ETIOLOGY: PSYCHOLOGICAL FACTORS AND THEORIES

Mood disorders are psychological as well as physiological conditions. As with biological data, there is less disagreement about whether specific psychological dimensions of mood disorders exist than about whether they are etiological. And as is true of biological factors, proving that a particular psychological factor is causal would require prospectively following up people at risk for depression to determine whether those with the factor are more likely to develop a mood disorder. A finding that patients who already have had an affective episode and who have the factor in question are more likely to have a recurrence could simply imply that the factor is a residual symptom of the index episode and not an independent risk factor. Even if expensive and difficult prospective studies of psychological risk factors were conducted, a positive finding would not guarantee that any factors identified were not markers of an underlying biological factor.

Because none of the psychological theories of mania (e.g., that it is a defense against depression) has ever been tested empirically, we focus on psychological hypotheses of depression.

Abnormal Reactions to Loss

Loss is the life event that has been most reliably linked to depression. Sigmund Freud (1917[1915]/1957) pointed out that both grief and depression are reactions to loss, but depressive symptoms include guilt and low self-esteem. On the basis of psychoanalytic experience with depressed patients, Freud believed that grieving turned into depression when the bereaved felt ambivalent about the lost object (i.e., person) and could not tolerate the negative side of the ambivalence. An unconscious attack against an internalized image of the lost object that undermines self-esteem that depends in part on identification with the lost person is manifested as depression. Freud thought that early, unresolved losses made the patient more likely to have difficulty dealing with losses as an adult (Whybrow et al. 1984). Later theorists pointed out that loss of anything that represents a person and that is overvalued or ambivalently viewed—a group, a profession, a cherished belief, or an ideal, for example—can result in depression.

In most studies comparing depressed patients with nondepressed control subjects, childhood loss—especially loss of a parent—has had a positive association with adult depression, which has been temporally associated with a recent loss, separation, or disappointment (Bemporad 1988; Paykel 1982). In primate studies, separation from a mother or from a peer in animals raised with peers reliably results in behavioral depression and in the physiology of human depression; separation depression can be prevented or reversed by use of antidepressants (Kaufman and Rosenblum 1967; Suomi et al. 1978). Separation during infancy from the mother or, in the case of animals raised with peers, from the peer group also notably increases the risk of adult separation depression (Kaufman and Rosenblum 1967; McKinney 1988). Similarly, experience with human infants has shown that early separation can produce a depressive syndrome that predisposes to later depression (Bowlby 1980). Taken together, these kinds of findings suggest a role for loss in the etiology of depression, but the role may involve the physiology as much as the psychology of loss. In particular, disruption of an attachment bond in any primate leads first to distress, which, from an evolutionary standpoint, helps to attract back a parent from whom an infant has been separated. If reunion does not occur promptly, separation distress is replaced by withdrawal, which conserves energy and reduces the chance of attack by a predator (Dubovsky 1997). Early separations may sensitize arousal and withdrawal systems to react excessively to subsequent losses, whether real or symbolic.

Although the association between depression and loss seems reliable, it is not as strong as was originally thought. Not only does loss account for only a relatively small portion of the variance in the risk of
depression (Paykel 1982), but losses of one kind or another precede many other medical and psychiatric illnesses (MagPhyl and Thomas 1981). Loss, an event that is stressful in itself and that removes an important external source of regulation of disrupted psychology and physiology, may be a more severe instance of a range of stresses that predispose to mood disorders.

Other Psychodynamic Theories
Psychoanalyst Karl Abraham postulated that depression is a manifestation of aggression turned against the self in a patient who is unable to express anger against loved ones (Whybrow et al. 1984). Attacks on the introjected other, who psychologically has become a part of the self, undermine adaptive capacities and produce negative affect. In support of this hypothesis is the fact that many depressed patients have difficulty expressing anger openly, either because they lack self-confidence or because they are afraid of being abandoned by a loved one on whom they are excessively dependent. However, it seems unlikely that anger is converted directly into depression in such individuals because many depressed patients are openly irritable. A more likely explanation is that dependency, sensitivity to loss, and lack of assertiveness lead depressed people to conceal anger, or even differences of opinion with others, until it becomes overwhelming, at which point it intrudes into everyday interactions. This problem may be compounded by intensification of all emotional experience in depression.

A hypothesis first clearly articulated by Edward Bibring is that the central psychological fault in depression is loss of self-esteem (Whybrow et al. 1984). According to this hypothesis, the depression-prone person is an overambitious, conventional individual with unrealistically high ego ideals. Depression represents deflation of self-confidence and vitality within the self that results from failing to live up to internalized standards that are essential to the patient's self-concept. This concept was expanded in self-theory (Kohut 1971), which emphasizes the central role of the self as an organizer and driving force of all mental functions. Without coherence, mental activities are fragmented and ineffective. Without a sense of vitality, there is inadequate psychic fuel for optimism and useful engagement with challenges and stress.

It is traditionally held that the premorbid personality of the depressed patient is perfectionistic, involving high expectations of the self and others. However, this opinion is based primarily on retrospective recall by patients, which is likely to be influenced by patients' current states. Low self-esteem is a symptom of depression, but it has not yet been shown to be a cause. On the other hand, unrealistic expectations and perceptions of the self and others are also invoked in cognitive theories of depression, which use more objective measures and more formal studies of this variable.

Interpersonal Theory
Interpersonal theory emphasizes four basic interpersonal issues: unresolved grief, disputes between partners and family members about roles and responsibilities in the relationship, transitions to new roles such as parent and retired person, and deficits in the social skills that are necessary to sustain a relationship (Klerman et al. 1984). As in other psychodynamic theories, depressed mood and altered biology are hypothesized to be responses to loss or the threat of loss. A psychotherapy derived from interpersonal theory (i.e., IPT), which is described later in this chapter (see subsection "Interpersonal Therapy"), has been found to be effective as a primary treatment for depression and an adjunct in the treatment of bipolar disorder, although this does not prove that the etiological concept behind the psychotherapy is accurate.

Cognitive Theory
Cognitive theory, which is related to hypotheses derived from an earlier construct called rational emotive therapy, holds that negative thinking is a cause rather than a result of depression (A.T. Beck et al. 1979, 1985; Thase 1996; Whybrow et al. 1984). According to the cognitive model, early experience leads to the development of global negative assumptions called schemata. Depressive schemata involve all-or-nothing
assumptions such as

- If I'm not completely happy, I'll be totally miserable.
- If something isn't done exactly right, it's worthless.
- If I'm not perfect, I'm a failure.
- If everyone doesn't love me unconditionally, then no one loves me at all.
- If I'm not in complete control, I'm helpless.
- If I depend on anyone for anything, I'm totally needy.

As long as experience seems to support a schema—for example, if everything a person does seems to work out or if a person never leans on anyone else—mood remains unambivalently positive. However, if something happens to contradict an all-or-nothing assumption, the negative side of the patient's thinking predominates. Failure in one endeavor makes the patient feel like a complete failure, or becoming ill or otherwise requiring assistance results in the patient's thinking, "I'm totally needy" or "I can't do anything for myself." These negative beliefs, or negative cognitions, are supported by self-fulfilling prophecies that reinforce negative thinking. For instance, the patient who feels helpless as a result of not having been able to influence the outcome of a complex and difficult situation stops trying to do anything to deal with later, simpler stresses. When this lack of effort results in subsequent failures, the patient's belief that nothing can be done to influence the environment seems to have been proven. Systematic errors in thinking lead to catastrophic thinking and generalization of single negative events to global negative expectations of the self, the environment, and the future (the "cognitive triad").

Much of the evidence in favor of the cognitive theory of depression comes from demonstrations that psychotherapy based on the theory (i.e., cognitive therapy, discussed in the "Cognitive Therapy" section later in this chapter) is an effective treatment for major depression. However, cognitive therapy is effective even when patients do not express negative cognitions, and any psychotherapy or antidepressant can reverse depression whether or not negative thinking is formally addressed. In addition, all-or-nothing thinking is characteristic of several conditions (e.g., personality disorders) in addition to depression.

Learned Helplessness
A concept related to cognitive theory is learned helplessness, which was first clearly shown experimentally by psychologist Martin Seligman (Abramson et al. 1978; Seligman 1975). The classic learned helplessness paradigm involves exposing an animal to an inescapable noxious but harmless stimulus such as a mild electrical shock. At first, the animal attempts to flee from the shock, but when escape proves impossible, it lies down and accepts the shock passively. If the situation is changed so that the animal can escape the stimulus (e.g., if the investigator removes a barrier that was preventing the animal from leaving the portion of the cage where the shock is applied), the animal continues to act as though it cannot get away. The animal cannot be coaxed away from the shock; only forcibly dragging the animal to safety reverses the learned helpless behavior. A second instance of learned helplessness develops more readily than a first episode. Learned helplessness that develops in one situation may generalize to other situations.

Learned helplessness resembles the passive, withdrawn behavior of depression, and resistance to reversing a negative experience is reminiscent of the self-fulfilling negative expectations of depression. Learned helplessness can be demonstrated in humans—for example, by exposing normal subjects to an inescapable noxious sound—and subjects who score higher on depression rating scales develop learned helplessness more readily than do those without depressive symptoms (Abramson et al. 1978). In animals, pretreatment with an antidepressant prevents learned helplessness. Considering all these data, it has been postulated that previous experiences with uncontrollable situations create a predisposition to learned helplessness. In response to a new uncontrollable circumstance, more severe learned helplessness develops more rapidly than in the past, resulting in the behaviors and cognitions of depression.
Experimental evidence in favor of the learned helplessness theory of depression may not be as strong as it might seem. It is not clear that learned helplessness in animals is equivalent to human depression. Human subjects with elevated depression scores have not had actual depressive disorders and have not sought treatment for any reason. In addition, depression involves symptoms beyond those of learned helplessness.

**Behavioral Theories**

Behavioral theories of depression, which are related to learned helplessness, hold that depression is caused by loss of reinforcement for nondepressive behaviors, resulting in deficits in adaptive social behaviors such as being assertive, responding positively to challenge, and otherwise seeking important reinforcers such as affection, caretaking, and attention (Whybrow et al. 1984). At the same time that environmental rewards are no longer forthcoming with positive behavior (this is known as *noncontingent reinforcement*), helplessness, expressions of distress, physical complaints, and other depressive behaviors may be rewarded, especially if significant others pay more attention to disability than to competence. Loss, in addition to rupturing an important attachment bond, removes a major social reinforcer and results in depressive behaviors if the patient has not developed an adequate repertoire of adaptive behaviors and does not have other sources of reinforcement. Like negative cognitions, depressive behaviors would be expected to drive a depressed mood.

There is little question that interpersonal rewards influence behavior. If important people pay more attention to expressions of helplessness and inadequacy than to expressions of competence, it may be more rewarding to be depressed than to be healthy. However, it remains to be proven that behavioral factors by themselves can induce depression or that treatment of clinically important depression by behavioral techniques alone is effective.

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