

Hopelessness Depression: A Theory-Based Subtype of Depression

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We present a revision of the 1978 reformulated theory of helplessness and depression and call it the *hopelessness theory of depression*. Although the 1978 reformulation has generated a vast amount of empirical work on depression over the past 10 years and recently has been evaluated as a model of depression, we do not think that it presents a clearly articulated theory of depression. We build on the skeletal logic of the 1978 statement and (a) propose a hypothesized subtype of depression—*hopelessness depression*, (b) introduce hopelessness as a proximal sufficient cause of the symptoms of hopelessness depression, (c) deemphasize causal attributions because inferred negative consequences and inferred negative characteristics about the self are also postulated to contribute to the formation of hopelessness and, in turn, the symptoms of hopelessness depression, and (d) clarify the diathesis-stress and causal mediation components implied, but not explicitly articulated, in the 1978 statement. We report promising findings for the hopelessness theory and outline the aspects that still need to be tested.

In this article, we present a revision of the 1978 reformulated theory of helplessness and depression (Abramson, Seligman, & Teasdale, 1978) and call it the *hopelessness theory of depression*. Our motive for proposing the revision is that, although the 1978 reformulation has generated a vast amount of empirical work on depression over the past 10 years (see Sweeney, Anderson, & Bailey, 1986, for a meta-analysis of 104 studies) and recently has been evaluated as a model of depression (Barnett & Gotlib, 1988; Brewin, 1985; Coyne & Gotlib, 1983; Peterson & Seligman, 1984), the 1978 article did not explicitly present a clearly articulated theory of depression. Instead, it presented an attributional account of human helplessness and only briefly discussed its implications for depression. Perhaps it is no surprise, then, that much controversy currently exists about the status of the reformulated theory of depression. Some reviewers have argued that it has strong empirical support (Peterson & Seligman, 1984), others have contended that it has a weak empirical base (Barnett & Gotlib, 1988; Coyne & Gotlib, 1983), and still others have suggested it never has been tested adequately (Abramson, Alloy, & Metalsky, 1988, in press; Abramson, Metalsky, & Alloy, 1988; Alloy, Abramson, Metalsky, & Hartlage, 1988; Brewin, 1985).

In constructing the hopelessness theory, we have built on the

skeletal logic of the 1978 statement. We were influenced by criticisms of the 1978 statement and by results of work to test it, as well as—more generally—by recent developments in the field of depression. In essence, the hopelessness theory hypothesizes the existence in nature of an as-yet unidentified subtype of depression—*hopelessness depression*. We describe the hypothesized cause, symptoms, course, therapy, and prevention of hopelessness depression. We delineate differences between hopelessness depression and other proposed subtypes of depression. Also, we discuss its relation to nondepression. In addition to proposing the hopelessness depression subtype, we set forth other key aspects of the revision that distinguish it from the 1978 statement; we (a) introduce hopelessness as a proximal sufficient cause of the symptoms of hopelessness depression, (b) deemphasize causal attributions, because inferred negative consequences and inferred negative characteristics about the self also are postulated to contribute to the formation of hopelessness and, in turn, to the symptoms of hopelessness depression, and (c) clarify and elaborate upon the diathesis-stress and causal mediation components that are implied, but are not explicitly articulated, in the 1978 statement. The revision is a hopelessness, rather than an attributional, theory of depression and is more similar to other cognitive theories of depression than were its precursors (e.g., 1978 statement). We emphasize that in the revision, hopelessness is a hypothesized proximal sufficient *cause* and is not included as one of the symptoms of hopelessness depression.

Because the hopelessness theory is new, the evidence about its validity is not yet in. However, we have conducted a number of studies to test it. Also, many of the studies conducted to test the reformulated theory, as well as other clinical and empirical work on depression, are relevant to evaluating the theory, although few provide a direct test. We report this work and outline the aspects of the theory still in need of testing.

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Preliminary Concepts

In presenting the hopelessness theory, we find it essential to distinguish among the concepts of *necessary*, *sufficient*, and *contributory* causes of symptoms. A necessary cause of a set of symptoms is an etiological factor that must be present or have occurred in order for the symptoms to occur. The symptoms cannot occur if the etiological factor is absent or has not occurred. However, the symptoms are not required to occur when the necessary cause is present or has occurred (i.e., necessary but not sufficient). A sufficient cause of a set of symptoms is an etiological factor whose presence or occurrence guarantees the occurrence of the symptoms. An additional feature of a sufficient causal relation is that if the symptoms do not occur, then the etiological factor must not be present or must not have occurred. However, the symptoms may occur in the absence of the sufficient cause (i.e., sufficient but not necessary). A contributory cause of a set of symptoms is an etiological factor that increases the likelihood of the occurrence of the symptoms but is neither necessary nor sufficient for their occurrence.

In addition to varying in their formal relation to the occurrence of symptoms (*necessary*, *sufficient*, or *contributory*), causes also vary in their sequential relation to the occurrence of symptoms. In an etiological chain culminating in the occurrence of a set of symptoms, some causes operate toward the end of the chain, proximate to the occurrence of symptoms, whereas other causes operate toward the beginning of the chain, distant from the occurrence of symptoms. The former are *proximal* causes, and the latter are *distal* causes.¹

The Hopelessness Theory

Clinicians have long suggested that depression is not a single disorder but rather a group of disorders heterogeneous with respect to symptoms, cause, course, therapy, and prevention (e.g., Beck, 1967; Craighead, 1980; Depue & Monroe, 1978; Gillespie, 1929; Kendell, 1968; Kraepelin, 1913). As a complement to clinical and taxonomic approaches, the hopelessness theory represents a theory-based approach to the classification of a subset of the depressive disorders and postulates the existence in nature of hopelessness depression, an as-yet unidentified subtype of depression.

Cause

In contrast to symptom-based approaches to the classification of the depressive disorders (see Kendell, 1968), *cause* figures prominently in the definition of hopelessness depression. Few would disagree that, when possible, classification of psychopathologies by etiology, in addition to other factors, is more desirable than classification by symptoms alone, insofar as the former generally has more direct implications for cure and prevention than does the latter (McLemore & Benjamin, 1979; Skinner, 1981). Overall, the hopelessness theory specifies a chain of distal and proximal contributory causes hypothesized to culminate in a proximal sufficient cause of the symptoms of hopelessness depression.

A proximal sufficient cause of the symptoms of hopelessness depression: Hopelessness. According to the hopelessness the-

ory, a proximal sufficient cause of the symptoms of hopelessness depression is an expectation that highly desired outcomes will not occur or that highly aversive outcomes will occur coupled with an expectation that no response in one's repertoire will change the likelihood of occurrence of these outcomes.² The common-language term *hopelessness* captures the two core elements of this proximal sufficient cause: (a) negative expectations about the occurrence of highly valued outcomes (a negative outcome expectancy), and (b) expectations of helplessness about changing the likelihood of occurrence of these outcomes (a helplessness expectancy). Thus, whereas helplessness is a necessary component of hopelessness, it is not sufficient to produce hopelessness (i.e., hopelessness is a subset of helplessness; Garber, Miller, & Abramson, 1980). For brevity, we use the term *hopelessness* to refer to the proximal sufficient cause; helplessness, of course, is an expectation. Whereas the term *hopelessness* sometimes implies negative affect as well as negative outcome and helplessness expectations, we do not include negative affect as part of our definition of hopelessness. Finally, we use the phrase *generalized hopelessness* when people exhibit the negative-outcome/helplessness expectancy about many areas of life. In contrast, *circumscribed pessimism* occurs when people exhibit the negative-outcome/helplessness expectancy about only a limited domain. We suggest that cases of generalized hopelessness should produce severe symptoms of hopelessness depression, whereas circumscribed pessimism is likely to be associated with fewer or less severe symptoms, or both. However, cases in which a person exhibits circumscribed pessimism about extremely important outcomes also may be associated with severe symptoms.

It is useful to compare the hopelessness theory and Seligman's (1975) original helplessness theory with respect to proximal sufficient cause. Seligman's original statement is best characterized as a helplessness theory because it featured the expectation that one cannot control outcomes (regardless of their hedonic valence or likelihood of occurrence) as the proximal sufficient cause. The evolution from a helplessness to a hopelessness theory is consistent with Mandler's (1964, 1972) view that hopelessness, not helplessness, is a cause of the symptoms of depression.

One hypothesized causal pathway to the symptoms of hopelessness depression. How does a person become hopeless and, in turn, develop the symptoms of hopelessness depression? An important advantage of the hopelessness theory is that it not only specifies a proximal sufficient cause of a subtype of depres-

¹ For simplicity of exposition, we have presented the proximal-distal distinction in terms of a dichotomy: Proximal versus distal. Strictly speaking, however, it is more appropriate to think in terms of a proximal-distal continuum.

² Abramson, Seligman, and Teasdale (1978) cautioned that the problem of current concerns (Klinger, 1975) existed in their statement of the proximal sufficient cause of depression featured in the reformulation. We feel depressed about the nonoccurrence of highly desired outcomes that we believe we cannot obtain only when they are "on our mind," "in the realm of possibility," "troubling us now," and so on. Although Abramson et al. (1978) found Klinger's concept heuristic, they felt it was not sufficiently well defined to be incorporated into the reformulation. We emphasize that the problem of current concerns still remains to be solved.

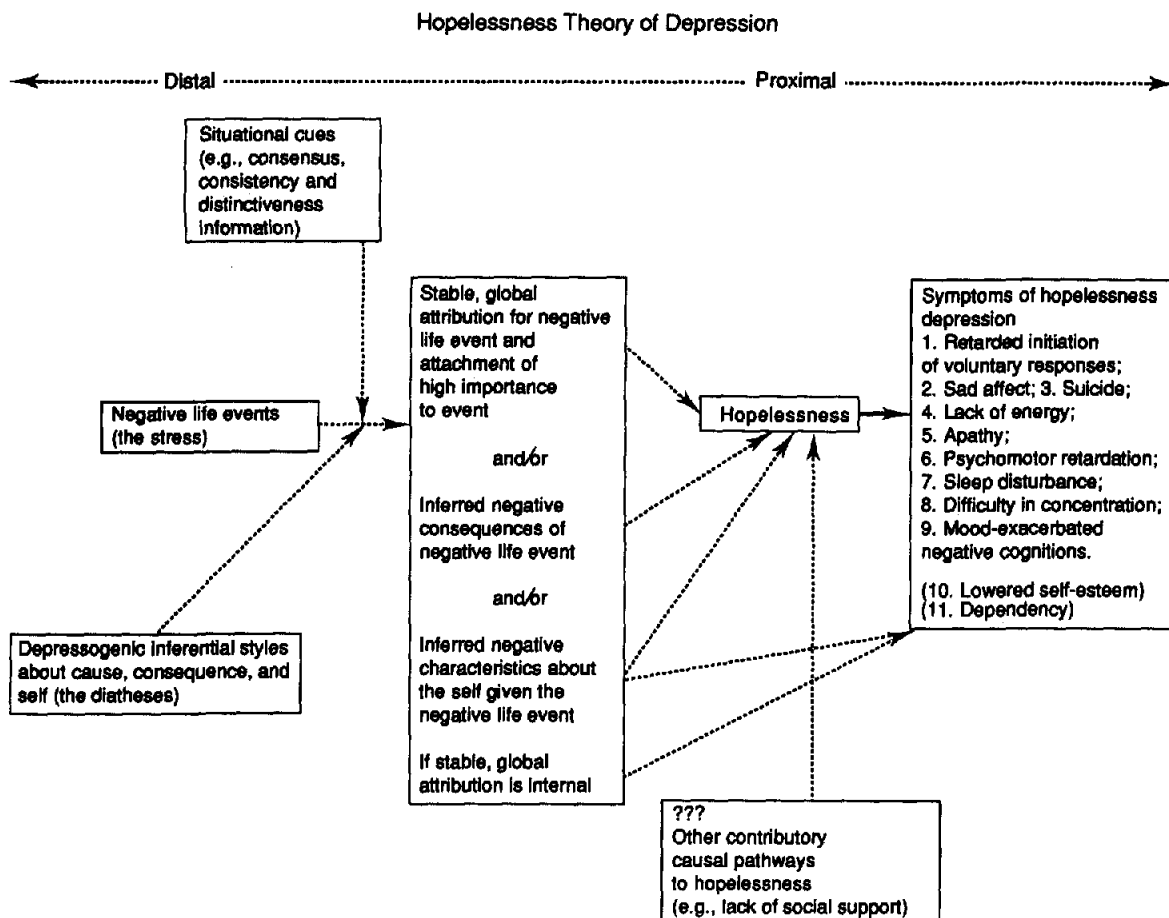


Figure 1. Causal chain specified in the hopelessness theory of depression. (Arrows with solid lines indicate sufficient causes. Arrows with broken lines indicate contributory causes.)

sion but also specifies a sequence of events in a causal chain hypothesized to culminate in this proximal sufficient cause. Each event in the chain leading to the proximal sufficient cause is a contributory cause because it increases the likelihood of, but is neither necessary nor sufficient for, the occurrence of the symptoms of hopelessness depression. In addition, these contributory causes vary in how proximal they are to the occurrence of the symptoms. Finally, with the exception of the relation between hopelessness and the symptoms of hopelessness depression, no link in the hypothesized causal chain is a sufficient condition for any other link. Instead, each link contributes to the occurrence or formation of the next link.

As can be seen in Figure 1, the hypothesized causal chain begins with the perceived occurrence of negative life events (or nonoccurrence of positive life events).³ In contrast to the 1978 reformulation (but consistent with later statements such as those of Peterson & Seligman, 1984), we begin the etiological chain with the perceived occurrence of a negative life event, rather than an uncontrollable event, because the logic of the hopelessness theory requires only the occurrence of the former, rather than the latter, to initiate the series of causes hypothesized to culminate in hopelessness and, in turn, the symptoms

of hopelessness depression (see also Rizley, 1978). Epidemiological research has shown that the occurrence of negative life events is involved in the development of depression (e.g., Brown & Harris, 1978; Lloyd, 1980a, 1980b). A fundamental question for the field of depression is why and how negative life events contribute to the onset of depression. In the hopelessness theory, negative events serve as "occasion setters" for people to become hopeless. However, people do not always become hopeless and depressed when confronted with negative life events. When do negative life events lead to depression and when do they not? According to the theory, there are at least three types of inferences people may make that modulate whether they become hopeless and, in turn, develop the symptoms of hopelessness depression in the face of negative life events: (a) inferences about why the event occurred (i.e., inferred cause or causal attribution), (b) inferences about consequences that will result from the occurrence of the event (i.e., inferred consequences), and

³ For the sake of brevity, we will use the phrase *negative life events* to refer to both the occurrence of negative life events and the nonoccurrence of positive life events.

(c) inferences about the self given that the event occurred (i.e., inferred characteristics about the self).

Proximal contributory causes: Inferred stable, global causes of particular negative life events and a high degree of importance attached to these events. The kinds of causal inferences people make for negative events and the degree of importance they attach to these events are important factors that contribute to whether they develop hopelessness and, in turn, the symptoms of hopelessness depression. In short, relatively generalized hopelessness and, in turn, the symptoms of hopelessness depression, are more likely to occur when negative life events are attributed to stable (i.e., enduring) and global (i.e., likely to affect many outcomes) causes and are viewed as important than when they are attributed to unstable, specific causes and are viewed as unimportant. For understanding hopelessness depression, we focus on stable, global, as opposed to stable, specific attributions for negative life events because only the former would be expected to contribute to relatively generalized hopelessness. The latter would be expected to contribute to relatively circumscribed pessimism. Whereas the attributional notion was featured in the 1978 statement, the importance concept was only briefly referred to in the 1978 statement and then more fully elaborated by Seligman, Abramson, Semmel, and von Baeyer (1979). Also, in contrast to the 1978 statement, we have de-emphasized the internality dimension of causal attributions and discuss its current role in the hopelessness theory in the section on symptoms of hopelessness depression.

If causal inferences for negative events do modulate the likelihood of becoming hopeless, then it is important to delineate what influences the kinds of causal inferences people make. During the past 20 years, social psychologists have conducted studies showing that people's causal attributions for events are, in part, a function of the situational information they confront (Kelley, 1967; McArthur, 1972). People tend to attribute an event to the factor or factors with which it covaries. According to this view, people would be predicted to make internal, stable, and global attributions for an event (e.g., failing a math exam) when they are confronted with situational information that suggests that the event is low in consensus (e.g., others do well on the math exam), high in consistency (e.g., typically failing exams in math), and low in distinctiveness (e.g., typically failing exams in other subjects as well as math; Kelley, 1967; Metalsky & Abramson, 1981). Thus, informational cues make some causal inferences for particular life events more plausible than others and some not plausible at all (see also Hammen & Mayol, 1982). Social psychologists have suggested a number of additional factors that also may guide the causal attribution process, including expectations for success and failure, motivation to protect or enhance one's self-esteem, focus of attention, salience of a potential causal factor, and self-presentational concerns, to name a few.

Proximal contributory causes: Inferred negative consequences of particular negative life events. Hammen and her colleagues (e.g., Gong-Guy & Hammen, 1980; Hammen & Cochran, 1981; Hammen & de Mayo, 1982) have argued that the inferred consequences of negative events, independently of causal inferences for these events, may modulate the likelihood that people will become depressed when confronted with a negative life event. For example, a student may attribute low scores

on the Graduate Record Examination (GRE) to distracting noises in the testing room (an unstable, specific attribution) but infer that a consequence of the poor performance on the GRE is that he or she never will be admitted to a graduate program in mathematics, the preferred career choice. We suggest that inferred negative consequences moderate the relation between negative life events and the symptoms of hopelessness depression by affecting the likelihood of becoming hopeless. If we follow the same logic as for causal attributions, inferred negative consequences should be particularly likely to lead to hopelessness when the negative consequence is viewed as important, not remediable, unlikely to change, and as affecting many areas of life. When the negative consequence is seen as affecting only a very limited sphere of life, relatively circumscribed pessimism rather than generalized hopelessness should result.

Proximal contributory causes: Inferred negative characteristics about the self given negative life events. In addition to inferred consequences of negative events, we suggest that inferred characteristics about the self, given these events, also may modulate the likelihood of formation of hopelessness and, in turn, the symptoms of hopelessness depression. Inferred characteristics about the self refer to the inferences a person draws about his or her own worth, abilities, personality, desirability, and so forth, from the fact that a particular negative life event occurred. Such a concept appears to be central in Beck's (1967) description of cognitive processes and depression. For example, Beck (1976, pp. 99–100) reported the case of a depressed, suicidal woman who previously had had a breach in her relationship with her lover, Raymond, and said, "I am worthless." When the therapist asked why she believed she was worthless, she replied, "If I don't have love, I am worthless." Again, if we follow the same logic as for causal attributions, inferred negative characteristics about the self should be particularly likely to lead to hopelessness when the person believes that the negative characteristic is not remediable or likely to change and that possession of it will preclude the attainment of important outcomes in many areas of life. When the negative characteristic is seen as precluding the attainment of outcomes in only a very limited sphere of life, relatively circumscribed pessimism, rather than generalized hopelessness, should result. Inferred characteristics about the self, given negative events, may not be independent of causal attributions for these events, but it is useful to conceptualize and operationally define them as distinct.

For the occurrence of a given negative life event, the three kinds of inferences (cause, consequence, and self-characteristics) may not be equally important in contributing to whether or not the person becomes hopeless and, in turn, develops the symptoms of hopelessness depression. For example, a young girl's inferences about the negative consequences of her mother's death, rather than about its cause or immediate implications for her view of herself, may be most important in contributing to whether or not she becomes hopeless. Perhaps events can be classified in terms of which of the three types of inferences will be most important in mediating whether the occurrence of the event leads to the development of hopelessness and, in turn, the symptoms of hopelessness depression.

Distal contributory causes: Cognitive styles. Complementing social psychologists' work on the situational determinants of causal attributions, Abramson et al. (1978) suggested a more

distal factor that may also influence the content of people's causal inferences for a particular event: individual differences in attributional style (see also Ickes & Layden, 1978). Some individuals may exhibit a general tendency to attribute negative events to stable, global factors and to view these events as very important, whereas other individuals may not. We use the phrase *hypothesized depressogenic attributional style* to refer to this tendency.

Individuals who exhibit the hypothesized depressogenic attributional style should be more likely than individuals who do not to attribute any particular negative event to a stable, global cause and view the event as very important, thereby incrementing the likelihood of becoming hopeless and, in turn, developing the symptoms of hopelessness depression. However, in the presence of positive life events or in the absence of negative life events, people who exhibit the hypothesized depressogenic attributional style should be no more likely to develop hopelessness, and therefore the symptoms of hopelessness depression, than people who do not exhibit this attributional style. This aspect of the theory is conceptualized usefully as a *diathesis-stress component* (Metalsky, Abramson, Seligman, Semmel, & Peterson, 1982). That is, the hypothesized depressogenic attributional style (the diathesis) is a distal contributory cause of the symptoms of hopelessness depression that operates in the presence, but not in the absence, of negative life events (the stress; see also Alloy, Kayne, Romer, & Crocker, 1988; Metalsky, Halberstadt, & Abramson, 1987).

The logic of the diathesis-stress component implies that a depressogenic attributional style in a particular content domain (e.g., for interpersonal-related events) provides "specific vulnerability" (cf. Beck, 1967) to the symptoms of hopelessness depression when an individual is confronted with negative life events in that same content domain (e.g., social rejection). This specific vulnerability hypothesis requires that there be a match between the content areas of an individual's depressogenic attributional style and the negative life events he or she encounters for the attributional diathesis-stress interaction to predict future symptoms of hopelessness depression (cf. Alloy, Clements, & Kolden, 1985; Alloy, Hartlage, & Abramson, 1988; Alloy, Kayne, et al., 1988; Anderson & Arnoult, 1985; Anderson, Horowitz, & French, 1983; Hammen, Marks, Mayol, & deMayo, 1985; Metalsky et al., 1987).

As with causal inferences, individual differences may exist in the general tendency to infer negative consequences and negative characteristics about the self, given the occurrence of negative life events. We do not know whether such cognitive styles are independent of the hypothesized depressogenic attributional style. We suggest that these two additional cognitive styles also are diatheses that operate in the presence, but not in the absence, of negative life events according to the specific vulnerability hypothesis. We will refer to these three negative styles as *cognitive diatheses*. Beck's concept of dysfunctional attitudes (Weissman, 1979) and Ellis's (1977) concept of irrational beliefs appear to overlap, in part, with these cognitive diatheses.

In discussing the diathesis-stress component, we have written as if cognitive styles and life events each are dichotomies. In fact, cognitive styles probably are better conceptualized as continua, with some people exhibiting more negative styles than others. Similarly, it may be more appropriate to speak of a con-

tinuum of negativity of life events. The continuum view suggests a *titration* model (cf. Zubin & Spring, 1977) of the diathesis-stress component. That is, the less negative a person's cognitive style, the more negative an event needs to be in order to interact with that style and contribute to the formation of symptoms. Thus, although many cases of hopelessness depression will occur among cognitively vulnerable people when they are confronted with negative events, people who do not exhibit the cognitive diatheses also may develop hopelessness depression when they are confronted with events sufficient to engender hopelessness in many or most people (e.g., a person who is put in a concentration camp and is repeatedly told by the guards that the only way to leave the camp is as a corpse). In a related vein, it is likely that although major negative life events often initiate the series of inferences hypothesized to culminate in the symptoms of hopelessness depression, they are not required to initiate the causal chain. The occurrence of more minor events, chronic stressors, or even daily hassles also may trigger the hypothesized depressogenic inferences among cognitively vulnerable people.

Our discussion underscores the importance of the causal mediation component of the hopelessness theory: Each causal factor depicted in Figure 1 contributes to the next causal factor in the proximal direction.

In addition to the cognitive factors previously described, interpersonal (e.g., lack of social support; Brown & Harris, 1978), developmental (e.g., death of mother during the child's early years; Brown & Harris, 1978), and even genetic factors may modulate the likelihood that a person will develop hopelessness and, in turn, the symptoms of hopelessness depression (see Tiger, 1979, for an intriguing discussion of genetic and biological factors in the development of hope and hopelessness). Moreover, future work needs to address the origins of cognitive diatheses (see Brown & Harris, 1978; Peterson & Seligman, 1984; Seligman et al., 1984). We eagerly await an elaboration of the theoretical statement of the causal pathway to the symptoms of hopelessness depression that includes cognitive, interpersonal, and other variables not included in our current statement (see Alloy & Koenig, 1988).

Symptoms

Hopelessness depression should be characterized by a number of symptoms (i.e., clinical manifestations or indicators). Two of these symptoms were described in the 1978 reformulation, and we retain them in the hopelessness theory: (a) retarded initiation of voluntary responses (motivational symptom), and (b) sad affect (emotional symptom). The logic by which hopelessness leads to these symptoms is straightforward. The motivational symptom derives from the helplessness expectancy component of hopelessness. If a person expects that nothing he or she does matters, why try? The incentive for emitting active instrumental responses decreases (Alloy, 1982; Bolles, 1972). Sadness derives from the negative outcome expectancy component of hopelessness and is a likely consequence of the expectation that the future is bleak. We no longer include the third symptom described in the 1978 reformulation, the cognitive symptom (associative deficit), because work on "depressive

realism" (e.g., Alloy & Abramson, 1979, 1988) has not supported it.

Hopelessness depression should be characterized by other symptoms as well (see Abramson, Alloy, et al., 1988, in press; Abramson, Metalsky, et al., 1988; Alloy & Koenig, 1988). Insofar as Beck and others have demonstrated that hopelessness is a key factor in serious suicide attempts and suicidal ideation, serious suicide attempts and suicidal ideation are likely symptoms of hopelessness depression (Beck, Kovacs, & Weissman, 1975; Kazdin, French, Unis, Esveldt-Dawson, & Sherick, 1983; Minkoff, Bergman, Beck, & Beck, 1973; Petrie & Chamberlain, 1983). If lack of energy, apathy, and psychomotor retardation are, in part, concomitants of a severe decrease in the motivation to initiate voluntary responses (see Beck, 1967), then they should be symptoms of hopelessness depression. We hypothesize that to the extent that people brood about the highly desired outcomes they feel hopeless to attain, sleep disturbance (e.g., initial insomnia) and difficulty in concentration will be important symptoms of hopelessness depression. The logic here is that people are likely to ruminate about their hopelessness because the outcomes involved are very important to them. Such rumination should interfere with falling asleep and make it difficult to focus attention on other aspects of life. On the basis of work showing that mood affects cognition (e.g., Bower, 1981), we predict that as individuals suffering from hopelessness depression become increasingly sad, their cognitions will become even more negative.

Although not necessarily symptoms of hopelessness depression, low self-esteem and/or dependency sometimes will accompany the other hypothesized symptoms. Lowered self-esteem will be a symptom of hopelessness depression when the event that triggered the episode was attributed to an internal, stable, global cause as opposed to any type of external cause or to an internal, unstable, specific cause. In contrast to the 1978 reformulation, then, the hopelessness theory postulates that attributing a negative life event to an internal cause does not, by itself, contribute to lowering self-esteem. Our revision requiring internal, stable, global attributions for lowered self-esteem is based on a number of studies (e.g., Crocker, Alloy, & Kayne, 1988b; Dweck & Licht, 1980; Janoff-Bulman, 1979) that show that internal attributions per se are not maladaptive and, in some cases, may be very adaptive (e.g., attributing failure to lack of effort leads to increased trying). The link between internal, stable, global attributions for negative life events and lowered self-esteem is based on social psychological work showing that people's self-esteem is influenced by their comparisons with others (e.g., Festinger, 1954; Morse & Gergen, 1970; Rosenberg, 1965; Schachter, 1959; Tesser & Campbell, 1983). If people make internal, stable, global attributions, then they expect that others could attain the outcomes about which they feel hopeless and therefore would feel inadequate compared with others. In addition, lowered self-esteem should occur in cases of hopelessness depression when people have inferred negative characteristics about themselves that they view as important to their general self-concept and not remediable or likely to change. Finally, dependency frequently may co-occur with lowered self-esteem because the conditions that give rise to lowered self-esteem will leave the person feeling inferior to others and thereby increase

the likelihood that he or she may become excessively dependent on them (Brewin & Furnham, 1987).

In general, circumscribed pessimism may not be associated with the full syndrome of the symptoms of hopelessness depression. Circumscribed pessimism is likely to produce fewer and/or less severe symptoms than generalized hopelessness, except when the person is pessimistic about an extremely important outcome. Whereas the motivational deficit should occur in cases of circumscribed pessimism, sadness may be less intense or even absent. Similarly, people with circumscribed pessimism should be less likely to commit suicide or exhibit the other hypothesized symptoms of hopelessness depression. Thus, circumscribed pessimism should lead to an identifiable behavioral syndrome, but this syndrome should be characterized primarily by a motivational deficit in the relevant domain.

Course

In considering the course of a disorder, the concepts of *maintenance*, *recovery*, *relapse*, and *recurrence* need to be distinguished (Klerman, 1978). Maintenance refers to the duration of a given episode of a disorder, and recovery refers to its remission. Relapse is a return of clinically significant symptoms within a relatively short period following remission, whereas recurrence is the onset of a new episode following a prolonged interval of remission.

Insofar as hopelessness is viewed as a proximal sufficient cause of the symptoms of hopelessness depression, the maintenance or duration of an episode of hopelessness depression should be influenced by how long this expectation is present. A prediction in the 1978 reformulation that we retain is that the more stable a person's attribution for a negative life event, the longer the person will be hopeless and, consequently, symptomatic. As a corollary, the maintenance of hopelessness not only should be influenced by the stability of the attribution for the event that triggered the given episode but also by the stability of attributions for newly occurring negative life events (see Brown & Harris, 1978; Lloyd, Zisook, Click, & Jaffe, 1981). Maintenance also may be influenced by the consequences the individual infers from the fact that he or she is depressed, as well as by the attribution he or she makes for the depression itself. Similarly, maintenance may be influenced by the characteristics the individual infers about himself or herself given that he or she is depressed. More generally, any factor that influences the duration of hopelessness should, in turn, influence the maintenance or chronicity of the symptoms of hopelessness depression (Abramson, Alloy, et al., 1988, in press; Abramson, Metalsky, et al., 1988; Alloy, Abramson, et al., 1988). These predictors of the duration of a given episode of hopelessness depression follow directly from the logic of the hopelessness theory. In addition, the possibility exists that once an individual becomes hopeless, some biological or psychological processes are triggered that need to run their course and do not dissipate as quickly as hopelessness. Such factors might maintain a hopelessness depression after hopelessness remits. Similarly, other factors such as lack of social support also may influence the duration of an episode of hopelessness depression after hopelessness remits.

Needles and Abramson (1988) proposed a model of recovery from hopelessness depression that highlights positive events.

They suggested that the occurrence of positive events provides the occasion for people suffering from hopelessness depression to become hopeful and, in turn, nondepressed. They suggested, analogous to the logic of the diathesis-stress component, that people with a style to attribute positive events to stable, global causes should be particularly likely to become hopeful and, in turn, nondepressed when confronted with a positive event. In addition, people with a style to infer positive characteristics about the self or positive consequences given positive events also should be likely to receive an emotional benefit when such events occur. Thus, positive events and inferences about them (cause, consequence, self-characteristics) may be particularly important in recovery from hopelessness depression.

Given the logic of the hopelessness theory, relapse or recurrence of hopelessness depression should be predicted by the re-appearance of hopelessness because, by definition, a relapse or recurrence is a new onset of hopelessness depression. Thus, the etiological chain hypothesized to culminate in the onset of the symptoms of hopelessness depression also applies directly to the relapse or recurrence of these symptoms. Hence, people with cognitive diatheses will be more likely to have relapses or recurrences of hopelessness depression when confronted with negative life events than people who do not exhibit these diatheses.

Therapy and Prevention

An important function of the hopelessness theory is to serve as an organizing rationale for the derivation of predictions about therapeutic interventions for hopelessness depression (Alloy, Clements, & Kolden, 1985; Beach, Abramson, & Levine, 1981; Halberstadt, Andrews, Metalsky, & Abramson, 1984). Because the hopelessness theory specifies an etiological chain, each link suggests a point for clinical intervention. A major advantage of using the proximal-distal continuum to order the events that cause hopelessness depression is that it not only suggests points of intervention for reversing current episodes but also suggests points for decreasing vulnerability to hopelessness depression.

Treating current episodes of hopelessness depression. Any therapeutic strategy that undermines hopelessness and restores hopefulness should be effective in remediating current symptoms of hopelessness depression (see also Hollon & Garber, 1980). Hopelessness could be attacked directly. Alternatively, the proximal causes (e.g., stable, global attributions for particular negative life events) that contribute to a person's current hopelessness could be attacked. Insofar as negative events and situational information supporting depressogenic inferences contribute to the maintenance of hopelessness, therapeutic interventions aimed at modifying the hopelessness-inducing environment should be helpful. Finally, if the person's own behavior is, to some degree, contributing to the depressogenic events and situational information he or she encounters, then personal behavior change would be an important therapeutic goal.

Preventing onset, relapse, and recurrence of hopelessness depression. According to the hopelessness theory, the three hypothesized cognitive diatheses put people at risk for initial onset, relapse, and recurrence of hopelessness depression. Therefore, modifying cognitive diatheses is an important goal for

prevention. Insofar as the cognitive diatheses require negative life events to exert their depressogenic effects, prevention efforts also might be directed toward lessening the stressfulness of events in the environments of cognitively vulnerable people. Finally, primary prevention efforts could be aimed at building nondepressive cognitive styles and environments.

We do not present the strategies and techniques for achieving these therapeutic goals because we have detailed them elsewhere (Alloy et al., 1985; Beach et al., 1981; Halberstadt et al., 1984). As with the other predictions about hopelessness depression, the clinical predictions can be corroborated or discredited only by empirical test.

Our therapeutic predictions generally are consistent with the theory and practice of cognitive therapy (Beck, Rush, Shaw, & Emery, 1979). However, the hopelessness theory suggests some interventions that depart from the emphases of cognitive therapy as currently conceptualized and practiced. For example, the hopelessness theory suggests a greater focus on environmental modifications than is currently practiced in cognitive therapy. Moreover, in contrast to a major theme in cognitive therapy, because the hopelessness theory does not postulate that depressogenic inferences necessarily are unrealistic or distorted (and allows for nondepressive cognitive distortions), the focus of therapy for hopelessness depressives would be on the content, rather than the realism, veridicality, or rationality of their inferences and beliefs (see also Hollon & Garber, 1988; Kayne & Alloy, 1988; Kruglanski & Jaffe, 1988).

Relation of Hopelessness Depression to Other Types of Depression and Psychopathology

One important descriptive psychiatric question is, Does the concept of hopelessness depression map onto any nosological category of affective disorders currently diagnosed (e.g., dysthymic disorder), or does this concept cut across the various nosological categories of affective or even nonaffective disorders currently diagnosed (cf. Halberstadt, Mukherji, Metalsky, & Abramson, 1988; Seligman, 1978)? Hopelessness depression most likely includes subsets of individuals from various currently diagnosed categories of depression (e.g., major depression, dysthymia) and may even include some depressed individuals who a priori would not be expected to be hopelessness depressive patients (e.g., some endogenous depressive subjects [see Eaves & Rush, 1984, and Hamilton & Abramson, 1983]; note, however, that just because some endogenous depressive subjects in these studies displayed a cognitive diathesis does not necessarily imply that they were suffering from hopelessness depression). Moreover, Alloy, Kelly, Mineka, and Clements (in press), on the basis of empirical and clinical studies of the comorbidity of anxiety and depression, suggested that many hopelessness depressive patients also may be suffering from anxiety. Finally, it is tempting to speculate that a subset of individuals who exhibit personality disorders (e.g., borderline personality) are characterized by extremely negative cognitive diatheses that make them particularly susceptible to hopelessness depression (Rose & Abramson, 1987; Silverman, Silverman, & Eardley, 1984).

A second descriptive psychiatric question is which diagnostic categories of depression, if any, involve fundamentally different etiological processes—and perhaps symptoms and therapy—

than those involved in hopelessness depression. Klein's (1974) concept of endogenomorphic depression (see also Costello's, 1972, concept of "reinforcer ineffectiveness depression") that maps closely onto the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition (DSM-III; American Psychiatric Association, 1980) category of major depressive episode, with melancholia, may be fundamentally distinct from the concept of hopelessness depression. The hypothesized core process in endogenomorphic depressions is impairment in the capacity to experience pleasure, rather than hopelessness.

A core question concerns the relation between the concept of hopelessness depression and general depression. We suggest that the relation of hopelessness depression to general depression is analogous to the relation between a subtype of mental retardation (e.g., Phenylketonuria, cretinism) and mental retardation in general. Just as some symptoms of a particular subtype of retardation may be a general feature of retardation (e.g., low IQ), particular hypothesized symptoms of hopelessness depression are considered symptoms of general depression (e.g., sadness). Other hypothesized symptoms of hopelessness depression (e.g., motivational deficit) may only partially overlap with the symptoms of general depression. Finally, still other symptoms of hopelessness depression (e.g., suicide and suicidal ideation) may not overlap at all with the symptoms of general depression. Thus, just as physicians do not define a particular subtype of retardation on the basis of symptoms alone because of potential overlap in some symptoms across subtypes, we do not define hopelessness depression on the basis of symptoms alone. Following the logic of workers in medicine more generally, we define hopelessness depression in terms of cause, symptoms, course, therapy, and prevention.

Nondepression

Ultimately, depression will be best understood in the larger context of a comprehensive theory of nondepression. The hopelessness theory offers some predictions about how people maintain a positive emotional state. According to the theory, the occurrence of a negative event provides a challenge to a positive emotional state. Making any of the three depressogenic inferences for negative events about cause, consequence, or self should increase the likelihood that hope will be lost and, as a result, the positive emotional state will break down. In contrast, refraining from making these inferences should allow hope to endure and, as a result, the challenge to be withstood and a positive state maintained.

The logic of the hopelessness theory also suggests that the occurrence of a positive event provides an opportunity to enhance one's emotional state. Making any of the following inferences when a positive event occurs should serve to facilitate a positive emotional state by restoring or increasing hope: (a) attributing the event to stable, global factors, (b) inferring positive consequences, and/or (c) inferring positive characteristics about the self.

The Hopelessness Theory Is Not Tautological

Some writers have asked whether precursors (e.g., the 1978 reformulation) of the hopelessness theory are tautological (e.g.,

Coyne & Gotlib, 1983). The hopelessness theory is not tautological because the predicted relation among each of its constructs do not follow of logical necessity. Moreover, all of the variables featured in the theory (e.g., depressogenic attributional style, hopelessness, reduced initiation of voluntary responses) can be identified and assessed independently of one another.

We suggest that the criticism of tautology has been leveled at precursors of the hopelessness theory because it has not been fully appreciated that these precursors (as well as the hopelessness theory itself) present a conceptual reorganization of the various phenomena typically associated with the concept of depression. In contrast to the traditional view, the hopelessness theory reorganizes the phenomena of depression into a hypothesized causal sequence, giving some features previously viewed as symptoms causal status (e.g., hopelessness) and maintaining symptom status for others (e.g., sadness). Because hopelessness is a hypothesized cause, its appearance must precede the appearance of the symptoms it is hypothesized to produce. However, at times, once present, hopelessness may persist and co-exist temporally with these symptoms. Such co-existence should not blur the distinction between the causal status of hopelessness and the symptom status of these resultant phenomena. Analogously, the AIDS virus, once present, may temporally co-exist with the symptoms it produces. Thus, to avoid tautology in testing the hopelessness theory, it is crucial that none of the hypothesized predictor variables (e.g., hopelessness, cognitive diatheses) be included in measures of the dependent variable(s) to be predicted (e.g., motivational symptom). The hopelessness theory is not unique in providing a conceptual reorganization of depressive phenomena. A number of theories of depression (e.g., Beck et al., 1979; Klein, 1974) organize these phenomena into (different) hypothesized causal sequences, giving some features previously viewed as symptoms of depression causal status instead.

Future Revisions of the Hopelessness Theory

We anticipate further expansions and revisions of the hopelessness theory. First, the theory will gain considerable power when it is able to specify the temporal intervals between links in the hypothesized etiological chain (e.g., the time lag between becoming hopeless and developing the symptoms of hopelessness depression; Cochran & Hammen, 1985; Metalsky et al., 1987). Second, how stable are cognitive diatheses? Do challenges (e.g., negative life events, negative moods) to an individual's cognitive system activate or prime these diatheses (cf. Alloy et al., 1985; Alloy, Abramson, et al., 1988; Riskind & Rholes, 1984)? Third, to what degree must the cognitions featured in the hopelessness theory be accessible (Higgins & King, 1980) to exert an influence? Fourth, the construct of *negative life events* in the theory is not fully elaborated. Do stressful episodic events, chronic stressors, and daily hassles all function as negative life events in the causal chain, and are some negative events more hopelessness-inducing than others? Also, individual differences may exist in what is perceived as a negative life event (Ahrens, 1987). Finally, are there feedback loops among the variables featured in the theory?

Comparison of the Hopelessness Theory to Other Theories of Depression

The hopelessness theory is more similar to other cognitive theories of depression, particularly the theories of Beck (1967, 1987) and Brown and Harris (1978), than were its precursors. Thus, we compare and contrast it with these two theories. Beck (1987) recently revised his theory. Although it is beyond the scope of this article to detail the revisions, we point to the major similarities and differences between his revision and the hopelessness theory. Important similarities include the following: (a) Both theories highlight the importance of maladaptive inferences in depression and give an important role to hopelessness, and (b) both theories have diathesis-stress components.

Key differences between the two theories are as follows: (a) Whereas the hopelessness theory postulates the existence of a particular subtype of depression—hopelessness depression—Beck has not elaborated a cognitively caused subtype of depression. Indeed, Beck's (1987) cross-sectional model emphasizes that negative cognitions are a necessary component of depression (see also Beck, 1984). (b) Whereas Beck (1987) emphasizes negative bias in depressive thinking, the hopelessness theory allows for the possibility of distortion (or accuracy) in both depressive and nondepressive cognition (cf. Alloy & Abramson, 1988; Dykman, Abramson, Alloy, & Hartlage, 1989; Taylor & Brown, 1988; Weinstein, 1980). (c) Whereas the hopelessness theory emphasizes the environment as well as cognitive processes in the etiology, maintenance, and treatment of depressive symptoms, Beck historically has focused more on cognitive processes (although a focus on the environment is not inconsistent with his theory). (d) Whereas Beck's theory describes the formal characteristics of depressive cognitions (e.g., automaticity, involuntariness, plausibility, and perseveration), the hopelessness theory does not. (e) Whereas the hopelessness theory specifies invulnerability factors for depressive symptoms (e.g., style to attribute negative life events to unstable, specific causes), Beck's theory does not.

Highlighting the social origins of depression, Brown and Harris's (1978) theory also bears important similarities to the hopelessness theory. (a) Most obviously, both theories feature hopelessness as a proximal cause of depressive symptoms. (b) Both theories emphasize people's appraisals of and inferences derived from negative life events rather than the mere occurrence of such events as a determinant of depressive reactions, similar to researchers of life stress and depression (as well as Beck, for that matter). (c) Both theories are diathesis-stress theories with specified causal mediation processes. (d) Both theories specify invulnerability factors for depressive symptoms.

Important differences between the two theories are the following: (a) Whereas the hopelessness theory postulates the existence of a particular subtype of depression—hopelessness depression—Brown and Harris (1978) have not elaborated a similar concept. (b) Whereas the hopelessness theory postulates that individuals not exhibiting cognitive diatheses may become depressed, Brown and Harris's theory focuses on the development of depression in depression-prone people (i.e., people exhibiting low self-esteem). (c) Whereas the hopelessness theory emphasizes negative cognitive styles as diatheses for depressive symptoms, Brown and Harris's theory emphasizes low self-

esteem as the diathesis; in fact, low self-esteem is a symptom of some cases of hopelessness depression in the hopelessness theory. (d) Whereas Brown and Harris's theory specifies distal social determinants of their featured diathesis (low self-esteem), the hopelessness theory is silent on this point.

Empirical Validity of the Hopelessness Theory

How would we know if hopelessness depression exists in nature and conforms to its theoretical description? At a conceptual level, the search for hopelessness depression is straightforward. To assert that hopelessness depression exists in nature is just to say that the hopelessness theory is true (cf. Clark, 1983). We search for hopelessness depression by testing the hopelessness theory.

Because the hopelessness theory is new, the evidence about its validity is not in. However, we have conducted a number of studies to test it. Also, many of the studies conducted to test the 1978 reformulation, as well as other clinical and empirical work on depression, are relevant to evaluating the hopelessness theory, although few provide a direct test. We now report this work.

Etiological Chain: Proximal Sufficient Cause Component

A key prediction of the hopelessness theory is that hopelessness temporally precedes and is a proximal sufficient cause of the symptoms of hopelessness depression. An alternative hypothesis is that hopelessness has no causal status and, instead, is simply another symptom of depression. Relevant to distinguishing between these two views, Rholes, Riskind, and Neville (1985) conducted a longitudinal study and reported that college students' levels of hopelessness at Time 1 predicted their levels of depression 5 weeks later at Time 2, over and above the predictive capacity of depression at Time 1. Similarly, in their prospective study, Carver and Gaines (1987) demonstrated that, after controlling statistically for earlier levels of depressive symptoms, dispositional pessimists were more likely to develop postpartum depression than were optimists. Although these results do not establish that hopelessness actually caused depressive symptoms at a later time, they do support the temporal precedence of hopelessness in predicting change in depressive symptoms (see also Riskind, Rholes, Brannon, & Burdick, 1987, for a demonstration that the interaction of attributional style and negative expectations predicts future depression).

In addition to the aforementioned longitudinal studies, a number of cross-sectional studies have examined the relation between hopelessness and depression. A notable feature of these studies is that they tested whether hopelessness is specific to depression or is a more general feature of psychopathology. Abramson, Garber, Edwards, and Seligman (1978) reported that hospitalized unipolar depressive patients were more hopeless than were both hospitalized nondepressed control and nondepressed schizophrenic subjects. It is interesting that the unipolar depressive subjects also were more hopeless than the depressed schizophrenic subjects. Hamilton and Abramson (1983) found that hospitalized episodic unipolar major depressive patients were more hopeless than a hospitalized nondepressed psychiatric group with mixed diagnoses (e.g., schizo-

phrenia, anxiety disorder, and personality disorders), as well as a nondepressed community control group. Recently, Beck, Riskind, Brown, and Steer (1988) found that psychiatric patients suffering from major depression were more hopeless than patients suffering from generalized anxiety disorder and a group of mixed psychiatric patients (diagnoses other than depression or anxiety). Taken together, these studies suggest that hopelessness is specific to depression and not a general feature of psychopathology.

Although the studies examining the association between hopelessness and depression are promising, they do not provide a wholly adequate test of the proximal sufficient cause component of the theory. As we have argued elsewhere (Abramson, Alloy, et al., 1988, in press; Abramson, Metalsky, et al., 1988; Halberstadt et al., 1984), insofar as hopelessness theory postulates a subtype of depression, it is inappropriate to simply lump together all depressive subjects and examine their levels of hopelessness to test the theory. Fortunately, some investigators have begun to examine the relation between hopelessness and the hypothesized individual symptoms of hopelessness depression and have reported a strong association between hopelessness and suicide attempts and ideation (Beck et al., 1975; Kazdin et al., 1983; Minkoff et al., 1973; Petrie & Chamberlain, 1983).

We note that many of the aforementioned investigators used the Hopelessness Scale (HS; Beck, Weissman, Lester, & Trexler, 1974) to operationally define the construct of hopelessness (e.g., Abramson, Garber, et al., 1978; Beck, Hollon, Young, Bedrosian, & Budenz, 1985). The HS is an adequate measure of hopelessness because it taps generalized hopelessness, as opposed to simply circumscribed pessimism. In addition, the HS provides an operational definition of hopelessness that is distinct from the symptoms of hopelessness depression. We look forward to the development of additional methods to assess hopelessness (e.g., interviews, behavioral methods).

Etiological Chain: Diathesis–Stress and Causal Mediation Components

Relevant to these components, a multitude of cross-sectional and longitudinal studies have examined the relation between attributional style and depression (see Barnett & Gotlib, 1988; Brewin, 1985; Coyne & Gotlib, 1983; Peterson & Seligman, 1984; Robins, 1988; and Sweeney et al., 1986, for reviews). Overall, these studies have shown that the tendency to make internal, stable, and global attributions for negative events is associated with severity of concurrent and future depressive symptoms in college student, patient, and other samples (see Sweeney et al., 1986, for a meta-analytic review). However, the corroborative findings have not always been strong.

We have argued elsewhere that this research strategy is inappropriate to test the diathesis–stress component (Abramson, Alloy, et al., 1988, in press; Abramson, Metalsky, et al., 1988; Alloy, Abramson, et al., 1988; Halberstadt et al., 1984). Recently, a number of studies have been conducted that do provide a more powerful test of the diathesis–stress component (and in some cases the causal mediation component) of the hopelessness theory. In a prospective field study, Metalsky et al. (1987) found that college students who showed a style to attribute negative achievement events to stable, global causes experienced a

more enduring depressive mood reaction to a low midterm grade than did students who did not exhibit this style. Consistent with the diathesis–stress component, attributional style for negative achievement events was not associated with students' mood reactions in the absence of the low grade. It is interesting that whereas students' more enduring depressive mood reactions were predicted by the interaction between attributional style and midterm grade (consistent with the diathesis–stress component), their immediate depressive mood reactions were predicted solely by the outcome on the exam (see also Follette & Jacobson, 1987). The results also provided support for the specific vulnerability hypothesis in that attributional style for negative achievement events, but not for negative interpersonal events, interacted with students' outcomes on the exam (an achievement event) to predict their enduring depressive mood reactions. Finally, consistent with the mediation component of the theory, the attributional styles of students who failed predicted their particular attributions for their midterm grades, which in turn, completely mediated the relation between attributional style and their enduring depressive mood responses.

With a design similar to that of Metalsky et al. (1987), Alloy, Kayne, et al. (1988) used causal modeling techniques to test the diathesis–stress and causal mediation components of the hopelessness theory and obtained support for both components. In addition, Alloy, Kayne, et al. reported that the interaction between attributional style and midterm grade predicted change in depressive symptoms as well as in transient depressive mood responses. In a longitudinal study, Nolen-Hoeksema, Girgus, and Seligman (1986) asked whether life events and attributional styles interacted to predict school children's future depression. They obtained partial support for the diathesis–stress component of the theory, with negative life events interacting with attributional style in some analyses but not in others. Finally, relevant to the causal mediation component, J. D. Brown and Siegel (1988) conducted a prospective study of stress and well-being in adolescence and reported that judgments of control over negative events interacted with attributions for them to predict future depression.

Two laboratory studies have examined the diathesis–stress component of the theory. Using a prospective design, Alloy, Peterson, Abramson, and Seligman (1984) found that students who typically attribute negative life events to global causes showed a wider generalization of learned helplessness to new situations when they were exposed to uncontrollable events than did individuals who typically attribute negative life events to more specific causes. Recently, Sacks and Bugental (1987) tested the diathesis–stress component in a laboratory study involving social failure or success (interaction with an unresponsive or responsive confederate). Supporting the diathesis–stress component, attributional style predicted short-term depressive reactions to the stressful social experience as well as the behaviors accompanying such a reaction.

Related to the diathesis–stress component, the theory's prediction that depression-prone students should be particularly likely to exhibit the hypothesized depressogenic attributional style was tested by Clements and Alloy (1988). Consistent with prediction, they found that depression-prone students had more negative attributional styles than did students who were not depression-prone, regardless of current depression level.

Another issue relevant to the diathesis–stress component that has been examined empirically concerns the relation between attributional style and self-esteem. Consistent with the hopelessness theory, and at odds with the 1978 reformulation, Crocker et al. (1988b) found that self-esteem was a function of all three attributional dimensions (internality, stability, and globality), as opposed to just internality. Subjects who made internal, stable, global, as opposed to simply internal, attributions for negative life events exhibited low self-esteem.

Insofar as dysfunctional attitudes overlap, in part, with the cognitive diatheses, studies that examine dysfunctional attitudes and negative life events in predicting depression are relevant to evaluating the diathesis–stress component. In this regard, Olinger, Kuiper, and Shaw (1987) administered the Dysfunctional Attitudes Scale (DAS; Weissman, 1979) and DAS-Contractual Contingencies Scale (DAS-CC; Olinger et al., 1987) to subjects. The DAS-CC was designed to measure the presence or absence of life events that impinge on a person's dysfunctional attitudes. Consistent with the diathesis–stress component, subjects who were cognitively vulnerable (high DAS) and experienced negative events that impinged on their vulnerability (high DAS-CC) were more depressed than were cognitively vulnerable subjects who did not experience the relevant negative life events (high DAS, low DAS-CC), as well as subjects who were not cognitively vulnerable (low DAS with either high or low DAS-CC scores). Similarly, Wise and Barnes (1986) reported that a normal sample of college students who were cognitively vulnerable (high DAS scores) and exposed to negative life events during the past year were more depressed than were students who also were cognitively vulnerable but were not exposed to a high rate of negative life events, as well as students who were not cognitively vulnerable regardless of life events. In a clinical sample, DAS scores and negative life events scores exerted main effects in predicting depression. A limitation of these two studies is that they used cross-sectional designs.

In relation to the mediation component, some investigators (e.g., Brewin, 1985) have questioned whether people's attributional styles predict their causal attributions for particular negative life events. As we previously indicated, in their tests of the causal mediation component of the theory, Metalsky et al. (1987) and Alloy, Kayne, et al. (1988) found that attributional styles did, in fact, predict particular causal attributions (see also Follette & Jacobson, 1987, for similar results). Moreover, support for the mediation component of the hopelessness theory challenges the alternative hypothesis that some antecedent or correlate of the cognitive diatheses is actually mediating depressive reactions.

A further aspect of the mediation component of the hopelessness theory involves whether people's attributions or attributional styles predict the formation of hopelessness. In a laboratory study, consistent with this component, Alloy and Ahrens (1987) demonstrated that a depressogenic attributional style contributed to depressive subjects' pessimism in predicting future events. More generally, Weiner's (1985) work has demonstrated that people's causal attributions affect their expectancies about future events.

The hopelessness theory predicts that attributions for life events should be predicted by situational information as well as attributional style. Consistent with this prediction, Haack,

Dykman, Metalsky, and Abramson (1988) found that both depressed and nondepressed students' causal attributions were influenced by consensus, consistency, and distinctiveness information. Similarly, Crocker, Alloy, and Kayne (1988a) found that people's perceptions of consensus information mediated their attributional styles (see also Alloy & Ahrens, 1987).

Course

Three studies have tested the course component directly. Consistent with prediction, Needles and Abramson (1988) reported that attributional style for positive outcomes interacted with positive life events to predict recovery from hopelessness. When positive events occurred in their lives, depressed students with a style to attribute positive events to stable, global causes showed a dramatic reduction in hopelessness relative to depressed students who did not exhibit this style. This change in hopelessness predicted a reduction of depressive symptoms. Students who did not experience an increase in positive events, regardless of style, also did not show such dramatic reduction of hopelessness.

In a 2-year follow-up, Evans et al. (1988) reported that patients treated cognitively (cognitive therapy alone or in combination with drugs) showed one half the rate of relapse of patients who were treated with drugs alone and then withdrawn from medication. Patients who were kept on medication also showed reduced relapse. Posttreatment attributional styles evidenced greater change in cognitively treated patients than in patients treated purely pharmacologically, and consistent with prediction, attributional style was the only cognitive variable that predicted subsequent relapse when residual depression was partialled out (the other two cognitive diatheses—consequences and self—were not assessed). Further analyses suggested that change in attributional styles mediated the relapse preventive effect of cognitive therapy.

Finally, in a follow-up of psychiatric patients, Rush, Weisenburger, and Eaves (1986) reported that the presence of dysfunctional attitudes (high DAS scores) at remission from depression predicted the presence of depression 6 months later. Although it was not statistically significant, a similar pattern was found for attributional style. A limitation of this study is the small sample size ($n = 15$).

Three additional studies indirectly tested predictions about course. As predicted, in a longitudinal study, Hamilton and Abramson (1983) found that among a sample of inpatient unipolar, episodic major depressive patients, dissipation of hopelessness was accompanied by remission of depressive symptoms. Unfortunately, their design did not allow a determination of whether dissipation of hopelessness preceded remission of symptoms, as required by the theory.

In line with the hopelessness theory, Paykel and Tanner (1976) reported that among recovered depressive subjects, those who relapsed experienced more undesirable events in the preceding 3-month period than did those who did not exhibit symptom return (see also Belsher & Costello, 1988). The inferences about cause, consequence, and self that these recovered depressive individuals made for those negative life events should provide even greater power for predicting relapse.

Lewinsohn, Steinmetz, Larson, and Franklin (1981), control-

ling for initial level of depression, found that unipolar depressed community volunteers who held negative expectations about the future and perceptions of low control (in our terminology, the two features of hopelessness) at Time 1 were less likely to recover over an 8-month period compared with unipolar depressives who did not exhibit these cognitions (see also Eaves & Rush, 1984).

Cure and Prevention

A number of studies (e.g., Beck et al., 1985; Shaw, 1977; Zeiss, Lewinsohn, & Munoz, 1979) have documented the efficacy of cognitive therapy for unipolar depression. The goals of cognitive therapy as currently practiced (cf. Beck et al., 1979) overlap with our previously stated goals for treatment and prevention of hopelessness depression. Therefore, empirical work demonstrating the efficacy of cognitive therapy for unipolar depression provides some support for the validity of the hopelessness theory's therapeutic predictions. Future work is needed to examine predictions about treatment of hopelessness depression in particular. In addition, predictions about the prevention of hopelessness depression need to be tested. Finally, the hopelessness theory's novel clinical predictions need to be tested.

Summary and Future Directions

On the basis of the aforementioned studies, the hopelessness theory appears promising. However, further research is needed. For example, although powerful tests of the attributional diathesis-stress component have been conducted, no one has examined the cognitive diatheses of inferring negative consequences or characteristics about the self or whether the cognitive style diathesis-stress interaction predicts clinically significant depression. Moreover, it is crucial to determine if this interaction predicts the development of the hypothesized symptoms of hopelessness depression. More generally, an important shortcoming of the prior work is that it has not focused on the symptoms of hopelessness depression in particular and, instead, simply has examined the symptoms of depression in general. Future investigators need to test more fine-grained predictions about the hypothesized symptoms of hopelessness depression. The issue of the stability of the cognitive diatheses has not been resolved satisfactorily. We have only begun, in a preliminary way, to investigate the issues of specific vulnerability and mediational processes. Finally, further tests of the predictions about course, cure, and prevention are needed. We eagerly await this research.

Difficult methodological issues may arise in the search for hopelessness depression, however. For example, the hopelessness theory is silent about the time lag between formation of hopelessness and onset of the symptoms of hopelessness depression. If it is very short, then a major challenge will be to develop methods with sufficient temporal resolving power to determine if hopelessness indeed precedes the occurrence of the hypothesized symptoms of hopelessness depression (see Alloy, Hartlage, et al., 1988, for proposed methods for testing the hopelessness theory). The results of work to test the hopelessness theory will determine if the concept of hopelessness depression needs to be revised. For example, perhaps the statement of the causal

pathway is correct but it culminates in a different set of symptoms than those currently hypothesized to compose hopelessness depression. In this case, the symptom—but not the cause—component of the hopelessness theory would need to be modified.

In discussing how to search for hopelessness depression, we note the possibility that future work may not corroborate the existence of hopelessness depression as a bona fide subtype with characteristic cause, symptoms, course, treatment, and prevention. Instead, the etiological chain featured in the hopelessness theory may be one of many pathways to a final common outcome of depression. In this case, it would be more compelling to speak of a hopelessness cause, as opposed to a hopelessness subtype, of depression.

Conclusion

In this article, we have focused on understanding depression. It is also important to understand nondepression, or normality, from the perspective of the hopelessness theory. In this regard, a passage from Solzhenitsyn's (1973) writings on the destructive labor camps in the Gulag Archipelago is provocative. In discussing corruption of prisoners in the camps, Solzhenitsyn says he is not going to explain the cases of corruption. Why, he says, should we worry about explaining the house that in subzero weather loses its warmth? What needs to be explained, he goes on to say, is that there are houses that retain their warmth even in subzero weather. Analogously, we suggest that perhaps what is to be explained by the depression researcher is not why certain people succumb to depression when confronted with the insults nature and our fellow humans deal to us all, but rather why many people maintain a nondepressed state in what sometimes is the psychological equivalent of subzero temperatures. The hopelessness theory attempts to explain not only how hope is lost in the face of adversity but also how it can endure.

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