MENTAL ILLNESS

GOALS AND OBJECTIVES

Course Description
"Mental Illness" is a home study continuing education course for rehabilitation professionals. This course presents current information (epidemiology, etiology, symptomology, and treatment) about commonly diagnosed mental illnesses including anxiety disorders, mood disorders, and schizophrenia.

Course Rationale
The purpose of this course is to present rehabilitation professionals with current information about mental illness. Both therapists and therapy assistants will find this information pertinent and useful when providing rehabilitative care to individuals who have, or may have, a concurring mental illness in addition to their primary therapy related diagnosis.

Course Goals and Objectives
Upon completion of this course, the therapist or assistant will be able to
1. identify and differentiate the symptoms and diagnosis criteria associated with each of the anxiety disorders, mood disorders, and schizophrenia.
2. identify and differentiate the pharmaceutical and psychological therapies currently utilized to treat each of the anxiety disorders, mood disorders, and schizophrenia.
3. identify and differentiate the etiology of anxiety disorders, mood disorders, and schizophrenia.
4. identify risk factors associated with mental illness.
5. recognize the factors that act as barriers to the receipt of treatment.
6. recognize the financial impact mental illness has on society.

Course Instructor
Michael Niss PT

Target Audience
Occupational Therapists, occupational therapist assistants, physical therapists, physical therapist assistants

Course Educational Level
This course is applicable for introductory learners.

Course Prerequisites
None

Criteria for issuance of Continuing Education Credits
A documented score of 70% or greater on the written post-test.

Continuing Education Credits
Five (5) hours of continuing education credit (5 NBCOT PDUs/5 contact hours)
AOTA - .5 AOTA CEU, Category 1: Domain of OT – Client Factors, Context

Determination of Continuing Education Contact Hours
"Mental Illness" has been established to be a 5 hour continuing education program. This determination is based on an accepted standard for home-based self-study courses of 12 pages of text (12 pt font) per hour. The complete instructional text for this course is 65 pages (excluding Post-Test).
# Outline

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Overview

A widely used standard of mental health is the absence of a defined mental disorder. This standard has its limitations, yet remains useful for epidemiological purposes. Surveys estimate that about 80 percent of the adult population of the United States do not have a mental disorder during a year and hence may be considered "mentally healthy" (i.e., absence of a mental disorder) during any given year (Kessler et al., 1994). Thus, the popular notion that everyone is “dysfunctional” is far from the truth. Yet, from time to time, many adults experience mental health problems.

Mental health is characterized by the successful performance of mental function, enabling individuals to cope with adversity and to flourish in their education, vocation, and personal relationships. An assortment of traits or personal characteristics have been viewed as contributing to mental health, including self-esteem, optimism, and resilience (Beardslee & Vaillant, 1997). These and related traits are seen as sources of personal resilience needed to weather the storms of stressful life events.

Stressful life events include the breakup of intimate romantic relationships, death of a family member or friend, economic hardship, role conflict, work overload, racism and discrimination, poor physical health, accidental injuries, and intentional assaults on physical safety. Stressful life events in adulthood also may reflect past events. Severe trauma in childhood, including sexual and physical abuse, may persist as a stressor into adulthood, or may make the individual more vulnerable to ongoing stresses. Although some kinds of stressful life events are encountered almost universally, certain demographic groups have greater exposure and/or vulnerability to their cumulative impact. These groups include women, younger adults, unmarried adults, African Americans, and individuals of lower socioeconomic status (Miranda & Green, 1999).

Anxiety disorders are the most prevalent mental disorders in adults. The anxiety disorders affect twice as many women as men. A broad category, anxiety disorders include panic disorder, phobias, obsessive-compulsive disorder, post-traumatic stress disorder, and generalized anxiety disorder, among others. Underlying this heterogeneous group of disorders is a state of heightened arousal or fear in relation to stressful events or feelings. The biological manifestations of anxiety, which are grounded in the “fight-or-flight” response, are unmistakable: they include surge in heart rate, sweating, and tensing of muscles. But this is certainly not the whole picture. Although the full array of biological causes and correlates of anxiety are not yet in our grasp, numerous effective treatments for anxiety disorders exist now. Treatment draws on an assortment of psychosocial and pharmacological approaches, administered alone or in combination.
Mood disorders take a monumental toll in human suffering, lost productivity, and suicide. Moreover, when unrecognized, they can result in unnecessary health care use. Mood disorders rank among the top 10 causes of worldwide disability (Murray & Lopez, 1996). Major depression and bipolar disorder are the most familiar mood disorders, but there are others including cyclothymia (alternating manic and depressive states that do not meet criteria for bipolar disorder) and dysthymia (a chronic, albeit symptomatically milder form of depression). The causes of mood disorders are not fully known. They may be triggered by stressful life events and enduring stressful social conditions (e.g., poverty and discrimination). With the exception of bipolar disorder, they too, like the anxiety disorders, are twice as common in women as men. One subtype of mood disorder, seasonal affective disorder, in which episodes of depression tend to occur in the late fall and winter, is seven times more common in women than in men. Many psychosocial and genetic factors interact to dictate the appearance and persistence of mood disorders.

Schizophrenia affects about 1 percent of the population, yet its severity and persistence reverberate throughout the mental health service system. Schizophrenia is marked by profound alterations in cognition and emotion. Symptoms frequently include hearing internal voices or experiencing other sensations not connected to an obvious source (hallucinations) and assigning unusual significance or meaning to normal events or holding false personal beliefs (delusions). The course of illness in schizophrenia is quite variable, with most people having periods of exacerbation and remission. Schizophrenia had once been thought to have a uniformly downhill course, but recent research dispels this view. Long-term follow up studies show that many individuals with schizophrenia significantly improve and some recover (Harding et al., 1992). Although the causes of schizophrenia are not fully known, research points to the prominent role of genetic factors and to the impact of adverse environmental influences during early brain development (Andreasen, 1997b). New pharmacological treatments are at least as effective as past pharmacological treatments with fewer troubling side effects.

Epidemiology

Few families in the United States are untouched by mental illness. Epidemiological estimates have shifted over time because of changes in the definitions and diagnosis of mental health and mental illness. In the early 1950s, the rates of mental illness estimated by epidemiologists were far higher than those of today. One study, for example, found 81.5 percent of the population of Manhattan, New York, to have had signs and symptoms of mental distress (Srole, 1962). This led the authors of the study to conclude that mental illness was widespread. However, other studies began to find lower rates when they used more restrictive definitions that reflected more contemporary views about
mental illness. Instead of classifying anyone with signs and symptoms as being mentally ill, this more recent line of epidemiological research only identified people as mentally ill if they had a cluster of signs and symptoms that, when taken together, impaired people’s ability to function. By 1978, the President’s Commission on Mental Health concluded conservatively that the annual prevalence of specific mental disorders in the United States was about 15 percent. This figure comports with recent estimates of the extent of mental illness in the population.

Adults

The current prevalence estimate is that about 20 percent of the U.S. population are affected by mental disorders during a given year. This estimate comes from two epidemiologic surveys: the Epidemiologic Catchment Area (ECA) study of the early 1980s and the National Comorbidity Survey (NCS) of the early 1990s. Those surveys defined mental illness according to the prevailing editions of the Diagnostic and Statistical Manual of Mental Disorders (i.e., DSM-III and DSM-III-R). The surveys estimate that during a 1-year period, 22 to 23 percent of the U.S. adult population—or 44 million people—have diagnosable mental disorders, according to reliable, established criteria. In general, 19 percent of the adult U.S. population have a mental disorder alone (in 1 year); 3 percent have both mental and addictive disorders; and 6 percent have addictive disorders alone. Consequently, about 28 to 30 percent of the population have either a mental or addictive disorder (Kessler et al., 1994).

Based on data on functional impairment, it is estimated that 9 percent of all U.S. adults have mental disorders and experience some significant functional impairment. Most (7 percent of adults) have disorders that persist for at least 1 year (Regier et al., 1993b). A subpopulation of 5.4 percent of adults is considered to have a “serious” mental illness (SMI). Serious mental illness is a term that generally applies to mental disorders that interfere with some area of social functioning. About half of those with SMI (or 2.6 percent of all adults) were identified as being even more seriously affected, that is, by having “severe and persistent” mental illness (SPMI) (Kessler et al., 1996). This category includes schizophrenia, bipolar disorder, other severe forms of depression, panic disorder, and obsessive-compulsive disorder. These disorders and the problems faced by these special populations with SMI and SPMI are described further in subsequent sections. Among those most severely disabled are the approximately 0.5 percent of the population who receive disability benefits for mental health-related reasons from the Social Security Administration (NAMHC, 1993).

Children and Adolescents

The annual prevalence of mental disorders in children and adolescents is not as well documented as that for adults. About 20 percent of children are estimated to have mental disorders with at least mild functional impairment. Federal
regulations also define a sub-population of children and adolescents with more severe functional limitations, known as “serious emotional disturbance” (SED). Children and adolescents with SED number approximately 5 to 9 percent of children ages 9 to 17.

Not all mental disorders identified in childhood and adolescence persist into adulthood, even though the prevalence of mental disorders in children and adolescents is about the same as that for adults (i.e., about 20 percent of each age population). While some disorders do continue into adulthood, a substantial fraction of children and adolescents recover or “grow out of” a disorder, whereas, a substantial fraction of adults develops mental disorders in adulthood. In short, the nature and distribution of mental disorders in young people are somewhat different from those of adults.

Older Adults

The annual prevalence of mental disorders among older adults (ages 55 years and older) is also not as well documented as that for younger adults. Estimates generated from the ECA survey indicate that 19.8 percent of the older adult population have a diagnosable mental disorder during a 1-year period. Almost 4 percent of older adults have SMI, and just under 1 percent has SPMI (Kessler et al., 1996); these figures do not include individuals with severe cognitive impairments such as Alzheimer’s disease.

Costs of Mental Illness

Mental disorders impose an enormous emotional and financial burden on ill individuals and their families. They are also costly for our Nation in reduced or lost productivity (indirect costs) and in medical resources used for care, treatment, and rehabilitation (direct costs).

Indirect Costs

The indirect costs of all mental illness imposed a nearly $79 billion loss on the U.S. economy. Most of that amount ($63 billion) reflects morbidity costs—the loss of productivity in usual activities because of illness. But indirect costs also include almost $12 billion in mortality costs (lost productivity due to premature death), and almost $4 billion in productivity losses for incarcerated individuals and for the time of individuals providing family care. For schizophrenia alone, the total indirect cost was almost $15 billion. These indirect cost estimates are conservative because they do not capture some measure of the pain, suffering, disruption, and reduced productivity that are not reflected in earnings.
The fact that morbidity costs comprise about 80 percent of the indirect costs of all mental illness indicates an important characteristic of mental disorders: Mortality is relatively low, onset is often at a younger age, and most of the indirect costs are derived from lost or reduced productivity at the workplace, school, and home (Rupp et al., 1998).

**Direct Costs**

Mental health expenditures for treatment and rehabilitation are an important part of overall health care spending but differ in important ways from other types of health care spending. Many mental health services are provided by separate specialty providers—such as psychiatrists, psychologists, social workers, and nurses in office practice—or by facilities such as hospitals, multiservice mental health organizations, or residential treatment centers for children. Insurance coverage of mental health services is typically less generous than that for general health, and government plays a larger role in financing mental health services compared to overall health care.

The United States spends more than $99 billion annually for the direct treatment of mental disorders, as well as substance abuse, and Alzheimer’s disease and other dementias. More than two-thirds of this amount ($69 billion or more than 7 percent of total health spending) is for mental health services.

**Spending by the Public and Private Sectors**

Funding for the mental health service system comes from both public and private sources. Approximately 53 percent ($37 billion) of the funding for mental health treatment come from public payers. Of the 47 percent ($32 billion) of expenditures from private sources, more than half ($18 billion) are from private insurance. Most of the remainder is out-of-pocket payments. These out-of-pocket payments include copayments from individuals with private insurance, copayments and prescription costs not covered by Medicare or Medigap (i.e., supplementary) insurance, and payment for direct treatment from the uninsured or insured who choose not to use their insurance coverage for mental health care.

Among the fastest-rising expenses for mental health services are outpatient prescription drugs, which account for about 9 percent of total mental health direct costs. Although these medications are prescribed in both specialty and general medical sectors, they are increasingly being covered under general medical rather than mental health private insurance benefits.

During the past two decades there have been important shifts in what parties have final responsibility for paying for mental health care. The role of direct state funding of mental health care has been reduced, whereas Medicaid funding of mental health care has grown in relative importance.
MENTAL ILLNESS

Private insurance coverage has played a somewhat more limited role in mental health financing in the past decade. Various cost containment efforts have been pursued aggressively in the private sector through the introduction of managed care. There is also some emerging evidence on the imposition of new benefit limits on coverage for mental health services.

Diagnosis of Mental Illness

A systematic approach to the classification and diagnosis of mental illness has been developed. Diagnosis is essential in all areas of health for shaping treatment and supportive care, establishing a prognosis, and preventing related disability. Diagnosis also serves as shorthand to enhance communication, research, surveillance, and reimbursement.

The diagnosis of mental disorders is often believed to be more difficult than diagnosis of somatic, or general medical, disorders, since there is no definitive lesion, laboratory test, or abnormality in brain tissue that can identify the illness. The diagnosis of mental disorders must rest with the patients’ reports of the intensity and duration of symptoms, signs from their mental status examination, and clinician observation of their behavior including functional impairment. These clues are grouped together by the clinician into recognizable patterns known as syndromes. When the syndrome meets all the criteria for a diagnosis, it constitutes a mental disorder. Most mental health conditions are referred to as disorders, rather than as diseases, because diagnosis rests on clinical criteria. The term “disease” generally is reserved for conditions with known pathology (detectable physical change). The term “disorder,” on the other hand, is reserved for clusters of symptoms and signs associated with distress and disability (i.e., impairment of functioning), yet whose pathology and etiology are unknown.

The standard manual used for diagnosis of mental disorders in the United States is the Diagnostic and Statistical Manual of Mental Disorders. Most recently revised in 1994, this manual now is in its fourth edition (American Psychiatric Association, 1994, hereinafter cited as DSM-IV). The first edition was published in 1952 by the American Psychiatric Association; subsequent revisions, which were made on the basis of field trials, analysis of data sets, and systematic reviews of the research literature, have sought to gain greater objectivity, diagnostic precision, and reliability. For each disorder within a diagnostic class, DSM-IV enumerates specific criteria for making the diagnosis. DSM-IV also lists diagnostic “subtypes” for some disorders. A subtype is a subgroup within a diagnosis that confers greater specificity. DSM-IV is descriptive in its listing of symptoms and does not take a position about underlying causation.

Knowledge about diagnosis continues to evolve. Evolution in the diagnosis of mental disorders generally reflects greater understanding of disorders as well as
the influence of social norms. Years ago, for instance, addiction to tobacco was not viewed as a disorder, but today it falls under the category of “Substance-Related Disorders.” Although DSM-IV strives to cover all populations, it is not without limitations. Diagnosis rests on clinician judgment about whether clients’ symptom patterns and impairments of functioning meet diagnostic criteria. Cultural differences in emotional expression and social behavior can be misinterpreted as “impaired” if clinicians are not sensitive to the cultural context and meaning of exhibited symptoms.

Anxiety Disorders

The anxiety disorders are the most common, or frequently occurring, mental disorders. They encompass a group of conditions that share extreme or pathological anxiety as the principal disturbance of mood or emotional tone. Anxiety, which may be understood as the pathological counterpart of normal fear, is manifest by disturbances of mood, as well as of thinking, behavior, and physiological activity.

Types of Anxiety Disorders

The anxiety disorders include panic disorder, agoraphobia, generalized anxiety disorder, specific phobia, social phobia, obsessive-compulsive disorder, acute stress disorder, and post-traumatic stress disorder. In addition, there are adjustment disorders with anxious features, anxiety disorders due to general medical conditions, substance-induced anxiety disorders, and the residual category of anxiety disorder not otherwise specified.

Anxiety disorders are not only common in the United States, but they are ubiquitous across human cultures (Weissman et al., 1997). There is significant overlap or comorbidity with mood and substance abuse disorders (Magee et al., 1996). The course of these disorders is characterized by relatively early ages of onset, chronicity, relapsing or recurrent episodes of illness, and periods of disability. Although few psychological autopsy studies of adult suicides have included a focus on comorbid conditions, it is likely that the rate of comorbid anxiety in suicide is underestimated. Panic disorder and agoraphobia, particularly, are associated with increased risks of attempted suicide (American Psychiatric Association, 1998).

Panic Attacks and Panic Disorder

A panic attack is a discrete period of intense fear or discomfort that is associated with numerous somatic and cognitive symptoms. These symptoms include palpitations, sweating, trembling, shortness of breath, sensations of choking or smothering, chest pain, nausea or gastrointestinal distress, dizziness or lightheadedness, tingling sensations, and chills or blushing and “hot flashes.” The
attack typically has an abrupt onset, building to maximum intensity within 10 to 15 minutes. Most people report a fear of dying, “going crazy,” or losing control of emotions or behavior. The experiences generally provoke a strong urge to escape or flee the place where the attack begins and, when associated with chest pain or shortness of breath, frequently results in seeking aid from a hospital emergency room or other type of urgent assistance. Yet an attack rarely lasts longer than 30 minutes. Current diagnostic practice specifies that a panic attack must be characterized by at least four of the associated somatic and cognitive symptoms described above. The panic attack is distinguished from other forms of anxiety by its intensity and its sudden, episodic nature. Panic attacks may be further characterized by the relationship between the onset of the attack and the presence or absence of situational factors. For example, a panic attack may be described as unexpected, situationally bound, or situationally predisposed. There are also attenuated or “limited symptom” forms of panic attacks.

Panic attacks are not always indicative of a mental disorder, and up to 10 percent of otherwise healthy people experience an isolated panic attack per year (Klerman et al., 1991). Panic attacks also are not limited to panic disorder. They commonly occur in the course of social phobia, generalized anxiety disorder, and major depressive disorder.

Panic disorder is diagnosed when a person has experienced at least two unexpected panic attacks and develops persistent concern or worry about having further attacks or changes his or her behavior to avoid or minimize such attacks. Whereas the number and severity of the attacks varies widely, the concern and avoidance behavior are essential features. The diagnosis is inapplicable when the attacks are presumed to be caused by a drug or medication or a general medical disorder, such as hyperthyroidism.

Lifetime rates of panic disorder of 2 to 4 percent and 1-year rates of about 2 percent are documented consistently in epidemiological studies (Weissman et al., 1997). Panic disorder is frequently complicated by major depressive disorder and alcoholism and substance abuse disorders (Liebowitz, 1997). Panic disorder is also concomitantly diagnosed, or co-occurs, with other specific anxiety disorders, including social phobia (up to 30 percent), generalized anxiety disorder (up to 25 percent), specific phobia (up to 20 percent), and obsessive-compulsive disorder (up to 10 percent).

Panic disorder is about twice as common among women as men (American Psychiatric Association, 1998). Age of onset is most common between late adolescence and mid-adult life, with onset relatively uncommon past age 50. There is developmental continuity between the anxiety syndromes of youth, such as separation anxiety disorder. Typically, an early age of onset of panic disorder carries greater risks of comorbidity, chronicity, and impairment. Panic disorder is a familial condition and can be distinguished from depressive disorders by family studies (Rush et al., 1998).
Agoraphobia
The ancient term agoraphobia is translated from Greek as fear of an open marketplace. Agoraphobia today describes severe and pervasive anxiety about being in situations from which escape might be difficult or avoidance of situations such as being alone outside of the home, traveling in a car, bus, or airplane, or being in a crowded area.

Most people who present to mental health specialists develop agoraphobia after the onset of panic disorder (American Psychiatric Association, 1998). Agoraphobia is best understood as an adverse behavioral outcome of repeated panic attacks and the subsequent worry, preoccupation, and avoidance. Thus, the formal diagnosis of panic disorder with agoraphobia was established.

The 1-year prevalence of agoraphobia is about 5 percent. Agoraphobia occurs about two times more commonly among women than men (Magee et al., 1996). The gender difference may be attributable to social-cultural factors that encourage, or permit, the greater expression of avoidant coping strategies by women, although other explanations are possible.

Specific Phobias
These common conditions are characterized by marked fear of specific objects or situations. Exposure to the object of the phobia, either in real life or via imagination or video, invariably elicits intense anxiety, which may include a panic attack. Adults generally recognize that this intense fear is irrational. Nevertheless, they typically avoid the phobic stimulus or endure exposure with great difficulty. The most common specific phobias include the following feared stimuli or situations: animals (especially snakes, rodents, birds, and dogs); insects (especially spiders and bees or hornets); heights; elevators; flying; automobile driving; water; storms; and blood or injections.

Approximately 8 percent of the adult population suffers from one or more specific phobias in 1 year. Much higher rates would be recorded if less rigorous diagnostic requirements for avoidance or functional impairment were employed. Typically, the specific phobias begin in childhood, although there is a second “peak” of onset in the middle 20s of adulthood. Most phobias persist for years or even decades, and relatively few remit spontaneously or without treatment.

The specific phobias generally do not result from exposure to a single traumatic event (i.e., being bitten by a dog or nearly drowning). Rather, there is evidence of phobia in other family members and social or vicarious learning of phobias (Cook & Mineka, 1989). Spontaneous, unexpected panic attacks also appear to play a role in the development of specific phobia, although the particular pattern of avoidance is much more focal and circumscribed.

Social Phobia
Social phobia, also known as social anxiety disorder, describes people with
marked and persistent anxiety in social situations, including performances and public speaking (Ballenger et al., 1998). The critical element of the fearfulness is the possibility of embarrassment or ridicule. Like specific phobias, the fear is recognized by adults as excessive or unreasonable, but the dreaded social situation is avoided or is tolerated with great discomfort. Many people with social phobia are preoccupied with concerns that others will see their anxiety symptoms (i.e., trembling, sweating, or blushing); or notice their halting or rapid speech; or judge them to be weak, stupid, or “crazy.” Fears of fainting, losing control of bowel or bladder function, or having one’s mind going blank are also not uncommon. Social phobias generally are associated with significant anticipatory anxiety for days or weeks before the dreaded event, which in turn may further handicap performance and heighten embarrassment.

The 1-year prevalence of social phobia ranges from 2 to 7 percent, although the lower figure probably better captures the number of people who experience significant impairment and distress. Social phobia is more common in women (Wells et al., 1994). Social phobia typically begins in childhood or adolescence and, for many, it is associated with the traits of shyness and social inhibition. A public humiliation, severe embarrassment, or other stressful experience may provoke an intensification of difficulties. Once the disorder is established, complete remissions are uncommon without treatment. More commonly, the severity of symptoms and impairments tends to fluctuate in relation to vocational demands and the stability of social relationships. Preliminary data suggest social phobia to be familial (Rush et al., 1998).

**Generalized Anxiety Disorder**

Generalized anxiety disorder is defined by a protracted (> 6 months’ duration) period of anxiety and worry, accompanied by multiple associated symptoms (DSM-IV). These symptoms include muscle tension, easy fatigability, poor concentration, insomnia, and irritability. In youth, the condition is known as overanxious disorder of childhood. An essential feature of generalized anxiety disorder is that the anxiety and worry cannot be attributable to the more focal distress of panic disorder, social phobia, obsessive-compulsive disorder, or other conditions. Rather, as implied by the name, the excessive worries often pertain to many areas, including work, relationships, finances, the well-being of one’s family, potential misfortunes, and impending deadlines. Somatic anxiety symptoms are common, as are sporadic panic attacks.

Generalized anxiety disorder occurs more often in women, with a sex ratio of about 2 women to 1 man (Brawman-Mintzer & Lydiard, 1996). The 1-year population prevalence is about 3 percent. Approximately 50 percent of cases begin in childhood or adolescence. The disorder typically runs a fluctuating course, with periods of increased symptoms usually associated with life stress or impending difficulties. There does not appear to be a specific familial association for general anxiety disorder. Rather, rates of other mood and anxiety disorders
typically are greater among first-degree relatives of people with generalized anxiety disorder (Kendler et al., 1987).

**Obsessive-Compulsive Disorder**

Obsessions are recurrent, intrusive thoughts, impulses, or images that are perceived as inappropriate, grotesque, or forbidden (DSM-IV). The obsessions, which elicit anxiety and marked distress, are termed “ego-alien” or “ego-dystonic” because their content is quite unlike the thoughts that the person usually has. Obsessions are perceived as uncontrollable, and the sufferer often fears that he or she will lose control and act upon such thoughts or impulses. Common themes include contamination with germs or body fluids, doubts, order or symmetry, or loss of control of violent or sexual impulses.

Compulsions are repetitive behaviors or mental acts that reduce the anxiety that accompanies an obsession or “prevent” some dreaded event from happening (DSM-IV). Compulsions include both overt behaviors, such as hand washing or checking, and mental acts including counting or praying. Not uncommonly, compulsive rituals take up long periods of time, even hours, to complete. For example, repeated hand washing, intended to remedy anxiety about contamination, is a common cause of contact dermatitis.

Although once thought to be rare, obsessive-compulsive disorder has now been documented to have a 1-year prevalence of 2.4 percent. Obsessive-compulsive disorder is equally common among men and women.

Obsessive-compulsive disorder typically begins in adolescence to young adult life (males) or in young adult life (females) (DSM-IV). For most, the course is fluctuating and, like generalized anxiety disorder, symptom exacerbations are usually associated with life stress. Common comorbidities include major depressive disorder and other anxiety disorders. Approximately 20 to 30 percent of people in clinical samples with obsessive-compulsive disorder report a past history of tics, and about one-quarter of these people meet the full criteria for Tourette’s disorder (DSM-IV). Conversely, up to 50 percent of people with Tourette’s disorder develop obsessive-compulsive disorder (Pitman et al., 1987).

Obsessive-compulsive disorder has a clear familial pattern and somewhat greater familial specificity than most other anxiety disorders. Furthermore, there is an increased risk of obsessive-compulsive disorder among first-degree relatives with Tourette’s disorder. Other mental disorders that may fall within the spectrum of obsessive-compulsive disorder include trichotillomania (compulsive hair pulling), compulsive shoplifting, gambling, and sexual behavior disorders (Hollander, 1996). The latter conditions are somewhat discrepant because the compulsive behaviors are less ritualistic and yield some outcomes that are pleasurable or gratifying. Body dysmorphic disorder is a more circumscribed condition in which the compulsive and obsessive behavior centers around a preoccupation with one’s appearance (i.e., the syndrome of imagined ugliness).
Acute and Post-Traumatic Stress Disorders
Acute stress disorder refers to the anxiety and behavioral disturbances that
develop within the first month after exposure to an extreme trauma. Generally,
the symptoms of an acute stress disorder begin during or shortly following the
trauma. Such extreme traumatic events include rape or other severe physical
assault, near-death experiences in accidents, witnessing a murder, and combat.
The symptom of dissociation, which reflects a perceived detachment of the mind
from the emotional state or even the body, is a critical feature. Dissociation also
is characterized by a sense of the world as a dreamlike or unreal place and may
be accompanied by poor memory of the specific events, which in severe form is
known as dissociative amnesia. Other features of an acute stress disorder
include symptoms of generalized anxiety and hyperarousal, avoidance of
situations or stimuli that elicit memories of the trauma, and persistent, intrusive
recollections of the event via flashbacks, dreams, or recurrent thoughts or visual
images.

If the symptoms and behavioral disturbances of the acute stress disorder persist
for more than 1 month, and if these features are associated with functional
impairment or significant distress to the sufferer, the diagnosis is changed to
post-traumatic stress disorder. Post-traumatic stress disorder is further defined in
DSM-IV as having three subforms: acute (< 3 months' duration), chronic (> 3
months' duration), and delayed onset (symptoms began at least 6 months after
exposure to the trauma).

By virtue of the more sustained nature of post-traumatic stress disorder (relative
to acute stress disorder), a number of changes, including decreased self-esteem,
loss of sustained beliefs about people or society, hopelessness, a sense of being
permanently damaged, and difficulties in previously established relationships, are
typically observed. Substance abuse often develops, especially involving alcohol,
marijuana, and sedative-hypnotic drugs.

About 50 percent of cases of post-traumatic stress disorder remit within 6
months. For the remainder, the disorder typically persists for years and can
dominate the sufferer's life. A longitudinal study of Vietnam veterans, for
example, found 15 percent of veterans to be suffering from post-traumatic stress
disorder 19 years after combat exposure (McFarlane & Yehuda, 1996). In the
general population, the 1-year prevalence is about 3.6 percent, with women
having almost twice the prevalence of men (Kessler et al., 1995). The highest
rates of post-traumatic stress disorder are found among women who are victims
of crime, especially rape, as well as among torture and concentration camp
survivors (Yehuda, 1999). Overall, among those exposed to extreme trauma,
about 9 percent develop post-traumatic stress disorder (Breslau et al., 1998).
Etiology of Anxiety Disorders

The etiology of most anxiety disorders, although not fully understood, has come into sharper focus in the last decade. In broad terms, the likelihood of developing anxiety involves a combination of life experiences, psychological traits, and/or genetic factors. The anxiety disorders are so heterogeneous that the relative roles of these factors are likely to differ. Some anxiety disorders, like panic disorder, appear to have a stronger genetic basis than others (National Institute of Mental Health, 1998), although actual genes have not been identified. Other anxiety disorders are more rooted in stressful life events.

It is not clear why females have higher rates than males of most anxiety disorders, although some theories have suggested a role for the gonadal steroids. Other research on women's responses to stress also suggests that women experience a wider range of life events (e.g., those happening to friends) as stressful as compared with men who react to a more limited range of stressful events, specifically those affecting themselves or close family members.

What the myriad of anxiety disorders have in common is a state of increased arousal or fear (Barbee, 1998). Anxiety disorders often are conceptualized as an abnormal or exaggerated version of arousal. Much is known about arousal because of decades of study in animals and humans of the so-called “fight-or-flight response,” which also is referred to as the acute stress response.

The term “stress” refers either to the external stressor, which can be physical or psychosocial in nature, as well as to the internal response to the stressor. Yet researchers distinguish the two, calling the stressor the stimulus and the body’s reaction the stress response. This is an important distinction because in many anxiety states there is no immediate external stressor.

Acute Stress Response

When a fearful or threatening event is perceived, humans react innately to survive: they either are ready for battle or run away (hence the term “fight-or-flight response”). The nature of the acute stress response is all too familiar. Its hallmarks are an almost instantaneous surge in heart rate, blood pressure, sweating, breathing, and metabolism, and a tensing of muscles. Enhanced cardiac output and accelerated metabolism are essential for mobilizing fast action. The host of physiological changes activated by a stressful event are unleashed in part by activation of a nucleus in the brain stem called the locus ceruleus. This nucleus is the origin of most norepinephrine pathways in the brain. Neurons using norepinephrine as their neurotransmitter project bilaterally from the locus ceruleus along distinct pathways to the cerebral cortex, limbic system, and the spinal cord, among other projections.

Normally, when someone is in a serene, unstimulated state, the “firing” of neurons in the locus ceruleus is minimal. A novel stimulus, once perceived, is
relayed from the sensory cortex of the brain through the thalamus to the brain stem. That route of signaling increases the rate of noradrenergic activity in the locus ceruleus, and the person becomes alert and attentive to the environment. If the stimulus is perceived as a threat, a more intense and prolonged discharge of the locus ceruleus activates the sympathetic division of the autonomic nervous system. The activation of the sympathetic nervous system leads to the release of norepinephrine from nerve endings acting on the heart, blood vessels, respiratory centers, and other sites. The ensuing physiological changes constitute a major part of the acute stress response. The other major player in the acute stress response is the hypothalamic-pituitary-adrenal axis, which is discussed in the next section.

In the 1980s, the prevailing view was that excess discharge of the locus ceruleus with the acute stress response was a major contributor to the etiology of anxiety (Coplan & Lydiard, 1998). Yet over the past decade, the limitations of the acute stress response as a model for understanding anxiety have become more apparent. The first and most obvious limitation is that the acute stress response relates to arousal rather than anxiety. Anxiety differs from arousal in several ways (Nutt et al., 1998). First, with anxiety, the concern about the stressor is out of proportion to the realistic threat. Second, anxiety is often associated with elaborate mental and behavioral activities designed to avoid the unpleasant symptoms of a full-blown anxiety or panic attack. Third, anxiety is usually longer lived than arousal. Fourth, anxiety can occur without exposure to an external stressor.

Other limitations of this model became evident from a lack of support from clinical and basic research (Coplan & Lydiard, 1998). Furthermore, with its emphasis on the neurotransmitter norepinephrine, the model could not explain why medications that acted on the neurotransmitter serotonin (the selective serotonin reuptake inhibitors, or SSRIs) helped to alleviate anxiety symptoms. In fact, these medications are becoming the first-line treatment for anxiety disorders (Kent et al., 1998). To probe the etiology of anxiety, researchers began to devote their energies to the study of other brain circuits and the neurotransmitters on which they rely. The locus ceruleus still participates in anxiety but is understood to play a lesser role.

**Anatomical and Biochemical Basis of Anxiety**

An exciting new line of research proposes that anxiety engages a wide range of neurocircuits. This line of research catapults to prominence two key regulatory centers found in the cerebral hemispheres of the brain—the hippocampus and the amygdala. These centers, in turn, are thought to activate the hypothalamic-pituitary-adrenocortical (HPA) axis (Sullivan et al., 1998). Researchers have long established the contribution of the HPA axis to anxiety but have been perplexed by how it is regulated. They are buoyed by new findings about the roles of the hippocampus and the amygdala.
The hippocampus and the amygdala govern memory storage and emotions, respectively, among their other functions. The hippocampus is considered important in verbal memory, especially of time and place for events with strong emotional overtones. The hippocampus and amygdala are major nuclei of the limbic system, a pathway known to underlie emotions. There are anatomical projections between the hippocampus, amygdala, and hypothalalamus (Coplan & Lydiard, 1998).

Anxiety differs from fear in that the fear-producing stimulus is either not present or not immediately threatening, but in anticipation of danger, the same arousal, vigilance, physiologic preparedness, and negative affects and cognitions occur. Different types of internal or external factors or triggers act to produce the anxiety symptoms of panic disorder, agoraphobia, post-traumatic stress disorder, specific phobias, and generalized anxiety disorder, and the prominent anxiety that commonly occurs in major depression. It is currently a matter of research to determine whether dysregulation of these fear pathways leads to the symptoms of anxiety disorders. It has now been established, using noninvasive neuroimaging, that the human amygdala is also involved in fear responses. Fearful facial expressions have been shown to activate the amygdala in MRI studies of normal human subjects (Breiter et al., 1996). Functional imaging studies in anxiety disorders, such as PET studies of brain activation in phobias (Rauch et al., 1995), are also beginning to investigate the precise neural circuits involved in the anxiety disorders.

There are many neurotransmitter alterations in anxiety disorders. At least five neurotransmitters are altered in anxiety: serotonin, norepinephrine, gamma-aminobutyric acid (GABA), corticotropin-releasing hormone (CRH), and cholecystokinin (Rush et al., 1998). There is such careful orchestration between these neurotransmitters that changes in one neurotransmitter system invariably elicit changes in another, including extensive feedback mechanisms. Serotonin and GABA are inhibitory neurotransmitters that quiet the stress response. All of these neurotransmitters have become important targets for therapeutic agents either already marketed or in development.

**Psychological Views of Anxiety**
There are several major psychological theories of anxiety: psychoanalytic and psychodynamic theory, behavioral theories, and cognitive theories. Psychodynamic theories have focused on symptoms as an expression of underlying conflicts (Thorn et al., 1999).

More recent behavioral theories have emphasized the importance of two types of learning: classical conditioning and vicarious or observational learning. These theories have some empirical evidence to support them. In classical conditioning, a neutral stimulus acquires the ability to elicit a fear response after repeated pairings with a frightening (unconditioned) stimulus. In vicarious learning, fearful behavior is acquired by observing others’ reactions to fear-inducing stimuli. With
general anxiety disorder, unpredictable positive and negative reinforcement is seen as leading to anxiety, especially because the person is unsure about whether avoidance behaviors are effective.

Cognitive factors, especially the way people interpret or think about stressful events, play a critical role in the etiology of anxiety. A decisive factor is the individual’s perception, which can intensify or dampen the response. One of the most salient negative cognitions in anxiety is the sense of uncontrollability. It is typified by a state of helplessness due to a perceived inability to predict, control, or obtain desired results (Barlow et al., 1996). Negative cognitions are frequently found in individuals with anxiety (Ingram et al., 1998). Many modern psychological models of anxiety incorporate the role of individual vulnerability, which includes both genetic (Smoller & Tsuang, 1998) and acquired (Coplan et al., 1997) predispositions. There is evidence that women may ruminate more about distressing life events compared with men, suggesting that a cognitive risk factor may predispose them to higher rates of anxiety and depression.

**Treatment of Anxiety Disorders**

The anxiety disorders are treated with some form of counseling or psychotherapy or pharmacotherapy, either singly or in combination (American Psychiatric Association, 1998).

**Counseling and Psychotherapy**

Anxiety disorders are responsive to counseling and to a wide variety of psychotherapies. More severe and persistent symptoms also may require pharmacotherapy.

During the past several decades, there has been increasing enthusiasm for more focused, time-limited therapies that address ways of coping with anxiety symptoms more directly rather than exploring unconscious conflicts or other personal vulnerabilities (Barlow & Lehman, 1996). These therapies typically emphasize cognitive and behavioral assessment and interventions.

The hallmarks of cognitive-behavioral therapies are evaluating apparent cause and effect relationships between thoughts, feelings, and behaviors, as well as implementing relatively straightforward strategies to lessen symptoms and reduce avoidant behavior. A critical element of therapy is to increase exposure to the stimuli or situations that provoke anxiety. Without such therapeutic assistance, the sufferer typically withdraws from anxiety-inducing situations, inadvertently reinforcing avoidant or escape behavior.

The therapist provides reassurance that the feared situation is not deadly and introduces a plan to enhance mastery. This plan may include approaching the feared situation in a graduated or stepwise hierarchy or teaching the patient to use responses that dampen anxiety, such as deep muscle relaxation or coping.
One fundamental principle is that prolonged exposure to a feared stimulus reliably decreases cognitive and physiologic symptoms of anxiety. With such experience generally comes greater self-efficacy and a greater willingness to encounter other feared stimuli. For panic disorder, interoceptive training (a type of conditioning technique) and breathing exercises are often employed to help the sufferer become more capable of recognizing and coping with the social cues, antecedents, or early signs of a panic attack. Cognitive interventions are used to counteract the exaggerated or catastrophic thoughts that characterize anxiety. For treatment of obsessive-compulsive disorder, the strategy of response prevention must be added to exposure to ensure that compulsions are not performed (Barlow, 1988).

There is now extensive evidence that cognitive-behavioral therapies are useful treatments for a majority of patients with anxiety disorders (Chambless et al., 1998). Poorer outcomes are observed, however, in more complicated patient groups. With obsessive-compulsive disorder, approximately 20 to 25 percent of patients are unwilling to participate in therapy (March et al., 1997). Another major limitation of cognitive-behavioral therapies is not their effectiveness but, rather, the limited availability of skilled practitioners (Ballenger et al., 1998).

It is possible that more traditional forms of therapy based on psychodynamic or interpersonal theories of anxiety also may prove to be effective treatments. However, these therapies have not yet received extensive empirical support. As a result, more traditional therapies are generally deemphasized in evidence-based treatment guidelines for anxiety disorders.

**Pharmacotherapy for Anxiety Disorders**

The medications typically used to treat patients with anxiety disorders are benzodiazepines, antidepressants, and the novel compound buspirone (Lydiard et al., 1996). In light of increasing awareness of numerous neurochemical alterations in anxiety disorders, many new classes of drugs are likely to be developed, expressly targeting CRH and other neuroactive agents (Nemeroff, 1998).

**Benzodiazepines** - The benzodiazepines are a large class of relatively safe and widely prescribed medications that have rapid and profound antianxiety and sedative-hypnotic effects. The benzodiazepines are thought to exert their therapeutic effects by enhancing the inhibitory neurotransmitter systems utilizing GABA. Benzodiazepines bind to a site on the GABA receptor and act as receptor agonists.

The four benzodiazepines currently widely prescribed for treatment of anxiety disorders are diazepam, lorazepam, clonazepam, and alprazolam. Each is now available in generic formulations. Among these agents, alprazolam and lorazepam have shorter elimination half-lives—that is, are removed from the body more quickly—while diazepam and clonazepam have a long period of
action (i.e., up to 24 hours). Diazepam also has multiple active metabolites, which increase the risk of “carryover” effects such as sedation and “hangover.” Benzodiazepines that undergo conjugation appear to have longer elimination time in women, and oral contraceptive can decrease clearance (Dawlans, 1995).

Benzodiazepines have the potential for producing drug dependence (i.e., physiological or behavioral symptoms after discontinuation of use). Shorter acting compounds have somewhat greater liability because of more rapid and abrupt onset of withdrawal symptoms.

Because the benzodiazepines do not have strong antiobsessional effects, their use in obsessive-compulsive disorder and post-traumatic stress disorder is generally viewed as palliative (i.e., relieving, but not eliminating symptoms). Rather, obsessive-compulsive disorder and post-traumatic stress disorder are more effectively treated by antidepressants, especially the SSRIs (as discussed below). When effective, benzodiazepines should be tapered after several months of use, although there is a substantial risk of relapse. Many clinicians favor a combined treatment approach for panic disorder and generalized anxiety disorder, in which benzodiazepines are used acutely in tandem with an antidepressant. The benzodiazepines are subsequently tapered as the antidepressant’s therapeutic effects begin to emerge (American Psychiatric Association, 1998).

Antidepressants - Most antidepressant medications have substantial antianxiety and antipanic effects in addition to their antidepressant action (Kent et al., 1998). Moreover, a large number of antidepressants have antiobsessional effects (Perry et al., 1997). The observation that the tricyclic antidepressant imipramine had a different anxiolytic profile than diazepam helped to differentiate panic disorder from generalized anxiety disorder and, subsequently, social phobia.

Clomipramine, a tricyclic antidepressant (TCA) with relatively potent reuptake inhibitory effects on serotonin (5-HT) neurons, subsequently was found to be the only TCA to have specific antiobsessional effects (March et al., 1997). The importance of this effect on 5-HT was highlighted when the SSRIs became available. By the late 1990s, it became clear that all of the SSRIs have antiobsessional effects (Kent et al., 1998).

Current practice guidelines rank the TCAs below the SSRIs for treatment of anxiety disorders because of the SSRIs' more favorable tolerability and safety profiles (American Psychiatric Association, 1998). Nevertheless, there are patients who respond to the TCAs after failing to respond to one or more of the newer agents. Similarly, although relatively rarely used, the monoamine oxidate inhibitors (MAOIs) have significant antiobsessional, antipanic, and anxiolytic effects. In the United States, the MAOIs phenoelzine, tranylcypromine, and isocarboxazid (which has not been consistently marketed this decade) are
seldom used unless simpler medication strategies have failed (American Psychiatric Association, 1998).

The five drugs within the SSRI class—fluoxetine, sertraline, paroxetine, fluvoxamine, and citalopram—have emerged as the preferred type of antidepressant for treatment of anxiety disorders. In addition to well-established efficacy in obsessive-compulsive disorder, there is convincing and growing evidence of antipanic and broader anxiolytic effects (American Psychiatric Association, 1998). Treatment of panic disorder often requires lower initial doses and slower upward titration. By contrast, treatment for obsessive-compulsive disorder ultimately may entail higher doses and longer durations to achieve desired outcomes (March et al., 1997).

Other newer antidepressants, including venlafaxine, nefazodone, and mirtazapine, also may have significant antianxiety effects. Paroxetine has been approved by the Food and Drug Administration (FDA) for social phobia, and sertraline is being developed for post-traumatic stress disorder. Nefazodone, which also is being studied in post-traumatic stress disorder, and mirtazapine may possess lower levels of sexual side effects, a problem that complicates longer term treatment with SSRIs, venlafaxine, TCAs, and MAOIs (Baldwin & Birtwistle, 1998).

When effective in treating anxiety, antidepressants should be maintained for at least 4 to 6 months, then tapered slowly to avoid discontinuation-emergent activation of anxiety symptoms (March et al., 1997; American Psychiatric Association, 1998; Ballenger et al., 1998). Although less extensively researched than depression, it is likely that many patients with anxiety disorders may warrant longer term, indefinite treatment to prevent relapse or chronicity.

**Buspirone** - This azopyrine compound is a relatively selective 5- HT_{1A} partial agonist (Stahl, 1996). It was approved by the FDA in the mid-1980s as an anxiolytic. However, unlike the benzodiazepines, buspirone is not habit forming and has no abuse potential. Buspirone also has a safety profile comparable to the SSRIs, and it is significantly better tolerated than the TCAs.

Buspirone does not block panic attacks, and it is not efficacious as a primary treatment of obsessive-compulsive disorder or post-traumatic stress disorder (Stahl, 1996). Buspirone is most useful for treatment of generalized anxiety disorder, and it is now frequently used as an adjunct to SSRIs. Buspirone takes 4 to 6 weeks to exert therapeutic effects, like antidepressants, and it has little value for patients when taken on an "as needed" basis.
Mood Disorders

Each year, about 7 percent of Americans suffer from mood disorders, a cluster of mental disorders best recognized by depression or mania. Mood disorders are outside the bounds of normal fluctuations from sadness to elation. They have potentially severe consequences for morbidity and mortality.

This section covers four mood disorders; major depressive disorder (also known as unipolar major depression), bipolar disorder, dysthymia, and cyclothymia.

Mood disorders rank among the top 10 causes of worldwide disability (Murray & Lopez, 1996). Unipolar major depression ranks first, and bipolar disorder ranks in the top 10. Moreover, disability and suffering are not limited to the patient. Spouses, children, parents, siblings, and friends experience frustration, guilt, anger, financial hardship, and, on occasion, physical abuse in their attempts to assuage or cope with the depressed person’s suffering. Women between the ages of 18 and 45 comprise the majority of those with major depression (Regier et al., 1993).

Depression also has a deleterious impact on the economy, both in diminished productivity and in use of health care resources. In the workplace, depression is a leading cause of absenteeism and diminished productivity. Although only a minority seek professional help to relieve a mood disorder, depressed people are significantly more likely than others to visit a physician for some other reason. Depression-related visits to physicians thus account for a large portion of health care expenditures. Seeking another or a less stigmatized explanation for their difficulties, some depressed patients undergo extensive and expensive diagnostic procedures and then get treated for various other complaints while the mood disorder goes undiagnosed and untreated (Wells et al., 1989).

Types and Classification of Mood Disorders

Major Depressive Disorder

Major depressive disorder features one or more major depressive episodes each of which lasts at least 2 weeks (DSM-IV). Since these episodes are also characteristic of bipolar disorder, the term “major depression” refers to both major depressive disorder and the depression of bipolar disorder.

The cardinal symptoms of major depressive disorder are depressed mood and loss of interest or pleasure. Other symptoms vary enormously. For example, insomnia and weight loss are considered to be classic signs, even though many depressed patients gain weight and sleep excessively. Such heterogeneity is partly dealt with by the use of diagnostic subtypes with differing presentations and prevalence. For example, a more severe depressive syndrome characterized by a constellation of classical signs and symptoms, called melancholia, is more common among older than among younger people, as are depressions.
MENTAL ILLNESS

characterized by psychotic features (i.e., delusions and hallucinations) (DSM-IV). The so-called reversed vegetative symptoms (oversleeping, overeating, and weight gain) may be more prevalent in women than men (Nemeroff, 1992). Anxiety symptoms such as panic attacks, phobias, and obsessions also are not uncommon.

When untreated, a major depressive episode may last, on average, about 9 months. Eighty to 90 percent of individuals will remit within 2 years of the first episode (Kapur & Mann, 1992). Thereafter, at least 50 percent of depressions will recur, and after three or more episodes the odds of recurrence within 3 years increases to 70 to 80 percent if the patient has not had preventive treatment (Thase & Sullivan, 1995). Thus, for many, an initial episode of major depression will evolve over time into the more recurrent illness sometimes referred to as unipolar major depression (Thase & Sullivan, 1995). Each new episode also confers new risks of chronicity, disability, and suicide.

DSM-IV 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.

1. depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). In children and 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. In children, consider failure to make expected weight gains.
4. insomnia or hypersomnia nearly every day.
5. psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings or 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 or guilt about being sick).
8. diminished ability to think or concentrate, or indecisiveness, nearly every day (either 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 a specific plan for committing suicide.

B. The symptoms do not meet criteria for a mixed episode.

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., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).

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.

**Dysthymia**

Dysthymia is a chronic form of depression. Its early onset and unrelenting, “smoldering” course are among the features that distinguish it from major depressive disorder (DSM-IV). Dysthymia becomes so intertwined with a person’s self-concept or personality that the individual may be misidentified as “neurotic”. Indeed, the onset of dysthymia in childhood or adolescence undoubtedly affects personality development and coping styles, particularly prompting passive, avoidant, and dependent “traits.” The term “dysthymia” is used in DSM-IV as a descriptive, or atheoretical, diagnosis for a chronic form of depression. Affecting about 2 percent of the adult population in 1 year, dysthymia is defined by its subsyndromal nature (i.e., fewer than the five persistent symptoms required to diagnose a major depressive episode) and a protracted duration of at least 2 years for adults and 1 year for children. Like other early-onset disorders, dysthymic disorder is associated with higher rates of comorbid substance abuse. People with dysthymia also are susceptible to major depression. When this occurs, their illness is sometimes referred to as “double depression,” that is, the combination of dysthymia and major depression (Keller & Shapiro, 1982). Unlike the superimposed major depressive episode, however, the underlying dysthymia seldom remits spontaneously. Women are twice as likely to be diagnosed with dysthymia as men (Robins & Regier, 1991).

**DSM-IV diagnostic criteria for Dysthymic Disorder**

A. Depressed mood for most of the day, for more days than not, as indicated either by subjective account or observation by others, for at least 2 years. In children and adolescents, mood can be irritable and duration must be at least 1 year.

B. Presence, while depressed, of two (or more) of the following:
1. poor appetite or overeating
2. insomnia or hypersomnia
3. low energy or fatigue
4. low self-esteem
5. poor concentration or difficulty making decisions
6. feelings of hopelessness

C. During the 2-year period (1 year for children or adolescents) of the disturbance, the person has never been without the symptoms in Criteria A and B for more than 2 months at a time.

D. No major depressive episode has been present during the first 2 years of the disturbance (1 year for children and adolescents); i.e., the disturbance is not better accounted for by chronic major depressive disorder, or major depressive disorder, in partial remission.

There may have been a previous major depressive episode provided there was a full remission (no significant signs or symptoms for 2 months) before development of the dysthymic disorder. In addition, after the initial 2 years (1 year in children or adolescents) of dysthymic disorder, there may be superimposed episodes of major depressive disorder, in which case both diagnoses may be given when the criteria are met for a major depressive episode.

E. There has never been a manic episode, a mixed episode, or a hypomanic episode, and criteria have never been met for cyclothymic disorder.

F. The disturbance does not occur exclusively during the course of a chronic psychotic disorder, such as schizophrenia or delusional disorder.

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

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

**Bipolar Disorder**

Bipolar disorder is a recurrent mood disorder featuring one or more episodes of mania or mixed episodes of mania and depression (DSM-IV). Bipolar disorder is distinct from major depressive disorder by virtue of a history of manic or hypomanic (milder and not psychotic) episodes. Other differences concern the nature of depression in bipolar disorder. Its depressive episodes are typically associated with an earlier age at onset, a greater likelihood of reversed vegetative symptoms, more frequent episodes or recurrences, and a higher familial prevalence. Another noteworthy difference between bipolar and
nonbipolar groups is the differential therapeutic effect of lithium salts, which are more helpful for bipolar disorder (Goodwin & Jamison, 1990).

Mania is derived from a French word that literally means crazed or frenzied. The mood disturbance can range from pure euphoria or elation to irritability to a labile admixture that also includes dysphoria. Thought content is usually grandiose but also can be paranoid. Grandiosity usually takes the form both of overvalued ideas (e.g., “My book is the best one ever written”) and of frank delusions (e.g., “I have radio transmitters implanted in my head and the Martians are monitoring my thoughts.”) Auditory and visual hallucinations complicate more severe episodes. Speed of thought increases, and ideas typically race through the manic person’s consciousness. Nevertheless, distractibility and poor concentration commonly impair implementation. Judgment also can be severely compromised; spending sprees, offensive or disinhibited behavior, and promiscuity or other objectively reckless behaviors are commonplace. Subjective energy, libido, and activity typically increase but a perceived reduced need for sleep can sap physical reserves. Sleep deprivation also can exacerbate cognitive difficulties and contribute to development of catatonia or a florid, confusional state known as delirious mania. If the manic patient is delirious, paranoid, or catatonic, the behavior is difficult to distinguish from that of a schizophrenic patient. Most people with bipolar disorder have a history of remission and at least satisfactory functioning before onset of the index episode of illness.

In DSM-IV, bipolar depressions are divided into type I (prior mania) and type II (prior hypomanic episodes only). About 1.1 percent of the adult population suffers from the type I form, and 0.6 percent from the type II form (Kessler et al., 1994). Episodes of mania occur, on average, every 2 to 4 years, although accelerated mood cycles can occur annually or even more frequently. The type I form of bipolar disorder is about equally common in men and women, unlike major depressive disorder, which is more common in women.

Hypomania, as suggested above, is the subsyndromal counterpart of mania. By definition, an episode of hypomania is never psychotic nor are hypomanic episodes associated with marked impairments in judgment or performance. In fact, some people with bipolar disorder long for the productive energy and heightened creativity of the hypomanic phase.

Hypomania can be a transitional state (i.e., early in an episode of mania), although at least 50 percent of those who have hypomanic episodes never become manic (Goodwin & Jamison, 1990). Whereas a majority have a history of major depressive episodes (bipolar type II disorder), others become hypomanic only during antidepressant treatment (Goodwin & Jamison, 1990). Despite the relatively mild nature of hypomania, the prognosis for patients with bipolar type II disorder is poorer than that for recurrent (unipolar) major depression, and there is some evidence that the risk of rapid cycling (four or more episodes each year) is greater than with bipolar type I (Coryell et al., 1992). Women are at higher risk for
rapid cycling bipolar disorder than men (Coryell et al., 1992). Women with bipolar disorder are also at increased risk for an episode during pregnancy and the months following childbirth (Blehar et al., 1998).

DSM-IV 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.

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 general medical condition (e.g., hyperthyroidism).

Cyclothymia
Cyclothymia is marked by manic and depressive states, yet neither are of
Mental Illness

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sufficient intensity nor duration to merit a diagnosis of bipolar disorder or major depressive disorder. The diagnosis of cyclothymia is appropriate if there is a history of hypomania, but no prior episodes of mania or major depression. Longitudinal followup studies indicate that the risk of bipolar disorder developing in patients with cyclothymia is about 33 percent; although 33 times greater than that for the general population, this rate of risk still is too low to justify viewing cyclothymia as merely an early manifestation of bipolar type I disorder (Howland & Thase, 1993).

DSM-IV 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. 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.

After the initial 2 years (1 year in children and adolescents) of cyclothymic disorder, there may be superimposed manic or mixed episodes (in which case both bipolar I disorder and cyclothymic disorder may be diagnosed) or major depressive episodes (in which case both bipolar II disorder and cyclothymic disorder may be diagnosed).

D. The symptoms in Criterion A are not better accounted for by schizoaffective disorder and are not superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or psychotic disorder not otherwise specified.

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

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

Etiology of Mood Disorders

The etiology of depression, the mood disorder most frequently studied, is far from ideally understood. Many cases of depression are triggered by stressful life events, yet not everyone becomes depressed under such circumstances. The intensity and duration of these events, as well as each individual’s genetic
endowment, coping skills and reaction, and social support network contribute to the likelihood of depression. That is why depression and many other mental disorders are broadly described as the product of a complex interaction between biological and psychosocial factors. The relative importance of biological and psychosocial factors may vary across individuals and across different types of depression.

**Biologic Factors**

Much of the scientific effort expended over the past 40 years on the study of depression has been devoted to the search for biologic alterations in brain function. Researchers have detected abnormal concentrations of many neurotransmitters and their metabolites in urine, plasma, and cerebrospinal fluid in subgroups of patients (Thase & Howland, 1995); dysregulation of the HPA axis; elevated levels of corticotropin-releasing factor (Mitchell, 1998); and, most recently, abnormalities in second messenger systems and neuroimaging (Steffens & Krishnan, 1998). Much current research focuses on how the biological abnormalities interrelate, how they correlate with behavioral and emotional patterns that seem to distinguish one subcategory of major depression from another, and how they respond to diverse forms of therapy.

In the search for biological changes with depression, it must be understood that a biological abnormality reliably associated with depression may not actually be a causal factor. For example, a biologic alteration could be a consequence of sleep deprivation or weight loss. Any biological abnormality found in conjunction with any mental disorder may be a cause, a correlate, or a consequence. What drives research is the determination to find which of the biological abnormalities in depression are true causes, especially ones that might be detectable and treatable before the onset of clinical symptoms.

**Monoamine Hypothesis** - For many years the prevailing hypothesis was that depression was caused by an absolute or relative deficiency of monoamine transmitters in the brain. This line of research was bolstered by the discovery many years ago that reserpine, a medication for hypertension, inadvertently caused depression. It did so by depleting the brain of both serotonin and the three principal catecholamines (dopamine, norepinephrine, and epinephrine). Such findings led to the “catecholamine hypothesis” and the “indoleamine (i.e., serotonin) hypothesis,” which in due course led to an integrated “monoamine hypothesis” (Thase & Howland, 1995).

After more than 30 years of research, however, the monoamine hypothesis has been found insufficient to explain the complex etiology of depression. One problem is that many other neurotransmitter systems are altered in depression, including GABA and acetylcholine (Rush et al., 1998). Another problem is that improvement of monoamine neurotransmission with medications and lifting of the clinical signs of depression do not prove that depression actually is caused by defective monoamine neurotransmission. To account for these discrepancies,
one new model of depression proposes that depression results from reductions in neurotrophic factors that are necessary for the survival and function of particular neurons, especially those found in the hippocampus (Duman et al., 1997).

Despite the problems with the hypothesis that monoamine depletion is the primary cause of depression, monoamine impairment is certainly one of the manifestations, or correlates, of depression. Therefore, the monoamine hypothesis remains important for treatment purposes. Many currently available pharmacotherapies that relieve depression or cause mania, or both, enhance monoamine activity. One of the foremost classes of drugs for depression, SSRIs, for example, boost the level of serotonin in the brain.

**Anxiety and Depression** - Anxiety and depression frequently coexist, so much so that patients with combinations of anxiety and depression are the rule rather than the exception (Barbee, 1998). And many of the medications used to treat either one are often used to treat the other. Why are anxiety and depression so interrelated?

Overlapping biochemical correlates are found, most notably, an elevation in CRH (Arborelius et al., 1999). Interestingly, one new line of research finds that long-term consequences of anxiety and depression are evident at the same anatomical site—the hippocampus. Human imaging studies of the hippocampus revealed it to have smaller volume in patients with post-traumatic stress disorder (McEwen, 1998) and in patients with recurrent depression (Sheline, 1996). In the latter study, the degree of volume reduction was correlated with the duration of major depression. In both conditions, excess glucocorticoid exposure was thought to be the culprit in inducing the atrophy of hippocampal neurons. But the complete chain of events leading up to and following the hippocampal damage is not yet known.

**Psychosocial Factors**

If stressful events are the proximate causes of most cases of depression, then why is it that not all people become depressed in the face of stressful events? The answer appears to be that social, psychological, and genetic factors act together to predispose to, or protect against, depression.

Adult life can be rife with stressful events, as noted earlier, and although not all people with depression can point to some precipitating event, many episodes of depression are associated with some sort of acute or chronic adversity (Ingram et al., 1998).

The death of a loved one is viewed as one of the most powerful life stressors. The grief that ensues is a universal experience. Common symptoms associated with bereavement include crying spells, appetite and weight loss, and insomnia. Grief, in fact, has such emotional impact that the diagnosis of depressive
disorder should not be made unless there are definite complications such as incapacity, psychosis, or suicidal thoughts.

The compelling impact of past parental neglect, physical and sexual abuse, and other forms of maltreatment on both adult emotional well-being and brain function is now firmly established for depression. Early disruption of attachment bonds can lead to enduring problems in developing and maintaining interpersonal relationships and problems with depression and anxiety. Research in animals bears this out as well. In both rodents and primates, maternal deprivation stresses young animals, and a pattern of repeated, severe, early trauma from maternal deprivation may predispose an animal to a lifetime of overreactivity to stress. Conversely, early experience with mild, nontraumatic stressors (such as gentle handling) may help to protect or “immunize” animals against more pathologic responses to subsequent severe stress.

**Cognitive Factors**

According to cognitive theories of depression, how individuals view and interpret stressful events contributes to whether or not they become depressed. One prominent theory of depression stems from studies of learned helplessness in animals. The theory posits that depression arises from a cognitive state of helplessness and entrapment (Seligman, 1991). The theory was predicated on experiments in which animals were trained in an enclosure in which shocks were unavoidable and inescapable, regardless of avoidance measures that animals attempted. When they later were placed in enclosures in which evasive action could have succeeded, the animals were inactive, immobile, and unable to learn avoidance maneuvers. The earlier experience engendered a behavioral state of helplessness, one in which actions were seen as ineffectual.

In humans there is now ample evidence that the impact of a stressor is moderated by the personal meaning of the event or situation. In other words, the critical factor is the person’s interpretation of the stressor’s potential impact. Thus, an event interpreted as a threat or danger elicits a nonspecific stress response, and an event interpreted as a loss (of either an attachment bond or a sense of competence) elicits more grief-like depressive responses.

Heightened vulnerability to depression is linked to a constellation of cognitive patterns that predispose to distorted interpretations of a stressful event (Ingram et al., 1998). For example, a romantic breakup will trigger a much stronger emotional response if the affected person believes, “I am incomplete and empty without her love,” or “I will never find another who makes me feel the way he does.” The cognitive patterns associated with distorted interpretation of stress include relatively harsh or rigid beliefs or attitudes about the importance of romantic love or achievement as well as the tendency to attribute three specific qualities to adverse events: (1) global impact—“This event will have a big effect on me”; (2) internality—“I should have done something to prevent this,” or “This is my fault”; and (3) irreversibility—“I'll never be able to recover from this.”
According to a recent model of cognitive vulnerability to depression, negative cognitions by themselves are not sufficient to engender depression. This model postulates, on the basis of previously gathered empirical evidence, that interactions between negative cognitions and mildly depressed mood are important in the etiology and recurrences of depression. Patterns or styles of thinking stem from prior negative experiences. When they are activated by adverse life events and a mildly depressed mood, a downward spiral ensues, leading to depression (Ingram et al., 1998).

**Personality and Temperament**

Responses to life events also can be linked to personality. Personality may be understood in terms of one’s attitudes and beliefs as well as more enduring neurobehavioral predispositions referred to as temperaments. The study of personality and temperament is gaining momentum. Neuroticism predisposes to anxiety and depression (Clark et al., 1994). Having an easy-going temperament, on the other hand, protects against depression (IOM, 1994). Further, those with severe personality disorder are particularly likely to have a history of early adversity or maltreatment (Browne & Finkelhor, 1986).

Temperaments are not destiny, however. Parental influences and individual life experiences may determine whether a shy child remains vulnerable or becomes a healthy adult. In adults, several constellations of personality traits are associated with mood disorders: avoidance, dependence, and traits such as reactivity and impulsivity (Hirschfeld & Shea, 1992). People who have such personality traits not only cope less effectively with stressors but also tend to provoke or elicit adversity. A personality disorder or temperamental disturbance may mediate the relationship between stress and depression.

**Gender**

Major depressive disorder and dysthymia are more prevalent among women than men, as noted earlier. This difference appears in different cultures throughout the world (Weissman et al., 1993). Understanding the gender-related difference is complex and likely related to the interaction of biological and psychosocial factors, including differences in stressful life events as well as to personality.

Keys to understanding the sex-related difference in rates in the United States may be found in two types of epidemiologic findings: (1) there are no sex-related differences in rates of bipolar disorder (type I) (NIMH, 1998) and, (2) within the culture of the Amish, the rate of major depressive disorder is both low and equivalent for men and women (Egeland et al., 1983). Something about the environment thus appears to interact with a woman’s biology to cause a disproportionate incidence of depressive episodes among women (Blumenthal, 1994a).

Research conducted in working-class neighborhoods suggests that the combination of life stress and inadequate social support contributes to women’s
greater susceptibility to depressive symptoms (Brown et al., 1994). Because women tend to use more ruminative ways of coping (e.g., thinking and talking about a problem, rather than seeking out a distracting activity) and, on average, have less economic power, they may be more likely to perceive their problems as less solvable. That perception increases the likelihood of feeling helpless or entrapped by one’s problem. Subtle sex-related differences in hemispheric processing of emotional material may further predispose women to experience emotional stressors more intensely (Baxter et al., 1987). Women’s greater vulnerability to depression may be amplified by endocrine and reproductive cycling, as well as by a greater susceptibility to hypothyroidism (Thase & Howland, 1995). Menopause, on the other hand, has little bearing on gender differences in depression. Contrary to popular beliefs, menopause does not appear to be associated with increased rates of depression in women (Pearlstein et al., 1997). Untreated mental health problems are likely to worsen at menopause, but menopause by itself is not a risk factor for depression (Thacker, 1997). The increased risk for depression prenatally or after childbirth suggests a role for hormonal influences, although evidence also exists for the role of stressful life events.

Poor young women (white, black, and Hispanic) appear to be at the greatest risk for depression compared with all other population groups (Miranda & Green, 1999). They have disproportionately higher rates of past exposure to trauma, including rape, sexual abuse, crime victimization, and physical abuse; poorer support systems; and greater barriers to treatment, including financial hardship and lack of insurance (Miranda & Green, 1999). Many of the same problems apply to single mothers, whose risk of depression is double that of married mothers (Brown & Moran, 1997).

The interaction between stressful life events, individual experiences, and genetic factors also plays a role in the etiology of depression in women. Some research suggests that genetic factors, which are discussed below, may alter women’s sensitivity to the depression-inducing effect of stressful life events (Kendler et al., 1995). A recent report of depression in a sample of 2,662 twins found genetic factors in depression to be stronger for women than men, for whom depression was only weakly familial. For both genders, individual environmental experiences played a large role in depression (Bierut et al., 1999).

**Genetic Factors**

Depression, and especially bipolar disorder, clearly tend to “run in families,” and a definite association has been scientifically established (Tsuang & Faraone, 1990). Numerous investigators have documented that susceptibility to a depressive disorder is twofold to fourfold greater among the first-degree relatives of patients with mood disorder than among other people (Tsuang & Faraone, 1990). The risk among first-degree relatives of people with bipolar disorder is about six to eight times greater. Some evidence indicates that first-degree
relatives of people with mood disorders are also more susceptible than other people to anxiety and substance abuse disorders (Tsuang & Faraone, 1990).

Remarkable as those statistics may be, they do not by themselves prove a genetic connection. Inasmuch as first-degree relatives typically live in the same environment, share similar values and beliefs, and are subject to similar stressors, the vulnerability to depression could be due to nurture rather than nature. One method to distinguish environmental from genetic factors is to compare concordance rates among same-sex twins. At least in terms of simple genetic theory, a solely hereditary trait that appears in one member of a set of identical (monozygotic) twins also should always appear in the other twin, whereas the trait should appear only 50 percent of the time in same-sex fraternal (dizygotic) twins.

The results of studies comparing the prevalence of depression among twins vary, depending on the specific mood disorder, the age of the study population, and the way the depression is defined. In all instances, however, the reported concordance for mood disorders is greater among monozygotic than among dizygotic twins, and often the proportion is 2 to 1 (Tsuang & Faraone, 1990). In Denmark, Bertelsen and colleagues (1977) found that among 69 monozygotic twins with bipolar illness, 46 co-twins also had bipolar disorder and 14 other co-twins had psychoses, affective personality disorders, or had died by suicide. In studies of monozygotic twins reared separately, the results also revealed an increased risk of depression and bipolar disorder compared with controls (Wender et al., 1986). Within the major depressive disorder grouping, greater heritable risk has been associated with more severe, recurrent, or psychotic forms of mood disorders. Those at greater heritable risk also appear more vulnerable to stressful life events (Kendler et al., 1995).

The availability of modern molecular genetic methods now allows the translation of clinical associations into identification of specific genes (Baron, 1997). Evidence collected to date strongly suggests that vulnerability to mood disorders may be associated with several genes distributed among various chromosomes. For bipolar disorder, numerous distinct chromosomal regions (called loci) show promise, yet the complex nature of inheritance and methodological problems have encumbered investigators. Heritability in some cases may be sex linked or vary depending on whether the affected parent is the father or mother of the individual being studied.

**Treatment of Mood Disorders**

So much is known about the assortment of pharmacological and psychosocial treatments for mood disorders that the most salient problem is not with treatment, but rather with getting people into treatment.
Surveys consistently document that a majority of individuals with depression receive no specific form of treatment (Thase, 1996). Nearly 40 percent of people with bipolar disorder are untreated in 1 year (Regier et al., 1993). Undertreatment of mood disorders stems from many factors, including societal stigma, financial barriers to treatment, underrecognition by health care providers, and underappreciation by consumers of the potential benefits of treatment. The symptoms of depression, such as feelings of worthlessness, excessive guilt, and lack of motivation, also deter consumers from seeking treatment.

This section describes specific types of pharmacotherapies and psychosocial therapies for episodes of depression and mania. Treatment generally targets symptom patterns rather than specific disorders.

**Pharmacotherapies for Depression**

Antidepressant medications are effective across the full range of severity of major depressive episodes in major depressive disorder and bipolar disorder (American Psychiatric Association, 1993).

There are four major classes of antidepressant medications. The tricyclic and heterocyclic antidepressants (TCAs and HCAs) are named for their chemical structure. The MAOIs and SSRIs are classified by their initial neurochemical effects. In general, MAOIs and SSRIs increase the level of a target neurotransmitter by two distinct mechanisms. But, as discussed below, these classes of medications have many other effects. They also have some differential effects depending on the race or ethnicity of the patient.

The mode of action of antidepressants is complex and only partly understood. Put simply, most antidepressants are designed to heighten the level of a target neurotransmitter at the neuronal synapse. This can be accomplished by one or more of the following therapeutic actions: boosting the neurotransmitter’s synthesis, blocking its degradation, preventing its reuptake from the synapse into the presynaptic neuron, or mimicking its binding to postsynaptic receptors. To make matters more complicated, many antidepressant drugs affect more than one neurotransmitter. Explaining how any one drug alleviates depression probably entails multiple therapeutic actions, direct and indirect, on more than one neurotransmitter system (Feighner, 1999).

Selection of a particular antidepressant for a particular patient depends upon the patient’s past treatment history, the likelihood of side effects, safety in overdose, and expense. A vast majority of U.S. psychiatrists favor the SSRIs as “first-line” medications. These agents are viewed more favorably than the TCAs because of their ease of use, more manageable side effects, and safety in overdose.

Four SSRIs have been approved by the FDA for treatment of depression: fluoxetine, sertraline, paroxetine, and citalopram. A fifth SSRI, fluvoxamine, is approved for treatment of obsessive-compulsive disorder, yet is used off-label for
depression. There are few compelling reasons to pick one SSRI over another for treatment of uncomplicated major depression, because they are more similar than different. There are, however, several distinguishing pharmacokinetic differences between SSRIs, including elimination half-life (the time it takes for the plasma level of the drug to decrease 50 percent from steady-state), propensity for drug-drug interactions (e.g., via inhibition of hepatic enzymes), and antidepressant activity of metabolite(s). In general, SSRIs are more likely to be metabolized more slowly by African Americans and Asians, resulting in higher blood levels (Lin et al., 1997).

The SSRIs as a class of drugs have their own class-specific side effects, including nausea, diarrhea, headache, tremor, daytime sedation, failure to achieve orgasm, nervousness, and insomnia. Attrition from acute phase therapy because of side effects is typically 10 to 20 percent (Preskorn & Burke, 1992).

Some concern persists that the SSRIs are less effective than the TCAs for treatment of severe depressions, including melancholic and psychotic subtypes (Nelson, 1994).

Side effects and potential lethality in overdose are the major drawbacks of the TCAs. An overdose of as little as 7-day supply of a TCA can result in potentially fatal cardiac arrhythmias. TCA treatment is typically initiated at lower dosages and titrated upward with careful attention to response and side effects. Doses for African Americans and Asians should be monitored more closely, because their slower metabolism of TCAs can lead to higher blood concentrations (Lin et al., 1997). Similarly, studies also suggest that there may be gender differences in drug metabolism and that plasma levels may change over the course of the menstrual cycle (Blumenthal, 1994b).

Regardless of the initial choice of pharmacotherapy, about 30 to 50 percent of patients do not respond to the initial medication. It has not been established firmly whether patients who respond poorly to one class of antidepressants should be switched automatically to an alternate class. Several studies have examined the efficacy of the TCAs and SSRIs when used in sequence. Approximately 30 to 50 percent of those not responsive to one class will respond to the other (Thase & Rush, 1997).

Among other types of antidepressants, the MAOIs and bupropion are important alternatives for SSRI and TCA nonresponders (Thase & Rush, 1995). These agents also may be relatively more effective than TCAs or SSRIs for treatment of depressions characterized by atypical or reversed vegetative symptoms. Bupropion and the MAOIs also are good choices to treat bipolar depression.

Bupropion’s efficacy and overall side effect profile might justify its first-line use for all types of depression. Furthermore, bupropion has a novel neurochemical profile in terms of effects on dopamine and norepinephrine. However, worries...
about an increased risk of seizures delayed bupropion’s introduction to the U.S. market by more than 5 years.

Although clearly effective for a broad range of depressions, use of the MAOIs has been limited for decades by concerns that when taken with certain foods containing the chemical tyramine (for example, some aged cheeses and red wines); these medications may cause a potentially lethal hypertensive reaction (Thase et al., 1995). There has been continued interest in development of safer, selective and reversible MAOIs.

**Pharmacotherapies for Mania**

Success rates of 80 to 90 percent were once expected with lithium for the acute phase treatment of mania (Schou, 1989); however, lithium response rates of only 40 to 50 percent are now commonplace (Frances et al., 1996). The apparent decline in lithium responsiveness may be partly due to sampling bias (i.e., university hospitals treat more refractory patients), but could also be attributable to factors such as younger age of onset, increased drug abuse comorbidity, or shorter therapeutic trials necessitated by briefer hospital stay (Solomon et al., 1995). The effectiveness of acute phase lithium treatment also is partially dependent on the clinical characteristics of the manic episode: dysphoric/mixed, psychotic, and rapid cycling episodes are less responsive to lithium alone (DSM-IV; Solomon et al., 1995).

A number of other medications initially developed for other indications are increasingly used for lithium-refractory or lithium-intolerant mania. The efficacy of two medications, the anticonvulsants carbamazepine and divalproex sodium, has been documented (Bowden et al., 1994). Divalproex sodium has received FDA approval for the treatment of mania. The specific mechanisms of action for these agents have not been established.

Another newer treatment, verapamil, is a calcium channel blocker initially approved by the FDA for treatment of cardiac arrhythmias and hypertension. Since the mid-1980s, clinical reports and evidence from small randomized clinical trials suggest that the calcium channel blockers may have antimanic effects (Janicak et al., 1998). Like lithium and the anticonvulsants, the mechanism of action of verapamil has not been established.

Adjunctive neuroleptics and high-potency benzodiazepines are used often in combination with mood stabilizers to treat mania. The very real risk of tardive dyskinesia has led to a shift in favor of adjunctive use of benzodiazepines instead of neuroleptics for acute stabilization of mania. For manic patients who are not responsive to or tolerant of pharmacotherapy, Electroconvulsive Therapy (ECT) is a viable alternative (Mukherjee et al., 1994).
Electroconvulsive Therapy (ECT)
ECT consists of a series of brief generalized seizures induced by passing an electric current through the brain by means of two electrodes placed on the scalp. A typical course of ECT entails 6 to 12 treatments, administered at a rate of three times per week, on either an inpatient or outpatient basis. The exact mechanisms by which ECT exerts its therapeutic effect are not yet known. The production of an adequate, generalized seizure using the proper amount of electrical stimulation at each treatment session is required for therapeutic efficacy.

With the development of effective medications for the treatment of major mental disorders a half-century ago, the need for ECT lessened but did not disappear. Prior to that time, ECT often had been administered for a variety of conditions for which it is not effective, and administered without anesthesia or neuromuscular blockade. The result was grand mal seizures that could produce injuries and even fractures. Despite the availability of a range of effective antidepressant medications and psychotherapies, as discussed above, ECT continues to be used (Rosenbach et al., 1997), occupying a narrower but important niche. It is generally reserved for the special circumstances where the usual first-line treatments are ineffective or cannot be taken, or where ECT is known to be particularly beneficial, such as depression or mania accompanied by psychosis or catatonia (Potter & Rudorfer, 1993).

Psychotherapy and Counseling
Many people prefer psychotherapy or counseling over medication for treatment of depression. Research conducted in the past two decades has helped to establish at least several newer forms of time-limited psychotherapy as being as effective as antidepressant pharmacotherapy in mild-to-moderate depressions (Persons et al., 1996). The newer depression-specific therapies include cognitive-behavioral therapy and interpersonal psychotherapy. These approaches use a time-limited approach, a present tense (“here-and-now”) focus, and emphasize patient education and active collaboration. Interpersonal psychotherapy centers around four common problem areas: role disputes, role transitions, unresolved grief, and social deficits. Cognitive-behavioral therapy takes a more structured approach by emphasizing the interactive nature of thoughts, emotions, and behavior. It also helps the depressed patient to learn how to improve coping and lessen symptom distress.

There is no evidence that cognitive-behavioral therapy and interpersonal psychotherapy are differentially effective. As reported earlier, both therapies appear to have some relapse prevention effects, although they are much less studied than the pharmacotherapies.
Combined Therapies

Combined therapies — also called multimodal treatments—are especially valuable for outpatients with severe forms of depression. According to a meta-analysis of six studies, combined therapy (cognitive or interpersonal psychotherapy plus pharmacotherapy) was significantly more effective than psychotherapy alone for more severe recurrent depression. In milder depressions, psychotherapy alone was nearly as effective as combined therapy (Thase et al., 1997b).

Prevention and Maintenance

Maintenance pharmacotherapy is the best-studied means to reduce the risk of recurrent depression (Thase & Sullivan, 1995). The magnitude of effectiveness in prevention of recurrent depressive episodes depends on the dose of the active agent used, the inherent risk of the population (i.e., chronicity, age, and number of prior episodes), the length of time being considered, and the patient's adherence to the treatment regimen.

There are few published studies on the prophylactic benefits of long-term pharmacotherapy with SSRIs, bupropion, nefazodone, or venlafaxine. However, available studies uniformly document 1-year efficacy rates of 80 to 90 percent in preventing recurrence of depression (Stewart et al., 1998). Thus, maintenance therapy with the newer agents is likely to yield outcomes comparable to the TCAs (Thase & Sullivan, 1995).

How does maintenance pharmacotherapy compare with psychotherapy? In one study of recurrent depression, monthly sessions of maintenance interpersonal psychotherapy had a 3-year success rate of about 35 percent (i.e., a rate falling between those for active and placebo pharmacotherapy) (Frank et al., 1990).

Schizophrenia

Overview

Our understanding of schizophrenia has evolved since its symptoms were first catalogued by German psychiatrist Emil Kraepelin in the late 19th century. Even though the cause of this disorder remains elusive, its frightening symptoms and biological correlates have come to be quite well defined. Yet misconceptions abound about symptoms: schizophrenia is neither “split personality” nor “multiple personality.” Furthermore, people with schizophrenia are not perpetually incoherent or psychotic (Mason et al., 1997).

Schizophrenia is characterized by profound disruption in cognition and emotion, affecting the most fundamental human attributes: language, thought, perception,
affect, and sense of self. The array of symptoms, while wide ranging, frequently includes psychotic manifestations, such as hearing internal voices or experiencing other sensations not connected to an obvious source (hallucinations) and assigning unusual significance or meaning to normal events or holding fixed false personal beliefs (delusions). No single symptom is definitive for diagnosis; rather, the diagnosis encompasses a pattern of signs and symptoms, in conjunction with impaired occupational or social functioning (DSM-IV).

Symptoms are typically divided into positive and negative symptoms because of their impact on diagnosis and treatment. Positive symptoms are those that appear to reflect an excess or distortion of normal functions (Peralta & Cuesta, 1998). The diagnosis of schizophrenia, according to DSM-IV, requires at least 1-month duration of two or more positive symptoms, unless hallucinations or delusions are especially bizarre, in which case one alone suffices for diagnosis. Negative symptoms are those that appear to reflect a diminution or loss of normal functions (Blanchard et al., 1998). These often persist in the lives of people with schizophrenia during periods of low (or absent) positive symptoms. Negative symptoms are difficult to evaluate because they are not as grossly abnormal as positives ones and may be caused by a variety of other factors as well.

Diagnosis is complicated by early treatment of schizophrenia's positive symptoms. Antipsychotic medications, particularly the traditional ones, often produce side effects that closely resemble the negative symptoms of affective flattening and avolition. In addition, other negative symptoms are sometimes present in schizophrenia but not often enough to satisfy diagnostic criteria: loss of usual interests or pleasures (anhedonia); disturbances of sleep and eating; dysphoric mood (depressed, anxious, irritable, or angry mood); and difficulty concentrating or focusing attention.

Currently, discussion is ongoing within the field regarding the need for a third category of symptoms for diagnosis: disorganized symptoms. Disorganized symptoms include thought disorder, confusion, disorientation, and memory problems. While they are listed by DSM-IV as common in schizophrenia—especially during exacerbations of positive or negative symptoms, they do not yet constitute a formal new category of symptoms. Some researchers think that a new category is not warranted because disorganized symptoms may instead reflect an underlying dysfunction common to several psychotic disorders, rather than being unique to schizophrenia (Toomey et al., 1998).

**DSM-IV diagnostic criteria for schizophrenia**

A. **Characteristic symptoms:** Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated):
1. delusions
2. hallucinations
3. disorganized speech (e.g., frequent derailment or incoherence)
4. grossly disorganized or catatonic behavior
5. negative symptoms, i.e., affective flattening, alogia, or avolition

Only one Criterion A symptom is required if delusions are bizarre or hallucinations consist of a voice keeping up a running commentary on the person’s behavior or thoughts, or two or more voices conversing with each other.

B. Social/occupational dysfunction: For a significant portion of the time since the onset of the disturbance, one or more major areas of functioning such as work, interpersonal relations, or self-care are markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, failure to achieve expected level of interpersonal, academic, or occupational achievement).

C. Duration: Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by only negative symptoms or two or more symptoms listed in Criterion A present in an attenuated form (e.g., odd beliefs, unusual perceptual experiences).

D. Schizoaffective and mood disorder exclusion: Schizoaffective disorder and mood disorder with psychotic features have been ruled out because either (1) no major depressive, manic, or mixed episodes have occurred concurrently with the active-phase symptoms; or (2) if mood episodes have occurred during active-phase symptoms, their total duration has been brief relative to the duration of the active and residual periods.

E. Substance/general medical condition exclusion: The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Relationship to a pervasive developmental disorder: If there is a history of autistic disorder or another pervasive developmental disorder, the additional diagnosis of schizophrenia is made only if prominent delusions or hallucinations are also present for at least a month (or less if successfully treated).

Symptomology of Schizophrenia

Psychotic symptoms (such as hallucinations and delusions) usually emerge in men in their late teens and early twenties and in women in their mid-twenties to
early thirties. They seldom occur after age 45 and only rarely before puberty, although cases of schizophrenia in children as young as five have been reported. In adolescents, the first signs can include a change of friends, a drop in grades, sleep problems, and irritability. Since many normal adolescents exhibit these behaviors as well, a diagnosis can be difficult to make at this stage. In young people who go on to develop the disease, this is called the "prodromal" period.

Research has shown that schizophrenia affects men and women equally and occurs at similar rates in all ethnic groups around the world.

The symptoms of schizophrenia fall into three broad categories:

- **Positive symptoms** are unusual thoughts or perceptions that include hallucinations, delusions and thought disorder.
- **Negative symptoms** represent a loss or a decrease in the ability to initiate plans, speak, express emotion, or find pleasure in everyday life. These symptoms are harder to recognize as part of the disorder and can be mistaken for laziness or depression.
- **Cognitive symptoms** (or cognitive deficits) are problems with attention, certain types of memory, and the executive functions that allow us to plan and organize. Cognitive deficits can also be difficult to recognize as part of the disorder but are the most disabling in terms of leading a normal life.

### Positive symptoms

Positive symptoms are easy-to-spot behaviors not seen in healthy people and usually involve a loss of contact with reality. They include hallucinations, delusions, thought disorder, and disorders of movement. Positive symptoms can come and go. Sometimes they are severe and at other times hardly noticeable, depending on whether or not the individual is receiving treatment.

**Hallucinations.** A hallucination is something a person sees, hears, smells, or feels that no one else can see, hear, smell, or feel. "Voices" are the most common type of hallucination in schizophrenia. Many people with the disorder hear voices that may comment on their behavior, order them to do things, warn them of impending danger, or talk to each other (usually about the patient). They may hear these voices for a long time before family and friends notice that something is wrong. Other types of hallucinations include seeing people or objects that are not there, smelling odors that no one else detects (although this can also be a symptom of certain brain tumors), or feeling things like invisible fingers touching their bodies when no one is close by.

**Delusions.** Delusions are false personal beliefs that are not part of the person's culture and do not change, even when other people present proof that the beliefs are not true or logical. People with schizophrenia can have delusions that are quite bizarre, such as believing that neighbors can control their behavior with
magnetic waves, people on television are directing special messages to them, or radio stations are broadcasting their thoughts aloud to others. They may also have delusions of grandeur and think they are a famous historical figure. People with paranoid schizophrenia can believe that others are deliberately cheating, harassing, poisoning, spying upon, or plotting against them or the people they care about. These beliefs are called delusions of persecution.

Thought Disorder. People with schizophrenia often have unusual thought processes. One dramatic form is disorganized thinking where the person may have difficulty organizing his thoughts or connecting them logically. Speech may be garbled or hard to understand. Another form is "thought blocking" where the person stops abruptly in the middle of a thought. When asked sometimes the person says it felt as if the thought had been taken out of his head. Finally, the individual might make up unintelligible words, so-called "neologisms."

Disorders of Movement. People with schizophrenia can be clumsy and uncoordinated. They may also show involuntary movements and may show grimacing or unusual mannerisms. They may repeat certain motions over and over or, in extreme cases, may become catatonic. Catatonia is a state of immobility and unresponsiveness that was more common when treatment for schizophrenia was not available; fortunately, it is now rare.

Negative symptoms
The term "negative symptoms" refers to reductions in normal emotional and behavioral states. These include:

- flat affect (immobile facial expression, monotonous voice),
- lack of pleasure in everyday life,
- diminished ability to initiate and sustain planned activity, and
- speaking infrequently, even when forced to interact.

People with schizophrenia often neglect basic hygiene and need help with everyday living activities. Because it is not as obvious that negative symptoms are part of a psychiatric illness, people with schizophrenia are often perceived by others as lazy and not willing to better their lives.

Cognitive symptoms
Cognitive symptoms are subtle and are often detected only when neuropsychological tests are performed. They include:

- poor executive functioning (the ability to absorb and interpret information and make decisions based on that information),
- inability to sustain attention, and
- problems with working memory (the ability to keep recently learned information in mind and use it right away).
Cognitive impairments often interfere with the patient's ability to lead a normal life and earn a living, and can cause great emotional distress.

**Functional Impairment**

The criteria for a diagnosis of schizophrenia include functional impairment in addition to the constellation of symptoms outlined above. For formal diagnosis, a person must be experiencing significant dysfunction in one or more major areas of life activities such as interpersonal relations, work or education, family life, communication, or self-care. These problems result from the complex of symptoms and their sequelae, but have been linked more to negative than to positive symptoms (Ho et al., 1998). They have serious economic, social, and psychological effects: unemployment, disrupted education, limited social relationships, isolation, legal involvement, family stress, and substance abuse. Such sequelae form the most distressing aspects of the illness for many people and contribute to the increased risk of suicide among people diagnosed with schizophrenia.

**Cultural Variation**

On first consideration, symptoms like hallucinations, delusions, and bizarre behavior seem easily defined and clearly pathological. However, increased attention to cultural variation has made it very clear that what is considered delusional in one culture may be accepted as normal in another. For example, among members of some cultural groups, “visions” or “voices” of religious figures are part of normal religious experience. In many communities, “seeing” or being “visited” by a recently deceased person are not unusual among family members. Therefore, labeling an experience as pathological or a psychiatric symptom can be a subtle process for the clinician with a different cultural or ethnic background from the patient; indeed, cultural variations and nuances may occur within the diverse subpopulations of a single racial, ethnic, or cultural group. Often, however, clinicians' training, skills, and views tend to reflect their own social and cultural influences.

Clinicians can misinterpret and misdiagnose patients whose cognitive style, norms of emotional expression, and social behavior are from a different culture, unless clinicians become culturally competent. For example, clinicians may misinterpret a client’s deferential avoidance of direct eye contact as a sign of withdrawal or paranoia, or a normal emotional reserve as flattened affect if they are unaware of the norms of cultural groups other than their own. There is some empirical evidence that such misinterpretations happen widely. One finding is that African-American patients are more likely than white patients to be diagnosed with severe psychotic disorders in clinical settings (Strakowski et al., 1995). The overdiagnosis of psychotic disorders among African Americans is interpreted by some as evidence of clinician bias.
People with differing cultural backgrounds also may experience and exhibit true schizophrenia symptoms differently. Culture shapes the content and form of positive and negative symptoms. For example, people in non-Western countries report catatonic behavior among psychiatric patients much more commonly than in the United States. How culture, societal conditions, and diagnosing tendencies among clinicians in various countries interact to create these differences is being studied but is not yet well understood.

No description of symptoms can adequately convey a person’s experience of schizophrenia or other serious mental illness. Two individuals with very different internal experiences and outward presentations may be diagnosed with schizophrenia, if both meet the diagnostic criteria. Additionally, their symptoms and presentation may vary considerably over time. This considerable variation has led to the naming of several subtypes of schizophrenia, depending on what symptoms are most prominent. Currently these are seen as variations within a single disorder. Similarly, the diagnosis is often difficult because other mental disorders share some common features. Diagnosis depends on the details of how people behave and what they report during an evaluation, the diagnostician, and variations in the illness over time. Therefore, many people receive more than one diagnostic label over the course of their involvement with mental health services. Refining the definition of schizophrenia and other serious mental illnesses to account for these individual and cultural variations remains a challenge to researchers and clinicians.

Prevalence

Studies of schizophrenia’s prevalence in the general population vary depending on the way diagnostic criteria are applied and the population, setting, and method of study. In general, 1-year prevalence in adults ages 18 to 54 is estimated to be 1.3 percent. Onset generally occurs during young adulthood (mid-20s for men, late-20s for women), although earlier and later onset do occur. It may be abrupt or gradual, but most people experience some early signs, such as increasing social withdrawal, loss of interests, unusual behavior, or decreases in functioning prior to the beginning of active positive symptoms. These are often the first behaviors to worry family members and friends.

Prevalence of Comorbid Medical Illness

The mortality rate in persons with schizophrenia is significantly higher than that of the general population. While elevated suicide accounts for some of the excess mortality—and is a serious problem in its own right—comorbid somatic illnesses also contribute to excess mortality. Until recently, there was little information on the prevalence of comorbid medical illnesses in people with schizophrenia. A recent study was among the first to document systematically that people with schizophrenia are beset by vision and dental problems, as well as by high blood pressure, diabetes, and sexually transmitted diseases. Their self-reported lifetime rates of high blood pressure (34.1 percent), diabetes (14.9 percent), and sexually
transmitted diseases (10.0 percent) are higher than those for people of similar age in the general population (Dixon et al., 1999). The reasons for excess medical comorbidity are unclear, yet medical comorbidity is independently associated with lower perceived physical health status, more severe psychosis and depression, and greater likelihood of a history of a suicide attempt.

**Course and Recovery**

Overall, research indicates that schizophrenia's course over time varies considerably from person to person (Wiersma et al., 1998) and varies for any one person. The variability may emanate from the underlying heterogeneity of the disease process itself, as well as from biological and genetic vulnerability, neurocognitive impairments, sociocultural stressors, and personal and social factors that confer protection against stress and vulnerability. Most individuals experience periods of symptom exacerbation and remission, while others maintain a steady level of symptoms and disability which can range from moderate to severe (Wiersma et al., 1998).

Most people experience at least one, often more, relapse after their first actively psychotic episode. Often these are periods of more intense positive symptoms, yet the person continues to struggle with negative symptoms in between episodes. However, whether such exacerbations have the same degree of disabling and distressing effects each time depends greatly on the person's coping skills and support system. Over time, many people learn successful ways of managing even severe symptoms to moderate their disruptiveness to daily life. Therefore, earlier years with the illness are often more difficult than later ones. Additionally, gradual onset and delays in obtaining treatment seem to raise the risk of longer episodes of acute illness over time. Early treatment with antipsychotic medications has been found to predict better long-term outcomes for people experiencing their first psychotic episode, as compared with a variety of control groups, including those in more advanced stages (Wyatt & Henter, 1998).

The course of schizophrenia is also influenced by personal orientation and motivation, and by supports in the form of skill-building assistance and rehabilitation. These, in turn, are heavily influenced by regional, cultural, and socioeconomic factors in addition to individual factors.

Family factors also are related to the course of illness. Following hospitalization, patients who return home are more likely to relapse if their family is identified as critical, hostile, or emotionally overinvolved than if their family is not so identified (Bebbington & Kuipers, 1994). This is a controversial finding because it appears to blame family members. However, studies suggest an interaction between families and the patient (Goldstein, 1995b), suggesting that the negative emotions of some family members may be a reaction to, more than a cause of relapse in, the family member. Blaming either the family or the patient overlooks
important ways both parties interact and how such interactions are associated with the course of schizophrenia.

Despite the variability, some generalizations about the long-term course of schizophrenia are possible largely on the basis of longitudinal research. Approximately 10 percent of patients seem to remain severely ill over long periods of time (Gerbaldo et al., 1995). Most do not return to their prior state of mental function. Yet several long-term studies reveal that about one-half to two-thirds of people with schizophrenia significantly improve or recover, some completely (Harding et al., 1992). These studies were important because they began to dispel the traditional view, dating back to the 19th century, that schizophrenia had a uniformly downhill course.

In summary, schizophrenia does not follow a single pathway. Rather, like other mental and somatic disorders, course and recovery are determined by a constellation of biological, psychological, and sociocultural factors. That different degrees of recovery are attainable has offered hope to consumers and families.

Gender and Age at Onset

There appear to be gender differences in the course and prognosis of schizophrenia. Women are more likely than men to experience later onset, more pronounced mood symptoms, and better prognosis, although the prognosis difference recently has come under question.

Research (Hafner et al., 1998) suggests that some of the apparent gender differences in course and outcome occur because for some women schizophrenia does not develop until after menopause. This delay is thought to be related to the protective effects of estrogen, the levels of which diminish at menopause. According to this line of reasoning, men have no such delay because they lack the protective estrogen levels. Therefore, a higher proportion of men develop schizophrenia earlier.

Generally, early onset (younger than age 25 in most studies) is associated with more gradual development of symptoms, more prominent negative symptoms across the course, and more neuropsychological problems, regardless of gender. Early onset also usually involves more disruption of adult milestones, such as education, employment achievements, and long-term social relationships. People with later onset often have reached these milestones, cushioning them from disruptive sequelae and enabling better coping with symptoms. Therefore, early onset (more men than women) often yields a more difficult first several years, although not necessarily a worse long-term outcome.
Etiology of Schizophrenia

The cause of schizophrenia has not yet been determined, although research points to the interaction of genetic endowment and major environmental upheaval during development of the brain. This section first discusses genetic studies and then turns to the evidence for neurodevelopmental disruption. These lines of research are beginning to converge: neurodevelopmental disruption may be the result of genetic and/or environmental stressors early in development, leading to subtle alterations in the brain. Furthermore, environmental factors later in development can either exacerbate or ameliorate expression of genetic or neurodevelopmental defects. The onset and course of schizophrenia are most likely the result of an interaction between genetic and environmental influences.

Family, twin, and adoption studies support the role of genetic influences in schizophrenia (Portin & Alanen, 1997). Immediate biological relatives of people with schizophrenia have about 10 times greater risk than that of the general population. Given prevalence estimates, this translates into a 5 to 10 percent lifetime risk for first-degree relatives (including children and siblings) and suggests a substantial genetic component to schizophrenia (Cannon et al., 1998). What also bolsters a genetic role are findings that the identical twin of a person with schizophrenia is at greater risk than a sibling or fraternal twin, and that adoptive relatives do not share the increased risk of biological relatives. However, in about 40 percent of identical twins in which one is diagnosed with schizophrenia, the other never meets the diagnostic criteria. The discordance among identical twins clearly indicates that environmental factors likely also play a role.

Current research proposes that schizophrenia is caused by a genetic vulnerability coupled with environmental and psychosocial stressors, the so-called diathesis-stress model (Portin & Alanen, 1997). Family studies suggest that people have varying levels of inherited genetic vulnerability, from very low to very high, to schizophrenia. Whether or not the person develops schizophrenia is partly determined by this vulnerability. At the same time, the development of schizophrenia also depends on the amount and types of stresses the person experiences over time. An analogy can be drawn to diabetes by virtue of both genetic factors (e.g., family history) and behavioral factors (e.g., diet, exercise, stress) that interact to determine whether or not a given person develops diabetes.

Despite the evidence for genetic vulnerability to schizophrenia, scientists have not yet identified the genes responsible. The current consensus is that multiple genes are responsible (Straub et al., 1998).

Numerous brain abnormalities have been found in schizophrenia. For example, patients often have enlarged cranial ventricles, especially the third ventricle, and decreased cerebral size compared with control groups. There is also some
evidence that at least some people with schizophrenia have unusual cortical laterality, with dysfunction localizing to the left hemisphere (Braun et al., 1995). To explain laterality, some have proposed a prenatal injury or insult at the time of left hemisphere development, which normally lags behind that of the right hemisphere.

The anatomical abnormalities found in different parts of the brain tend to correlate with schizophrenia’s positive symptoms and negative symptoms. Positive symptoms are often linked to temporal lobe dysfunction. Disorganized speech has been associated with abnormalities in brain regions associated with speech regulation (McGuire et al., 1998). Negative and cognitive symptoms, especially those related to volition and planning, are commonly associated with prefrontal lobe dysfunction. This is perhaps related to unusual neuronal density and may be more prevalent among patients whose families have a history of schizophrenia than those whose do not. However, mapping patients’ symptoms with brain regions is complex and variable. Researchers believe that the dysfunctions are present in brain circuitry rather than in one or two localized areas of the brain (Wiser et al., 1998).

Excessive levels of the neurotransmitter dopamine have long been implicated in schizophrenia, although it is unclear whether the excess is a primary cause of schizophrenia or a result of a more fundamental dysfunction. More recent evidence implicates much greater complexity in the dysregulation of dopamine and other neurotransmitter systems (Olie & Bayle, 1997). Some of this research ties schizophrenia to certain variations in dopamine receptors (Serretti et al., 1998), while other research focuses on the serotonin system (Inayama et al., 1996). However, it must be emphasized that in many cases it is possible that perturbations in neurotransmitter systems may result from complications of schizophrenia, or its treatment, rather than from its causes.

The “stressors” investigated in schizophrenia research include a wide range of biological, environmental, psychological, and social factors. There is consistent evidence that prenatal stressors are associated with increased risk of the child developing schizophrenia in adulthood, although the mechanisms for these associations are unexplained. Some interesting preliminary research suggests risk factors include maternal prenatal poverty, poor nutrition (Susser et al., 1998), and depression (Jones et al., 1998). Other stressors are exposure to influenza outbreaks (Rantakallio et al., 1997), war zone exposure (van Os & Selten, 1998), and Rh-factor incompatibility (Hollister, 1996). Their variety suggests other stressors might also be risk factors, under the general rubric of “maternal stress.”

As a result of such stresses, newborns of low birth weight and short gestation have been linked to increased risk of later developing schizophrenia (Jones et al., 1998), as have delivery complications (Jones & Cannon, 1998) and other early developmental problems. Among children, especially infants, viral central nervous system infections may be associated with greater risk (Iwahashi et al.,
However, support for these hypotheses is inconsistent and incomplete. In fact, it is possible that prenatal and obstetric complications associated with schizophrenia could reflect already disrupted fetal development, rather than being causal themselves. More generally, across the life span, the chronic stresses of poverty and some facets of minority social status appear to alter the course of schizophrenia.

At the same time, researchers and clinicians are striving to integrate findings concerning both diathesis and stress into models of how schizophrenia develops. Not only does brain biology influence behavior and experience, but behavior and experience mold brain biology as well. One promising integrative model is the neurodevelopmental theory of schizophrenia developed by Weinberger and others. It posits that schizophrenia develops from “a subtle defect in cerebral development that disrupts late-maturing, highly evolved neocortical functions, and fully manifests itself years later in adult life”.

As promising as these theories are, the causes and mechanisms of schizophrenia remain unknown. Nonetheless, research has uncovered several of treatments for schizophrenia that are effective in reducing symptoms and functional impairments.

**Schizophrenia Treatment**

The treatment of schizophrenia has advanced considerably in recent years. A battery of treatments has become available to ameliorate symptoms, to improve quality of life, and to restore productive lives. Treatment and other service interventions often are linked to the clinical phases of schizophrenia: acute phase, stabilizing phase, stable (or maintenance) phase, and recovery phase.

Optimal treatment across all phases of treatment includes some form of pharmacotherapy with antipsychotic medication, usually combined with a variety of psychosocial interventions. Psychosocial interventions include supportive psychotherapy, and family psychoeducational interventions, as well as psychosocial and vocational rehabilitation. The treatment of individuals with schizophrenia who are high service users should be orchestrated by an interdisciplinary treatment team to ensure continuity of services. Others may benefit from less intensive forms of case management and various self-help and consumer-operated services, described later. It is also important to assist individuals with schizophrenia in meeting their many related needs, such as for supported housing, transportation, and general medical care.

**Pharmacotherapy**

Antipsychotic medications have been available since the mid-1950s. They effectively alleviate the positive symptoms of schizophrenia. While these drugs have greatly improved the lives of many patients, they do not cure schizophrenia.
Everyone responds differently to antipsychotic medication. Sometimes several different drugs must be tried before the right one is found. People with schizophrenia should work in partnership with their doctor to find the medications that control their symptoms best with the fewest side effects.

The older antipsychotic medications include chlorpromazine (Thorazine®), haloperidol (Haldol®), perphenazine (Etrafon, Trilafon®), and fluphenazine (Prolixin®). The older medications can cause extrapyramidal side effects, such as rigidity, persistent muscle spasms, tremors, and restlessness.

In the 1990s, new drugs, called atypical antipsychotics, were developed that rarely produced these side effects. The first of these new drugs was clozapine. Clozapine (Clozaril®) was introduced in 1990. It treats psychotic symptoms effectively even in people who do not respond to other medications, but can produce a serious problem called agranulocytosis, a loss of the white blood cells that fight infection. Therefore, patients who take clozapine must have their white blood cell counts monitored every week or two. The inconvenience and cost of both the blood tests and the medication itself has made treatment with clozapine difficult for many people, but it is the drug of choice for those whose symptoms do not respond to the other antipsychotic medications, old or new.

Some of the drugs that were developed after clozapine was introduced — such as risperidone (Risperdal®), olanzapine (Zyprexa®), quietiapine (Seroquel®), sertindole (Serdolect®), and ziprasidone (Geodon®) — are effective and don’t produce extrapyramidal symptoms or agranulocytosis; but they can cause weight gain, which increases the risk of diabetes and high cholesterol, together called metabolic syndrome.

People respond very individually to antipsychotic medications, although agitation and hallucinations usually improve within days and delusions in a few weeks. Many people see substantial improvement in both types of symptoms by the sixth week of treatment. No one can tell beforehand exactly how a medication will affect a particular individual, and sometimes several medications must be tried before the right one is found.

When people first start to take atypical antipsychotics, they may become drowsy; experience dizziness when they change positions; have blurred vision; or develop a rapid heartbeat, menstrual problems, a sensitivity to the sun, or skin rashes. Most of these symptoms will go away after the first days of treatment, but people who are taking atypical antipsychotics should not drive until they adjust to their new medication.

If people with schizophrenia become depressed, it may be necessary to add an antidepressant to their drug regimen.
**Length of Treatment.** Like diabetes or high blood pressure, schizophrenia is a chronic disorder that needs constant management. At the moment, it cannot be cured but the rate or recurrence of psychotic episodes can be decreased significantly by staying on medication. Although responses vary from person to person, most people with schizophrenia need to take some type of medication for the rest of their lives and use other approaches, such as supportive therapy or rehabilitation, as well.

Relapses occur most often when people with schizophrenia stop taking their antipsychotic medication because they feel better, or only take it occasionally because they forget or don't think taking it regularly is important. It is very important for people with schizophrenia to take their medication on a regular basis and for as long as their doctors recommend. If they do so, they will experience fewer psychotic symptoms.

No antipsychotic medication should be discontinued without talking to the doctor who prescribed it, and it should always be tapered off under a doctor's supervision rather than being stopped all at once.

There are a variety of reasons why people with schizophrenia do not adhere to treatment. If they don't believe they are ill, they may not think they need medication at all. If their thinking is too disorganized, they may not remember to take their medication every day. If they don't like the side effects of one medication, they may stop taking it without trying a different medication. Substance abuse can also interfere with treatment effectiveness.

There are many strategies that can be used to help people with schizophrenia take their drugs regularly. Some medications are available in long-acting, injectable forms that eliminate the need to take a pill every day. Medication calendars or pill boxes labeled with the days of the week can both help patients remember to take their medications and let caregivers know if medication has been taken. Electronic timers on clocks or watches can be programmed to beep when people need to take their pills, and pairing medication with routine daily events, like meals, can help patients adhere to dosing schedules.

**Medication Interactions.** Antipsychotic medications can produce unpleasant or dangerous side effects when taken with certain other drugs. For this reason, the doctor who prescribes the antipsychotics should be told about all medications (over-the-counter and prescription) and all vitamins, minerals, and herbal supplements the patient takes. Alcohol or other drug use should also be discussed.

**Psychosocial Treatments**
Numerous studies have found that psychosocial treatments can help patients who are already stabilized on antipsychotic medication deal with certain aspects of schizophrenia, such as difficulty with communication, motivation, self-care,
work, and establishing and maintaining relationships with others. Learning and using coping mechanisms to address these problems allows people with schizophrenia to attend school, work, and socialize. Patients who receive regular psychosocial treatment also adhere better to their medication schedule and have fewer relapses and hospitalizations. A positive relationship with a therapist or a case manager gives the patient a reliable source of information, sympathy, encouragement, and hope, all of which are essential for recovery. By explaining the nature and causes of schizophrenia and the need for medication, the therapist can also help patients acknowledge the reality of their disorder and adjust to the limitations it imposes.

**Illness Management Skills.** - People with schizophrenia can take an active role in managing their own illness. Once they learn basic facts about schizophrenia and the principles of schizophrenia treatment, they can make informed decisions about their care. If they are taught how to monitor the early warning signs of relapse and make a plan to respond to these signs, they can learn to prevent relapses. Patients can also be taught more effective coping skills to deal with persistent symptoms.

**Integrated Treatment for Co-occurring Substance Abuse** - Substance abuse is the most common co-occurring disorder in people with schizophrenia, but ordinary substance abuse treatment programs usually do not address this population's special needs. When schizophrenia treatment programs and drug treatment programs are integrated, better outcomes result.

**Rehabilitation** - Rehabilitation emphasizes social and vocational training to help people with schizophrenia function more effectively in the community. Because people with schizophrenia frequently become ill during the critical career-forming years of life (ages 18-35), and because the disease often interferes with normal cognitive functioning, most patients do not receive the training required for skilled work. Rehabilitation programs can include vocational counseling, job training, money management, learning to use public transportation, and practicing social and workplace communication skills.

**Family Education** - Patients with schizophrenia are often discharged from the hospital into the care of their families, so it is important that family members know as much as possible about the disease in order to prevent relapses. Family members should be able to use different kinds of treatment adherence programs and have an arsenal of coping strategies and problem-solving skills to manage their ill relative effectively. Knowing where to find outpatient and family services that support people with schizophrenia and their caregivers is also valuable.

**Cognitive Behavioral Therapy Cognitive** - behavioral therapy is useful for patients with symptoms that persist even when they take medication. The cognitive therapist teaches people with schizophrenia how to test the reality of their thoughts and perceptions, how to "not listen" to their voices, and how to
shake off the apathy that often immobilizes them. This treatment appears to be effective in reducing the severity of symptoms and decreasing the risk of relapse.

**Self-Help Groups** - Self-help groups for people with schizophrenia and their families are becoming increasingly common. Although professional therapists are not involved, the group members are a continuing source of mutual support and comfort for each other, which is also therapeutic. People in self-help groups know that others are facing the same problems they face and no longer feel isolated by their illness or the illness of their loved one. The networking that takes place in self-help groups can also generate social action. Families working together can advocate for research and more hospital and community treatment programs, and patients acting as a group may be able to draw public attention to the discriminations many people with mental illnesses still face in today's world.

**The U.S. Mental Health Service System**

Mental disorders and mental health problems are treated by a variety of caregivers who work in diverse, relatively independent, and loosely coordinated facilities and services—both public and private—that referred to, collectively, as the *de facto mental health service system*.

About 15 percent of all adults and 21 percent of U.S. children and adolescents use services in the de facto system each year. The system is usually described as having four major components or sectors:

- **Specialty mental health** sector consists of mental health professionals such as psychiatrists, psychologists, psychiatric nurses, and psychiatric social workers who are trained specifically to treat people with mental disorders. The great bulk of specialty treatment is now provided in outpatient settings such as private office-based practices or in private or public clinics. Most acute hospital care is now provided in special psychiatric units of general hospitals or beds scattered throughout general hospitals. Private psychiatric hospitals and residential treatment centers for children and adolescents provide additional intensive care in the private sector. Public sector facilities include state/county mental hospitals and multiservice mental health facilities, which often coordinate a wide range of outpatient, intensive case management, partial hospitalization, and inpatient services. Altogether, slightly less than 6 percent of the adult population and about 8 percent of children and adolescents (ages 9 to 17) use specialty mental health services in a year.

- **General medical/primary care** sector consists of health care professionals such as general internists, pediatricians, and nurse practitioners in office-based practice, clinics, acute medical/surgical hospitals, and nursing homes. More than 6 percent of the adult U.S.
population use the general medical sector for mental health care, with an average of about 4 visits per year—far lower than the average of 14 visits per year found in the specialty mental health sector. The general medical sector has long been identified as the initial point of contact for many adults with mental disorders; for some, these providers may be their only source of mental health services. However, only about 3 percent of children and adolescents contact general medical physicians for mental health services; the human services sector (see below) plays a much larger role in their care.

- **Human services** sector consists of social services, school-based counseling services, residential rehabilitation services, vocational rehabilitation, criminal justice/prison-based services, and religious professional counselors. In the early 1980s, about 3 percent of U.S. adults used mental health services from this sector. But by the early 1990s, the National Comorbidity Survey (NCS) revealed that 5 percent of adults used such services. For children, school mental health services are a major source of care (used by 16 percent), as are services in the child welfare and juvenile justice systems, which serve about 3 percent.

- **Voluntary support network** sector, which consists of self-help groups, such as 12-step programs and peer counselors, is a rapidly growing component of the mental and addictive disorder treatment system.

**The Public and Private Sectors**

The mental health service system is divided into public and private sectors. The term “public sector” refers both to services directly operated by government agencies (e.g., state and county mental hospitals) and to services financed with government resources (e.g., Medicaid, a Federal-state program for financing health care services for people who are poor and disabled, and Medicare, a Federal health insurance program primarily for older Americans and people who retire early due to disability). Publicly financed services may be provided by private organizations. The term “private sector” refers both to services directly operated by private agencies and to services financed with private resources (e.g., employer-provided insurance).

State and local government has been the major payer for public mental health services historically and remains so today. Since the mid-1960s, however, the role of the Federal government has increased. In addition to Medicare and Medicaid, the Federal government funds special programs for adults with serious mental illness and children with serious emotional disability. Although small in relation to state and local funding, these Federal programs provide additional resources.

These federally funded public sector programs buttress the traditional responsibility of state and local mental health systems and serve as the mental
health service “safety net” and “catastrophic insurer” for those citizens with the most severe problems and the fewest resources in the United States. The public sector serves particularly those individuals with no health insurance, those who have insurance but no mental health coverage, and those who exhaust limited mental health benefits in their health insurance.

Each sector of the mental health service system has different patterns and types of care and different patterns of funding. Within the specialty mental health sector, state- and county-funded mental health services have long served as a safety net for people unable to obtain or retain access to privately funded mental health services. The general medical sector receives a relatively greater proportion of Federal Medicaid funds, while the voluntary support network sector, staffed principally by people with mental illness and their families, is largely funded by private donations of time and money to emotionally supportive and educational groups. The relative quality of care in these various sectors is a matter of intense interest and discussion, although there is little definitive research to date.

Effective functioning of the mental health service system requires connections and coordination among many sectors (public–private, specialty–general health, health–social welfare, housing, criminal justice, and education). Without coordination, it can readily become organizationally fragmented, creating barriers to access. Adding to the system’s complexity is its dependence on many streams of funding, with their sometimes competing incentives.

**Self-Help Groups**

Self-help refers to groups led by peers to promote mutual support, education, and growth. Self-help is predicated on the belief that individuals who share the same health problem can help themselves and each other to cope with their condition. The self-help approach enjoys a long history, most notably with the formation of Alcoholics Anonymous in 1935 (IOM, 1990). Over time, the self-help approach has been brought to virtually every conceivable health condition.

Since the 1970s, many mental health consumer groups emphasized self-help as well as advocacy (Chamberlin, 1995), although to different degrees. Self-help for recovering mental patients initially emphasized no involvement with mental health professionals. Over time the numbers and types of self-help groups began to flourish and more moderate viewpoints became represented. Self-help groups assume three different postures toward health professionals: the separatist model, the supportive model that allows professionals to aid in auxiliary roles, and partnership models in which professionals act as leaders alongside patients (Chamberlin, 1978; Emerick, 1990). The focus of groups varies, with some groups united on the basis of diagnosis, such as Schizophrenics Anonymous and
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the National Depressive and Manic-Depressive Association, whereas others are more broad based.

Programs entirely run by consumers include drop-in centers, case management programs, outreach programs, businesses, employment and housing programs, and crisis services. Drop-in centers are places for consumers to obtain social support and assistance with problems.

Family Advocacy

The family movement has experienced spectacular growth and influence since its beginnings in the late 1970s. Although several advocacy and professional organizations speak to the needs of families, the family movement is principally represented by three large organizations. They are the National Alliance for the Mentally Ill (NAMI), the Federation of Families for Children's Mental Health (FFCMH), and the National Mental Health Association (NMHA). NAMI serves families of adults with chronic mental illness, whereas the Federation serves children and youth with emotional, behavioral, or mental disorders. NMHA serves a broad base of family members and other supporters of children and adults with mental disorders and mental health problems. Though the target populations are different, these organizations are similar in their devotion to advocacy, family support, research, and public awareness.

NAMI was created as a grassroots organization in 1979 by a small cadre of families in Madison, Wisconsin. Since then, its membership has skyrocketed to 208,000 in all 50 states (NAMI, 1999). NAMI's principal goal is to advocate for improved services for persons with severe and persistent mental illness—for example, schizophrenia and bipolar disorder. Its sole emphasis on the most severely affected consumers distinguishes it from most other consumer and family organizations. Another NAMI goal is to transform public attitudes and reduce stigma by emphasizing the biological basis of serious mental disorders, as opposed to poor parenting (NAMI, 1999). Correspondingly, NAMI advocates for intensification of research in the neurosciences. Through state and local affiliates, NAMI operates a network of family groups for self-help and education purposes.

NAMI's accomplishments are formidable. The organization has become a powerful voice for the expansion of community-based services to fulfill the vision of the community support reform movement. NAMI has successfully pressed for Federal legislation for family membership in state mental health planning boards. It is a prime force behind congressional legislation for parity in the financing of mental health services. It also has made substantial inroads in the training of mental health professionals to sensitize them to the predicament of the chronically mentally ill. It has promoted “psychoeducation,” specific information to
family members, usually in small-group settings, about schizophrenia and about strategies for dealing with relatives with schizophrenia (Lamb, 1994). Finally, NAMI has successfully lobbied for increased Federal research funding, and it has set up private research foundations (Lefley, 1996).

Patterns of Use

Adults

Americans use the mental health service system in complex ways, or patterns. A total of about 15 percent of the U.S. adult population use mental health services in any given year. (ECA, NCS) About 6 percent of the adult population use specialty mental health care; 5 percent of the population receive their mental health services from general medical and/or human services providers, and 3 to 4 percent of the population receive their mental health services from other human service professionals or self-help groups.

Also, slightly more than half of the 15 percent of the population that use mental health services have a specific mental or addictive disorder (8 percent), while the remaining portion has a mental health problem or a disorder not included in the ECA or NCS (7 percent). The surveys estimate that during a 1-year period, about one in five American adults—or 44 million people—have diagnosable mental disorders, according to reliable, established criteria. To be more specific, 19 percent of the adult U.S. population have a mental disorder alone (in 1 year); 3 percent have both mental and addictive disorders; and 6 percent have addictive disorders alone. Consequently, about 28 percent of the population have either a mental or addictive disorder (Regier et al., 1993; Kessler et al., 1994).

Given that 28 percent of the population have a diagnosable mental or substance abuse disorder and only 8 percent of adults both have a diagnosable disorder and use mental health services, one can conclude that less than one-third of adults with a diagnosable mental disorder receives treatment in one year. In short, a substantial majority of those with specific mental disorders do not receive treatment.

Among the service users with specific disorders, between 30 and 40 percent perceived some need for care. However, most of those with disorders who did not seek care believed their problems would go away by themselves or that they could handle them on their own (Kessler et al., 1997).
Barriers to the Receipt of Treatment

Most people with mental disorders do not seek treatment. This general statement applies to adults and older adults and to parents and guardians who make treatment decisions for children with mental disorders. There is a multiplicity of reasons why people fail to seek treatment for mental disorders but few detailed studies. The barriers to treatment fall under several umbrella categories: demographic factors, patient attitudes toward a service system that often neglects the special needs of racial and ethnic minorities, financial, and organizational.

Several demographic factors predispose people against seeking treatment. African Americans, Hispanics, and poor women are less inclined than non-Hispanic whites—particularly females—to seek treatment (Miranda & Green, 1999). Common patient attitudes that deter people from seeking treatment are not having the time, fear of being hospitalized, thinking that they could handle it alone, thinking that no one could help, and stigma (being too embarrassed to discuss the problem). Above all, the cost of treatment is the most prevalent deterrent to seeking care. Cost is a major determinant of seeking treatment even among people with health insurance because of inferior coverage of mental health as compared with health care in general. Finally, the organizational barriers include fragmentation of services and lack of availability of services. Members of racial and ethnic minority groups often perceive that services offered by the existing system do not or will not meet their needs, for example, by taking into account their cultural or linguistic practices.

Demographic, attitudinal, financial, and organizational barriers operate at various points and to various degrees. Seeking treatment is conceived of as a complex process that begins with an individual or parent recognizing that thinking, mood, or behaviors are unusual and severe enough to require treatment; interpreting symptoms as a "medical" or mental health problem; deciding whether or not to seek help and from whom; receiving care; and, lastly, evaluating whether continuation of treatment is warranted.

Trends and Advances in Mental Health

The past 25 years have been marked by several discrete, defining trends in the mental health field. These have included:

1. The extraordinary pace and productivity of scientific research on the brain and behavior;
2. The introduction of a range of effective treatments for most mental disorders;
3. A dramatic transformation of our society’s approaches to the organization and financing of mental health care; and
4. The emergence of powerful consumer and family movements.

Scientific Research. The brain has emerged as the central focus for studies of mental health and mental illness. New scientific disciplines, technologies, and insights have begun to weave a seamless picture of the way in which the brain mediates the influence of biological, psychological, and social factors on human thought, behavior, and emotion in health and in illness. Molecular and cellular biology and molecular genetics, which are complemented by sophisticated cognitive and behavioral science, are preeminent research disciplines in the contemporary neuroscience of mental health. These disciplines are affording unprecedented opportunities for “bottom-up” studies of the brain. This term refers to research that is examining the workings of the brain at the most fundamental levels. Studies focus, for example, on the complex neurochemical activity that occurs within individual nerve cells, or neurons, to process information; on the properties and roles of proteins that are expressed, or produced, by a person’s genes; and on the interaction of genes with diverse environmental influences. All of these activities now are understood, with increasing clarity, to underlie learning, memory, the experience of emotion, and, when these processes go awry, the occurrence of mental illness or a mental health problem.

Equally important to the mental health field is “top-down” research; here, as the term suggests, the aim is to understand the broader behavioral context of the brain’s cellular and molecular activity and to learn how individual neurons work together in well-delineated neural circuits to perform mental functions.

Effective Treatments. As information accumulates about the basic workings of the brain, it is the task of translational research to transfer new knowledge into clinically relevant questions and targets of research opportunity—to discover, for example, what specific properties of a neural circuit might make it receptive to safer, more effective medications. To elaborate on this example, theories derived from knowledge about basic brain mechanisms are being wedded more closely to brain imaging tools such as functional Magnetic Resonance Imaging (MRI) that can observe actual brain activity. Such a collaboration would permit investigators to monitor the specific protein molecules intended as the “targets” of a new medication to treat a mental illness or, indeed, to determine how to optimize the effect on the brain of the learning achieved through psychotherapy.

In its entirety, the new “integrative neuroscience” of mental health offers a way to circumvent the antiquated split between the mind and the body that historically has hampered mental health research. It also makes it possible to examine scientifically many of the important psychological and behavioral theories regarding normal development and mental illness that have been developed in years past. The unswerving goal of mental health research is to develop and refine clinical treatments as well as preventive interventions that are based on an
understanding of specific mechanisms that can contribute to or lead to illness but also can protect and enhance mental health.

Mental health clinical research encompasses studies that involve human participants, conducted, for example, to test the efficacy of a new treatment. A noteworthy feature of contemporary clinical research is the new emphasis being placed on studying the effectiveness of interventions in actual practice settings. Information obtained from such studies increasingly provides the foundation for services research concerned with the cost, cost-effectiveness, and deliverability of interventions and the design—including economic considerations—of service delivery systems.

Organization and Financing of Mental Health Care. Another of the defining trends has been the transformation of the mental illness treatment and mental health services landscapes, including increased reliance on primary health care and other human service providers. Today, the U.S. mental health system is multifaceted and complex, comprising the public and private sectors, general health and specialty mental health providers, and social services, housing, criminal justice, and educational agencies. These agencies do not always function in a coordinated manner. Its configuration reflects necessary responses to a broad array of factors including reform movements, financial incentives based on who pays for what kind of services, and advances in care and treatment technology. Although the hybrid system that exists today serves diverse functions well for many people, individuals with the most complex needs and the fewest financial resources often find the system fragmented and difficult to use. A challenge for the Nation in the near-term future is to speed the transfer of new evidence-based treatments and prevention interventions into diverse service delivery settings and systems, while ensuring greater coordination among these settings and systems.

Consumer and Family Movements. The emergence of vital consumer and family movements promises to shape the direction and complexion of mental health programs for many years to come. Although divergent in their historical origins and philosophy, organizations representing consumers and family members have promoted important, often overlapping goals and have invigorated the fields of research as well as treatment and service delivery design. Among the principal goals shared by much of the consumer movement are to overcome stigma and prevent discrimination in policies affecting persons with mental illness; to encourage self-help and a focus on recovery from mental illness; and to draw attention to the special needs associated with a particular disorder or disability, as well as by age or gender or by the racial and cultural identity of those who have mental illness.
References


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Post-Test

1. Approximately, how much does the U.S. spend annually on mental health services?
   A. 99 billion
   B. 69 billion
   C. 37 billion
   D. 18 billion

2. What is the most commonly occurring mental disorder?
   A. Anxiety disorders
   B. Mood disorders
   C. Schizophrenia
   D. None of the above

3. Which of the following is NOT one of the neurotransmitters altered during anxiety attacks?
   A. Serotonin
   B. Epinephrine
   C. Gamma-Aminobutyric Acid
   D. Cholecystokinin

4. Which of the following is rarely used to treat anxiety disorders?
   A. Benzodiazepines
   B. TCA Anti-depressants
   C. SSRI Anti-depressants
   D. Monoamine Oxidase Inhibitors

5. Grandiosity and dysphoria are symptoms commonly associated with
   A. major depressive disorder
   B. dysthymia
   C. bipolar disorder
   D. cyclothymia

6. Which of the following statements is TRUE?
   A. Anxiety and depression rarely coexist.
   B. Past parental neglect has been identified as a psychosocial factor associated with depression.
   C. Menopause has been linked to increased rates of depression in women.
   D. The risk among first degree relatives of people with bipolar disorder is about two to three times greater.
7. Which of the following statements concerning medications for mood disorders is False?
   A. A side effect of SSRIs is tremor.
   B. TCA overdose can result in fatal cardiac arrythmias.
   C. Bupropion may exacerbate symptoms of bipolar depression.
   D. Taking MAOIs with aged cheese may result in a lethal hypertensive reaction.

8. Which of the following is a negative symptom of schizophrenia?
   A. Seeing objects that aren’t there.
   B. Believing that people are plotting against you.
   C. Frequently speaking in neologisms.
   D. Having a flat affect

9. Which of the following statements is TRUE concerning schizophrenia?
   A. Enlarged cranial ventricles are frequently seen in individuals with schizophrenia.
   B. The genes responsible for schizophrenia are located on chromosomes 6, 14, and 17.
   C. Dopamine deficiency has been linked to schizophrenia.
   D. High birth weight increases the risk for schizophrenia.

10. What is the most prevalent deterrent for individuals seeking care for mental illness?
    A. Cultural stigma
    B. Cost of treatment
    C. Fear of hospitalization
    D. Distrust of the medical community