of care for this disorder.

Finally, to arm the therapist with current and accurate information about all relevant aspects of manic-depressive disorder, we continue to strive to walk the line between in-depth scholarship and day-to-day utility. Accordingly, we have attempted to make the text of the first part of the book simple and readable, yet have provided an extensive primary source bibliography in the References section for those desiring more in-depth information. As in the first edition, we urge the Life Goals therapist to become familiar with Frederick Goodwin and Kay Redfield Jamison's definitive *Manic-Depressive Illness* (1990), currently being updated, as a comprehensive scholarly text.

In terms of the Life Goals Program procedures themselves, although the focus of the program remains intact, several changes have been made to improve the clarity of the content and ease of delivery. These changes evolve from additional years of personal experience leading groups and the generous suggestions offered by our colleagues and group members, both in the United States and abroad.

The recommended time frame for facilitating Phase 1 and Phase 2 sessions has increased from 60 to 75 minutes, although the pace of each session is determined by therapist judgment and how well group members integrate the session process and content. This time adjustment provides adequate time to deliver Phase 1 Focus Points and Phase 2 behavioral and interpersonal interventions to more impaired group members.

Sessions 1 through 5 have received several minor revisions, as detailed below. Sessions on depression and mania have been reordered based on feedback that commencing the program with sessions on depression was discouraging to group members. The two sessions on mania now precede sessions on depression. A new Session 6, "Treatments for Manic-Depressive Disorder." has been added. A comprehensive Personal Treatment Plan now completes the group psychoeducation component of Phase 1.

In Session 1, the Mood Disorders Spectrum has been reformatted to improve the representation of mood cycling and the intensity of depressive episodes, notably in manic-depressive disorder type II. Additionally, the years have enhanced our insight regarding the range of personal social and intrapsychic manifestations of psychiatric stigma and its tremendous influence on how group members may integrate or reject learning illness management skills. Therefore, psychiatric stigma is more thoroughly processed to increase group member awareness of how tradition and culture may influence their coping responses.

The integration of life events and the impact of stress on mood episode intensity and recurrence has been expanded in the sessions on mania and depression. Personal mania and depression profiles in sessions 2 and 4 now incorporate feedback from family, friends, and coworkers who may offer valuable insights. Many of the session exhibits have been reformatted to facilitate group member use in their everyday lives.

Session 6 provides group members with a summary structure for identifying specific goals of treatment for manic-depressive disorder with an emphasis on self-management and collaboration. Emphasis is placed not only on the medication regime but also on such collaborative and self-management tasks as integration of a structured daily routine, sleep–wake cycle regulation, and the pertinent role of specific psychotherapies to improve mood stability and overall functioning. The session concludes with each group member completing a detailed Personal Care Plan.

Phase 2 retains its behavioral and cognitive orientation to facilitate group member personal goal attainment plans, supplemented with low-intensity interpersonal and psychodynamic therapeutic interventions. An orientation session has been added to the manual to illustrate therapist delivery of the main components in Phase 2. This session fills the gap identified by many of our colleagues during the transition from the completely structured didactic format of Phase 1 to the semi-structured format of Phase 2. The orientation session clarifies strategies to cultivate a supportive group milieu and the ongoing integration of illness management skills learned in Phase 1. It introduces the concepts associated with goal attainment to assist group members as they navigate the identification and development of realistic and meaningful personal life goals.

The stages of goal attainment have been more explicitly operationalized. They now include the Description of the Challenge, overall Goal Identification, Subgoal Development, Construction of Behavioral Steps and Monitoring Progress and Troubleshooting Roadblocks. These changes are intended to facilitate ease of application by the group members, many of whom initially feel overwhelmed by the expectation they will succeed in achieving personal life goals. The term *Challenge* replaces the customary term *problem* at the request of group members working toward improving their self-image.

The section on Therapist Roadblocks and Strategies is revised and includes vignettes to illustrate detailed application of the ideas described. Table 8.4 has been added, offering a quick reference for therapists as they manage the multitude of therapeutic challenges in the Phase 2 process.

Finally, as in the introduction to the first edition, we offer a note on language. We continue our commitment to recognizing the dignity and equality of those who come to us for help with manic-depressive disorder through the language we use. As in the first edition, the words *patient* and *client* appear seldom if at all. As we noted in the Introduction to the first edition:

> When we refer to persons with a mental illness as "patients," we bundle together their illness with their identity. Certainly there is justification for using this shorthand in clinical situations, as we busily try to communicate in concise yet accurate terms during our busy workdays. However, in disorders of mood, behavior, cognition, and perception the distinction between illness and identity becomes easily blurred. This is the rule rather than the exception for the lay public, and frequently a problem with an individual's family and friends. It is an endemic and profoundly demoralizing problem for the individuals we treat, who not infrequently come to consider themselves little more than the product of their bipolar disorder. And it is a temptation for us as providers also to forget that when we treat bipolar disorder that we are treating an individual with a long and unique life history that has nothing to do with the illness—a life history filled with hopes, aspirations, failures, loves, losses, preferences, likes, dislikes, and everything else that makes of us a unique and dignified human being.
>
> Unfortunately many of the individuals we treat have themselves forgotten this, so overwhelmed are they with the burden of their illness and the impact it has had. Part of our job in rehabilitating is to lend hope, and part of lending hope is to reframe their illness: You are a person, and you carry the burden of an illness that you must and can manage.

Therefore, we prefer to speak of persons, individuals, or group members who happen to have this disorder, rather than more clinical—and often distancing and sometimes dehumanizing—terms like *patient.* Perhaps, if culturally we eventually return to the connotation of the original Latin meaning of the word *patient,* "one who suffers," we will then be able to use the term without its less savory baggage.

What of the more obvious change to use of the term *manic-depressive,* rather than *bipolar,* in the text of this edition? We made this decision at a time when the media is becoming more free in the use of the term "bipolar," and when it appears to be entering common usage. In fact, the National Depressive and Manic-Depressive Association has just changed its name to The Depressive and Bipolar Support Alliance, in large part because of feedback from its members who perceived negative annotations to the term "manic-depressive." Why go "backwards"? Why use the old term?

Reexamining the descriptive data on the disorder in chapter 1 makes it clear that mania and hypomania are not typically the polar opposite of depression. In some classic instances, they can be, and in some individuals for a time during their manic or hypomanic episodes they find themselves euphoric, overly optimistic, and without a care in the world. However, this is by no means the rule—and perhaps such euphoric manic periods may be more the exception now than in the past (for reasons that are open to speculation). Hyperactivation, variously defined, appears to be the core symptom of mania and hypomania, and mood and sense of well-being appear to be much more variable and transient. Moreover, additional data reviewed in chapter 2 indicate that subjective quality of life reported by individuals themselves during an episode of mania or hypomania is unequivocally not better, and by some measures worse, than in normal mood. Thus, if we look for, or expect, individuals with manic-depressive illness to regularly have euphoric highs or productive manic periods, we will miss many diagnoses. This perception is also anecdotally shared by many individuals who suffer from the disorder [see, for example, the first-person account of Hartmann, (2002)]. We will also run the risk of underestimating the morbidity and suffering associated with this illness.

Again, language can be a guide to our perceptions. We choose, therefore, to use the more accurate term *manic-depressive* than the more recently introduced yet less accurate term *bipolar* throughout the the book.

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PART I

Overview of Manic-Depressive Disorder

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Chapter 1

Diagnosis of Manic-Depressive Disorder

CONCEPTUALIZATIONS OF MANIC-DEPRESSIVE DISORDER

Diagnosis is the cornerstone of treatment in psychiatry, as in other areas of medicine. In all fields of mental health, there is increasing awareness of the importance of using specific diagnoses as the basis of developing interventions for persons with mood and behavioral problems.

This is particularly important in the management of manic-depressive disorder, because it can be confused with other conditions that have similar features but vastly different treatments. It is striking, for instance, that a survey of individuals with manic-depressive disorder conducted by the National Depressive and Manic Depressive Association (NDMDA) found that almost half of the sample had had symptoms for at least 5 years but were not diagnosed until after having seen at least three professionals (Lish, Dime-Meenan, Whybrow, Price, & Hirschfeld, 1994). Thus, it is worthwhile to review in some detail the basis for diagnosis of manic-depressive disorder so that the clinician may be comfortable with this critical first step in its treatment.

Diagnosis in psychiatry is based almost exclusively on phenomenology, the descriptive appearance of the syndrome of interest. This is because there are few diagnoses for which the pathophysiology is known or for which valid and reliable diagnostic tests are available.

Over the past several years, as part of the development of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994a, 2000), an extensive effort has been made to review evidence to identify core characteristics and limits of the various psychiatric syndromes, including mood disorders. Of all psychiatric

nosological systems, DSM-IV has had perhaps the highest standards for requiring scientific data for additions, deletions, or modifications of the various syndromes. A sample of these data can be found in the multivolume *Sourcebook for DSM-IV,* published as a companion to the manual itself (Widiger & Frances, 1994).

Diagnosis in manic-depressive disorder is made on the basis of two types of descriptive data: *cross-sectional* and *longitudinal.* Cross-sectional data refer to descriptive aspects of a syndrome that occur at a particular point in time, such as the number and type of depressive symptoms that occur during an episode of depression. Longitudinal data refer to the course of symptoms over time, such as the timing, duration, and recurrence of depressive episodes. Both cross-sectional and longitudinal data are essential for the proper diagnosis of manic-depressive disorder. Frequently, diagnostic errors occur when longitudinal data are neglected and the clinician focuses solely on cross-sectional presentation: "This must be manic-depressive disorder because the person appears manic at the present time," or "This cannot be manic-depressive disorder because the person is depressed now."

This longitudinal orientation is not new. Kraepelin's treatise *Manic-Depressive Insanity and Paranoia* (Kraepelin, 1921; see also Berrios & Hauser, 1988) is a classic in large part because of its emphasis on longitudinal as well as cross-sectional data. Kraepelin described two main types of individuals: those whose illness followed a progressive downhill course and those whose illness remitted and recurred frequently with return to their normal baseline. Kraepelin referred to the former as dementia praecox (early dementia), which we now call schizophrenia, the latter as manic-depressive illness. Kraepelin's use of the term *psychosis* differed from our current use. For Kraepelin, psychosis was an indicator of severity rather than of specific paranoid symptoms or hallucinations. This latter group included almost all severe mood disorders, grouping together persons with severe depressive episodes regardless of whether they experienced mania as well. The key point here is that Kraepelin used not only cross-sectional data (the occurrence of mood episodes) but also longitudinal data (the tendency of mood episodes to remit and recur) to separate mood disorders from schizophrenia.

Leonhard (1979) proposed the distinction between manic-depressive, or bipolar disorder and pure depressive disorder, which has come to be called ***unipolar depression.*** He based this distinction on the occurrence of manic episodes in manic-depression but not in pure depression. He extended evidence of this distinction with the observation that mania tended to occur more frequently in family members of persons with bipolar rather than unipolar disorder. This is an early example of the type of approach used to evaluate evidence in later studies of psychiatric syndromes, (e.g., Robins & Guze, 1970), which eventually served as the basis for evaluating most of the evidence for mood disorder validation for DSM-IV.

Kraepelin's and Leonhard's investigations represent a ***categorical approach*** to diagnosis, in which manic-depressive disorder is clearly separated from schizophrenia, on the one hand, and unipolar depression, on the other. This approach is most successful when only classic cases are considered. In reality, however, there exist many borderline cases in which features of more than one syndrome exist and clear categorization is not possible. In recognition of this reality, Bleuler's classic *Textbook of Psychiatry* (Bleuler, 1924) proposed that manic-depressive disorder and schizophrenia lie on a continuum that has no sharp border, with persons often exhibiting characteristics of both syndromes and evolving a course midway between the two. This ***continuum,*** or ***dimensional, approach*** has led to the identification of ***schizoaffective disorder*** (Blacker & Tsuang, 1992; Levitt & Tsuang, 1988), which shares characteristics with both schizophrenia and manic-depressive or depressive disorders.

Furthermore, within manic-depressive disorder, levels of severity have been recognized, as outlined in more detail below. The severe end of the spectrum is ***type I*** manic-depressive disorder, characterized by depressive episodes plus manic episodes. ***Type II*** manic-depressive disorder is characterized not by manic episodes but by the less severe hypomanic episodes. At the milder end of the spectrum, cyclothymia consists of depressed periods that do not meet criteria for major depression, alternating with hypomania or subsyndromal hypomanic symptoms.

CLINICAL CHARACTERISTICS OF MANIC-DEPRESSIVE DISORDER

Core Characteristics of Manic-Depressive Disorder

The phenomenologic approach to diagnosis of the current DSM system is based most recently on the St. Louis, or Feighner, Criteria (Feighner et al., 1972), which specified phenomenologic criteria for the identification of various psychiatric disorders. The St. Louis Criteria, as well as the closely related Research Diagnostic Criteria (RDC) (Spitzer, Endicott, & Bobins, 1978), served as the diagnostic system for most clinical psychiatric research in the 1970s and early 1980s. They provide a common language for disorder description among investigators and increasing comparability of diagnostic samples across various sites. These research tools became the underpinning of clinical practice, with the descriptive approach to clinical diagnosis formally becoming the basis for diagnosis by the third edition of the DSM and its revision (American Psychiatric Association, 1980, 1987). Currently, the DSM-IV and the closely related International Classification of Diseases (ICD-9-CM; World Health Organization, 1977) serve as the basis for the diagnosis of manic-depressive disorder both in clinical practice and in psychiatric research.

The DSM-based definition of manic-depressive ***disorder*** is built on the identification of individual mood ***episodes*** (Table 1.1). DSM-IV criteria for individual mood episodes are summarized in Tables 1.2 through 1.5 for ***major depressive, manic, mixed,*** and ***hypomanic episodes.*** Criteria for these types of episodes are reviewed in greater detail below. Periods of normal mood are sometimes called ***euthymia.***

For the purposes of this portion of the discussion, it is important to understand that *the diagnosis of manic-depressive disorder derives from the occurrence of individual episodes over time.* Persons who experience a manic, hypomanic, or mixed episode, virtually all of whom also have a history of one or more major depressive episodes (Winokur, Clayton, & Reich, 1969), are diagnosed with manic-depressive disorder. Those who experience major depressive and manic episodes are diagnosed with manic-depressive type I disorder, and those with major depressive and hypomanic (milder manic) episodes are diagnosed with manic-depressive type II disorder.

Mood episodes are discrete periods of altered feeling, thought, and behavior. Typically, they have a distinct onset and offset, beginning over days or weeks and eventually ending gradually after several weeks or months. ***Major depressive episodes*** are defined by discrete periods of depressed or blue mood or loss of interest or pleasure in life that endure over weeks (see Table 1.2). The major symptom of depression is a marked decrease in energy and drive (American Psychiatric Association, 1994a, 2000). Extreme effort may be required to accomplish small tasks and self-care. The depressed person may become completely unable to perform social and occupational roles. Neurovegetative symptoms of depression include disturbances in sleep, appetite, and psychomotor activity. Depressed mood may be experienced as feeling

TABLE 1.1 Summary of Mood Episodes and Mood Disorders

Episode	Disorder
Major depressive episode	Major depressive disorder, single episode
Major depressive episode + major depressive episode	Major depressive disorder, recurrent
Major depressive episode + manic/mixed episode	Manic-depressive (bipolar) disorder, type I
Major depressive episode + hypomanic episode	Manic-depressive (bipolar) disorder, type II
Chronic fluctuations between subsyndromal depression and hypomania	Cyclothymic disorder
Chronic subsyndromal depression	Dysthymic disorder

TABLE 1.2 Criteria for Major Depressive Episode

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation by others (e.g., appears tearful). *Note:* In adolescents, can be irritable mood.
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others).
3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. *Note:* In children, consider failure to make weight gains.
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or specific plan for committing suicide.

B. The symptoms do not meet criteria for a mixed episode (see Table 1.4).

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a substance (e.g., drug of abuse or medication) or a general medical condition (e.g., hypothyroidism).

E. The symptoms are not better accounted for by bereavement (i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation).

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empty, slowed down, irritable, or angry. Often one's perception of self and others becomes distorted. One may feel inadequate or experience an unwarranted sense of worthlessness or guilt.

Depressive episodes in manic-depressive disorder are indistinguishable from those in major depressive disorder. About half of persons with manic-

TABLE 1.3 Criteria for Manic Episode

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least 1 week (or any duration if hospitalization is necessary).
B. During the period of mood disturbance, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:
   1. inflated self-esteem or grandiosity
   2. decreased need for sleep (e.g., feels rested after only 3 hours of sleep)
   3. more talkative than usual or pressure to keep talking
   4. flight of ideas or subjective experience that thoughts are racing
   5. distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli)
   6. increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation
   7. excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments)
C. The symptoms do not meet criteria for a mixed episode (see Table 1.4).
D. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.
E. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or a general medical condition (e.g., hyperthyroidism).

Note: Manic-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, or light therapy) should not count toward a diagnosis of bipolar disorder type I.

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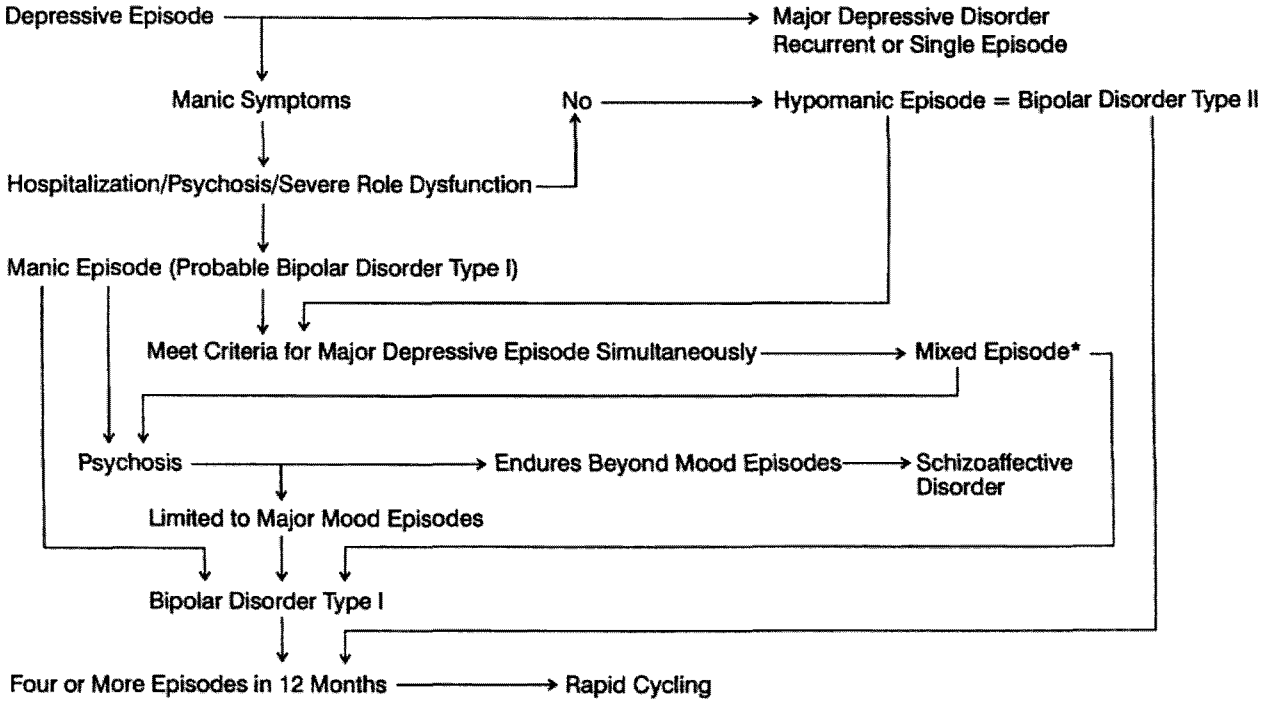
depressive disorder experience depressive episodes characterized by decreased sleep and appetite, and about half experience more atypical symptoms of increased sleep and appetite. Recall that the differential diagnosis between major depressive and manic-depressive disorders is made not by cross-sectional symptom analysis but by longitudinal course. The diagnostic decision tree for manic-depressive disorder is outlined in Figure 1.1.

Manic episodes are defined by discrete periods of abnormally elevated, expansive, or irritable mood accompanied by marked impairment in judgment and social and occupational function. These symptoms are often accompanied by unrealistic grandiosity, excess energy, and increases in goal-directed activity that frequently have a high potential for damaging consequences (see Tables 1.3 and 1.5 for a summary of manic and hypomanic



*Does not apply to hypomanic episode, per DSM-IV, but see McElroy et al, 1992 and Bauer et al, 1994c

FIGURE 1.1 Decision tree for diagnosis of manic-depressive disorder.

The building blocks for a diagnosis of manic-depressive disorder are individual episodes and their characteristics, as summarized in Table 1.1. This decision tree takes the clinician through the steps that lead to diagnosis of manic-depressive disorder and identification of its subtypes, as well as possible course and episode specifiers.

TABLE 1.4 Criteria for Mixed Episode

A. The criteria are met both for a manic episode and for a major depressive episode (except for duration) nearly every day during at least a 1-week period.

B. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

C. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, medication, or other treatment) or a general medical condition (e.g., hyperthyroidism).

Note: Mixed-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, or light therapy) should not count toward a diagnosis of bipolar disorder type I.

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symptoms, respectively). The mood associated with mania is expansive and labile, often combined with euphoria, irritability, or anger (American Psychiatric Association, 1994a, 2000). Mania is also characterized by increased energy and drive, which classically can lead to impulsive buying, dangerous driving, hypersexuality, substance abuse, and arguments. A person with mania has racing thoughts, a flight of ideas, and pressured speech. These behaviors may incur painful consequences, especially social and functional performance decline (e.g., Romans & McPherson, 1992). Often, the expansive mood associated with mania is accompanied by marked distortions in the reality testing called ***psychosis*** (see below).

Hypomanic and manic symptoms are identical, but *hypomanic episodes* are less severe. A person is "promoted" from hypomania to mania (type II to type I manic-depressive disorder) by the presence of one of three features: psychosis during the episode, sufficient severity to warrant hospitalization, or marked social role impairment. This is an imperfect set of criteria, however, because psychosis may or may not be an integral part of manic-depressive disorder (see below), because hospitalization may be due to social or personal factors or comorbidities not related to the disorder itself, and because the concept of marked role function impairment is not well operationalized (reviewed in Bauer, Crits-Christoph, & Whybrow, 1993). From time to time individual authors propose subtypes of manic-depressive disorder in addition to type I and type II, but these are not formally or consistently recognized.

Classically, mania and hypomania have been considered to be the opposite of depression: Individuals with mania were said to be cheery, optimistic, and self-confident. Hence the term *bipolar.* However, in most descriptive

TABLE 1.5 Criteria for Hypomanic Episode

A. A distinct period of persistently elevated, expansive, or irritable mood, lasting throughout at least 4 days, that is clearly different from the usual nondepressed mood.

B. During the period of mood disturbance, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:

1. inflated self-esteem or grandiosity
2. decreased need for sleep (e.g., feels rested after only 3 hours of sleep)
3. more talkative than usual or pressure to keep talking
4. flight of ideas or subjective experience that thoughts are racing
5. distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli)
6. increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation
7. excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., the person engages in unrestrained buying sprees, sexual indiscretions, or foolish business investments)

C. The episode is associated with an unequivocal change in functioning that is uncharacteristic of the person when not symptomatic.

D. The disturbance in mood and the change in functioning are observable by others.

E. The episode is not severe enough to cause marked impairment in social or occupational functioning, or to necessitate hospitalization, and there are no psychotic features.

F. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or a general medical condition (e.g., hyperthyroidism).

Note: Hypomanic-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, or light therapy) should not count toward a diagnosis of bipolar disorder type II.

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studies, a proportion of individuals with mania actually exhibit substantial dysphoric symptoms (reviewed in Bauer et al., 1991). Furthermore, quality of life in mania is worse, rather than better, than in euthymia (Vojta, Kinosian, Glick, Altshuler, & Bauer, 2001; see also chapter 2). Hence, as noted in the Introduction, our decision to return to the more informative and accurate term *manic-depressive disorder.*

Mixed episodes, defined as the simultaneous occurrence of full-blown manic and depressive episodes (see Table 1.4), are the most prominent example of dysphoria during mania. Although it has been suggested that dysphoric mania may comprise a separate subtype of mania, the addition of this

additional dichotomy may be premature, and it may be of more use scientifically and clinically to consider dysphoric symptoms dimensionally rather than categorically (Bauer, Gyulai, Yeh, Gonnel, & Whybrow, 1994).

THE MANIC-DEPRESSIVE, OR BIPOLAR, SPECTRUM

DSM-IV is the first version of the DSM series to recognize formally manic-depressive disorder type II. Previously, persons with depressive and hypomanic episodes were grouped under the broad category of manic-depressive disorder "not otherwise specified," which included a variety of unusual presentations. On the basis of evidence reviewed by Dunner (1993), the disorder was given separate categorical status.

The separation of type II from both type I and major depressive disorder was supported by several types of evidence. For instance, type II disorder occurs more frequently in families of persons with type II, compared to families of persons with type I or with major depressive disorder (Coryell, Endicott, Andreasen, & Keller; Endicott et al., 1985). Study of the course over time of type II disorder indicated that persons with hypomania tended to have recurrent hypomanic episodes, but not convert into type I by developing mania (Coryell et al., 1985). In addition, persons with type II may have more episodes over time than persons with type I (Goodwin & Jamison, 1990), indicating that the course of type II differs from that of type I. However, biological differences between these manic-depressive types have not been reliably demonstrated (Dunner, 1993).

Nonetheless, as outlined below, it should not be construed that type II disorder is in all respects milder than type I, although hypomania is by definition less severe than mania. Specifically, the social and occupational function and quality of life for persons with type II are similar to that for persons with type I disorder, as reviewed in chapter 2.

Persons who experience *subsyndromal* manic-depressive mood fluctuations (i.e., hypomanic and depressive symptoms that do not meet criteria for a full mood episode) over an extended period of time without major mood episodes are diagnosed with *cyclothymic disorder* (Table 1.6). Much less is known about this milder disorder because afflicted persons present for medical attention less frequently than those with full-blown manic-depressive disorder. Cyclothymia has been considered at various times a temperament, a personality disorder, and a disorder at the milder end of the manic-depressive spectrum (Akiskal, 1981). Available data clearly indicate that cyclothymia is related to the more severe manic-depressive disorders (Akiskal, Djenderedjian, Rosenthal, & Khani, 1977; Goodwin & Jamison, 1990). Nonetheless, it is not clear to what degree such categorical disorders may be related to underlying

TABLE 1.6 Diagnostic Criteria for Cyclothymic Disorder

A. For at least 2 years, the presence of numerous periods with hypomanic symptoms and numerous periods with depressive symptoms that do not meet criteria for a major depressive episode. *Note:* In children and adolescents, the duration must be at least 1 year.
B. During the above 2-year period (1 year in children and adolescents), the person has not been without the symptoms in criterion A for more than 2 months at a time.
C. No major depressive episode, manic episode, or mixed episode has been present during the first 2 years of the disturbance.

dimensional characteristics, such as temperament (Akiskal & Akiskal, 1988), however vaguely we are able presently to define that construct.

ADDITIONAL ATTRIBUTES OF MANIC-DEPRESSIVE DISORDER

Psychosis (i.e., delusions or hallucinations; American Psychiatric Association, 1994a, 2000) can occur in either "pole" of the disorder. If psychotic symptoms are limited to the major mood episode, persons are considered to have manic-depressive disorder with psychotic features. If psychotic symptoms endure for at least 2 weeks into periods of normal mood, the diagnosis of *schizoaffective disorder* is made (American Psychiatric Association, 1994a, 2000; Spitzer et al., 1978). However, the 2-week cut point is fairly arbitrary, and its validity is not well established (Blacker & Tsuang, 1992; Levitt & Tsuang, 1988). For example, it may be that psychotic symptoms actually represent a separate, comorbid disorder. Or they may be an integral feature of severe manic-depressive disorder that simply takes longer to resolve. Identification of pathophysiologic and genetic bases of psychosis and of manic-depressive disorder will certainly help to resolve these issues. Delusions may be paranoid, persecutory, punishing, or somatic. Auditory and, less commonly, visual hallucinations and other disruptions of thought processes may occur. Hallucinations may have an authoritative or punitive quality. Psychosis makes it difficult for the person to think and concentrate. Thoughts of death or suicide are common and vary in intensity, from wishing one was dead to frightening auditory hallucinations that command the person to take his or her life.

Rapid cycling is defined if four or more mood episodes occur within 12 months. It should be noted that, despite the name, the episodes are not necessarily or even commonly truly cyclic; the diagnosis is based simply on episode counting (see Table 1.7; see also American Psychiatric Association,

TABLE 1.7 Criteria for Rapid-Cycling Specifier

Specify *with rapid cycling* (can be applied to bipolar disorder type I or type II)

At least four episodes of a mood disturbance in the previous 12 months that meet criteria for a major depressive, manic, mixed, or hypomanic episode.

Note: Episodes are demarcated either by partial or full remission for at least 2 months or a switch to an episode of opposite polarity (e.g., major depressive episode to manic episode).

1994a, 2000; Bauer & Whybrow, 1993). This subcategory is of significance because it predicts a relatively poorer outcome and worse response to lithium and other treatments (Bauer & Whybrow, 1993; Bauer, Calabrese, et al., 1994). Although rapid cycling has been considered by some to be an "end stage" of the disorder, empirical evidence indicates that it may have its onset at any time during the disorder (Bauer & Whybrow, 1993) and may come and go over the course of illness (Bauer, Calabrese, et al., 1994; Coryell, Endicott, & Keller, 1992). Several specific risk factors may be associated with rapid cycling, each of which may give clues to its pathophysiology. These include female gender, antidepressant use, and prior or current hypothyroidism (reviewed in Bauer & Whybrow, 1993).

"PSEUDO-" MANIC-DEPRESSIVE DISORDER

It should be noted that a number of medications and medical conditions commonly encountered in medical and mental health practice can mimic manic-depressive disorder. It is important to recognize these, as the individual may not necessarily have true manic-depressive disorder. Moreover, removing the inciting medication (or treating the underlying medical condition) may lead to the remission of the apparent "mood" symptoms. Although either mania or depression has been reported to be caused either by medications or by medical conditions, it is our experience that it is more common to find certain medications as culprits for mania and medical conditions for depression. These most frequent causes are summarized in Tables 1.8 through 1.11.

MANIC-DEPRESSIVE DISORDER AND DEMOGRAPHIC OR CULTURAL CHARACTERISTICS

There are no major differences in the manifestations of manic-depressive disorder across genders, age groups, or cultures. However, women appear to be

TABLE 1.8 Medical Disorders Commonly Associated with Mania

Neurologic disorders
Stroke
Head trauma
Dementia
Brain tumors
Infection (including HIV and syphilis)
Multiple sclerosis
Huntington's disease
Endocrine disorders
Hyperthyroidism (in those with preexisting manic-depressive disorder)
Postpartum status

at higher risk for depressive episodes in manic-depressive disorder (Liebenluft, 1999), rapid cycling (Bauer, Calabrese, et al., 1994), dysphoria during mania (Bauer, Gyulai, Yeh, Gonnel, & Whybrow, 1994; McElroy et al., 1992), and comorbid disorders (Strakowski, Shelton, & Kolbrener, 1993).

Among children and adolescents, the diagnosis of manic-depressive disorder is often complicated by less consistent mood and behavior baseline than occurs in adults (Carlson & Kashani, 1988). Thus, diagnosis is more difficult, particularly determining whether a child has manic-depressive disorder or attention-deficit disorder or both (reviewed in Wozniak & Biederman, 2001). Onset is rare before puberty. Moreover, little is known currently regarding outcome of and optimal treatment for children and adolescents with manic-depressive disorder.

COMORBIDITY: THE CO-OCCURRENCE OF MANIC-DEPRESSIVE DISORDER WITH OTHER PSYCHIATRIC DISORDERS

Comorbidity refers to disorders or conditions that co-occur with a disorder of interest. Alcohol and drug abuse and dependence represent the most consistently described and most clinically important psychiatric comorbidities with manic-depressive disorder. Although rates of alcohol abuse/dependence run from 3% to 13% in the general population, lifetime rates of alcohol dependence from Epidemiologic Catchment Area (ECA) Study data indicate that rates for alcohol dependence in type I disorder are over 30% (Regier et al., 1990). Furthermore, ECA lifetime rates for drug dependence are over 25%, and rates for any substance abuse or dependence are over 60%. Comparable rates for major depressive disorder in ECA data are, respectively, 12%, 11%,

TABLE 1.9 Medical Disorders Commonly Associated with Depression

Neurologic disorders
- Stroke
- Head trauma
- Dementia
- Brain tumors
- Infection (including HIV and syphilis)
- Multiple sclerosis
- Parkinson's disease
- Huntington's disease

Endocrine disorders
- Addison's disease
- Cushing's disease
- Hypothyroidism
- Hyperthyroidism
- Postpartum status

Cancer
- Pancreatic

Metabolic disorders
- B_{12}, folate deficiencies

Any medical disease that causes significant loss of function or self-esteem

and 27%. Thus, manic-depressive disorder represents an enriched sample for substance use disorders, with substantially greater rates than for general population or even unipolar depression.

A more recent study of a clinical population (those requesting treatment, as opposed to a community sample, which the ECA study investigated) was conducted by the Stanley Foundation Bipolar Network investigators (McElroy et al., 2001). They found lifetime substance abuse rates of 42%, including 33% lifetime alcohol dependence. Current rates were much lower: 4% and 2%, respectively. This may reflect the fact that to be able to present for care (or be accepted into a specialty network like the Stanley Foundation's), such substance use disorders had to be in remission. In a sample of veterans with manic-depressive disorder enrolled in a controlled treatment trial (see chapter 5), we are finding higher lifetime rates of comorbid substance abuse and dependence, on the order of 70% (Kilbourne, Bauer, & Williford, submitted for publication).

In addition, the National Comorbidity Study (Kessler et al., 1997) has found that, among individuals with alcohol dependence, 6.2% of men and 6.8% of women have had a history of mania (rates for a major depressive episode are, respectively, 24.3% and 48.5%). Thus, individuals with alcohol

TABLE 1.10 Treatments and Drugs Associated with Mania

Antidepressants
- Antidepressant drugs
- Bright visible spectrum light treatment
- Electroconvulsant therapy

Adrenergic agents
- Decongestants
- Bronchodilators
- Stimulants

Other agents
- Isoniazid
- Corticosteroids
- Anabolic steroids

Dopaminergic agents
- Levodopa
- Disulfiram

Drugs of abuse
- Alcohol
- Cocaine
- Hallucinogens
- Amphetamines
- Caffeine

dependence comprise an enriched sample for mood disorders, including manic-depressive disorder type I, and screening this population is likely to identify previously undiagnosed individuals with manic-depressive disorder.

The reasons for the high rates of co-occurrence of manic-depressive and substance use disorders are not clear. One hypothesis for this co-occurrence suggests that persons with manic-depressive disorder self-medicate with drugs or alcohol. According to this hypothesis, persons blunt the painful symptoms of depression with drugs (e.g., McLellan, Childress, & Woody, 1985); similarly, they may heighten the manic energy with stimulants (Weiss, Mirin, Griffin, & Michael, 1988). They may also use substances to decrease manic symptoms, particularly if the symptoms are predominantly irritable or dysphoric. Alternatively, chronic substance use may convert otherwise unipolar depression into manic-depressive disorder by inducing substance-induced manic episodes (in DSM-IV, such persons would not be classified as having manic-depressive disorder, but would be considered to have unipolar depression with substance-induced manic episodes). Furthermore, chronic substance use may cause chronic changes in the brain that in turn alter the course of the illness irreversibly, as Himmelhoch and colleagues have proposed (Himmelhoch, Mullar, Neil, Detre, & Kupfer, 1976).

Finally, it is possible that some common genetic predisposition for mood instability is associated both with manic-depressive mood phenomenology and increased craving for substances, and the predominant expression of the predisposition is then determined by other genetic or environmental factors. According to this hypothesis, some persons possessing the gene develop manic-depressive disorder, some develop substance dependence, and some

TABLE 1.11 Treatments and Drugs Commonly Associated with Depression

High blood pressure medications
- Alpha-methyldopa
- Clonidine

Ulcer medications
- Cimetidine
- Ranitidine

Drugs of abuse
- Alcohol
- Sedatives
- Amphetamine (withdrawal)
- Cocaine (withdrawal)
- Nicotine (withdrawal)

Hormones
- Corticosteroids
- Oral contraceptives
- Anabolic steroids

Psychotropic agents
- Benzodiazepines
- Neuroleptics

develop both. Regardless of the mechanism, comorbid substance dependence represents an important clinical challenge for clinicians treating persons with manic-depressive disorder.

Other psychiatric comorbidities have been described in modest proportions of persons with manic-depressive disorder. Interestingly, recent data indicate that comorbidity may be higher in females with manic-depressive disorder than males (Strakowski et al., 1992), which may contribute to the tendency for females to be associated with more complex forms of manic-depressive disorder such as rapid cycling (Bauer, Calabrese, et al., 1994; Bauer & Whybrow, 1993) and dysphoric mania (Bauer, Kurtz, et al., 1994; McElroy et al., 1992). Interestingly, the recent Stanley Foundation study (McElroy et al., 2001) also found high rates of comorbid anxiety disorders (panic, posttraumatic stress disorder, obsessive compulsive disorder, and phobias), with lifetime diagnoses in 42% and current diagnoses in 30% of cases.

COGNITIVE DISORDERS AND DYSFUNCTION IN MANIC-DEPRESSIVE DISORDER

There has been relatively little investigation of cognitive dysfunction in manic-depressive disorder (reviewed in Martinez-Áran, Vieta, Colom, Reinares, & Benabarre, 2000). There are several reasons for highlighting this issue, however. First, there is an accumulating body of evidence from imaging studies that brain structures may be abnormal in measurable ways that tell us about the pathogenesis of the illness (see chapter 3). Second, and of

more direct clinical relevance to treatment, if cognitive function is compromised in subtle ways in individuals with manic-depressive disorder, it may be that their ability to comply with treatment is also subtly compromised.

Martinez-Áran and coworkers (2000) found in reviewing the literature that various types of cognitive dysfunction have been found in individuals with manic-depressive disorder, including problems with memory and executive function (organizing and planning behaviors). Although these deficits tend to improve with remission of symptoms, up to one third of individuals may have deficits that persist in periods of normal mood as well. Consistent with this, Zubieta, Huguelet, O'Neil, and Giordani (2001) found executive dysfunction as well as motor coordination deficits and deficits in verbal learning in individuals with manic-depressive disorder not in mood episodes.

Importantly, data from Savard, Rey, and Post (1980) indicate that older individuals with manic-depressive disorder have greater deficits cognitively than younger individuals. Whether this is a function of increased time ill with manic-depressive disorder or simply the combination of effects of manic-depressive disorder and aging has yet to be elucidated.

Ali and coworkers (2000) found that neuropsychological deficits may be associated with increases in size in the right hippocampus in manic-depressive disorder. However, as with all promising imaging studies that find an association with function, this will require replication.

Overall, then, although manic-depressive disorder does not have the relentless downhill course that Kraepelin (1921) proposed for schizophrenia (and which led him to call that disorder dementia praecox), one cannot rule out the possibility that individuals with manic-depressive disorder may have cognitive compromise to at least a mild degree, even during periods of euthymia. In particular, executive function deficits may interfere with treatment planning and compliance. Clearly, such issues must be taken into consideration when assessing and developing treatment plans for individuals with manic-depressive disorder.

Chapter 2

Impact of Manic-Depressive Disorder

This chapter summarizes the impact of manic-depressive disorder both on the individual and on society. To understand personal impact, one must understand the pattern of onset and the course of the disorder, as well as its associated morbidity, mortality, and personal costs. These sequelae are discussed in terms of ***outcome.*** To understand the societal impact of manic-depressive disorder, one must understand both its epidemiological characteristics and the costs borne by society for the disorder.

EPIDEMIOLOGIC STUDIES OF MANIC-DEPRESSIVE DISORDER

Epidemiology assesses the ***incidence*** (onset frequency), ***prevalence*** (overall population load), and related characteristics of a disorder. Epidemiological studies typically employ large samples of persons who are found in the community but do not necessarily come into clinical care.

These studies are a valuable complement to clinic-based studies. For example, epidemiological studies avoid biases inherent in studying clinic-based samples. For example, clinical populations may underrepresent the milder (or most severe) variants of a disorder; furthermore, willingness to request clinical care may be associated with sample characteristics that bias the sample in unknown ways. In addition, epidemiological studies may be helpful in determining overall population load for a particular disorder, which can be helpful in planning health services. On the other hand, the large sample size and required methodology for most epidemiological studies limit the extent to which any individual subject can be assessed. Smaller higher intensity, clinic-based samples serve as the basis for our most fine-grained phenomenological data and virtually all of our neurobiologic data.