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Although depression has long been recognized as an important form of psychopathology, experimental psychopathologists neglected this disorder until the 1970s. At that time, research on depression burgeoned within clinical psychology, and many investigators began to emphasize cognitive processes in the etiology, maintenance, and treatment of depression.

Two developments at the University of Pennsylvania in the late 1960s and early 1970s galvanized this cognitive approach to depression. Coming from a clinical perspective, Aaron Beck (1967), a psychiatrist, had abandoned the psychoanalytic account of depression and had begun to develop a cognitive theory that emphasized the negative "automatic thoughts" that seemed to trigger depressive symptoms among his patients. At the same time, Martin Seligman (1975), an experimental psychologist, had begun to formulate his theory of learned helplessness and depression, based on laboratory research about the deleterious effects of uncontrollable, aversive events. Similar to Beck, Seligman emphasized that maladaptive cognitions such as pervasive expectancies of no control over events could precipitate depressive symptoms. These two cognitive theories ignited a veritable explosion of research on depression.

Although it is difficult to reconstruct the forces that fueled cognitive approaches to depression, Dykman and Abramson (1990) listed a few whose impact appears likely. First, the rise of information-processing approaches in general (e.g., Neisser, 1967) and the study of social cognition in particular (e.g., Nisbett & Ross, 1980) provided a basic science foundation for a cognitive approach to depression. Second, basic researchers (e.g., Lazarus, 1966) demonstrated that cognitive processes mediate emotional reactions, with the specific con-

tents of thought (e.g., threat and danger) producing specific emotional reactions (e.g., anxiety). Third, many researchers had become disenchanted with the psychoanalytic approach to depression, in part because it seemed untestable. In contrast, the cognitive theories of Beck and Seligman were particularly appealing because of their scientific testability. Finally, many psychologists had begun to question the adequacy of purely behavioral accounts of various psychological phenomena (e.g., language acquisition; Chomsky, 1959).

In this chapter we present two cognitive theories of depression: the hopelessness theory (Abramson, Metalsky, & Alloy, 1989), a successor of Seligman's original helplessness theory of depression, and Beck's theory (Beck, 1967, 1987; Clark, Beck, & Alford, 1999). After evaluating the empirical status of these theories, we ask how well they can explain the "big facts" of depression. In addition, we elaborate the comparison of their respective conceptualization and assessment of cognitive vulnerability. We show that despite conceptual similarities, the cognitive vulnerability factors for depression hypothesized by the two theories are not identical. Then we use a self-regulatory perspective to elaborate the relation between cognitive vulnerability and rumination. Finally, in discussing future directions for research in this area, we embed the cognitive theories in a broader psychobiological context.

TWO COGNITIVE THEORIES OF DEPRESSION: HOPELESSNESS THEORY AND BECK'S THEORY

Overview of the Theories

Why are some people vulnerable to depression whereas others never seem to become depressed at all? According to the hopelessness theory and Beck's theory, the meaning or interpretation that people give to their experiences importantly influences whether they will become depressed and whether they will suffer repeated, severe, or long-duration episodes of depression. Indeed, the demonstrated efficacy of cognitive therapy for depression underscores the powerful clinical implications of a cognitive approach to depression.

Hopelessness Theory

According to the hopelessness theory (Abramson et al., 1989), the expectation that highly desired outcomes will not occur or that highly aversive outcomes will occur and that one cannot change this situation—hopelessness—is a proximal sufficient cause of depressive symptoms, specifically the hypothesized syndrome of "hopelessness depression" (HD). Symptoms of HD are hypothesized to include sadness, retarded initiation of voluntary responses, suicidality, low energy, apathy, psychomotor retardation, sleep disturbance, poor concentration, and mood-exacerbated negative cognitions.

How does a person become hopeless and, in turn, develop the symptoms of HD? As presented graphically in Figure 11.1, negative life events (or the nonoccurrence of desired positive life events) are "occasion setters" for people to become hopeless. However, the relation between negative life events and depression is imperfect; not all people become depressed when confronted with negative life events. According to hopelessness theory, three kinds of inferences that people may make when confronted with negative events contribute to the development of hopelessness and, in turn, depressive symptoms: causal attributions, inferred consequences, and inferred characteristics about the self. In brief, hopelessness and, in turn, depressive symptoms are likely to occur when negative life events are (1) attributed to stable (i.e., likely to persist over time) and global (i.e., likely to affect many areas of life) causes and viewed as important; (2) viewed as likely to lead

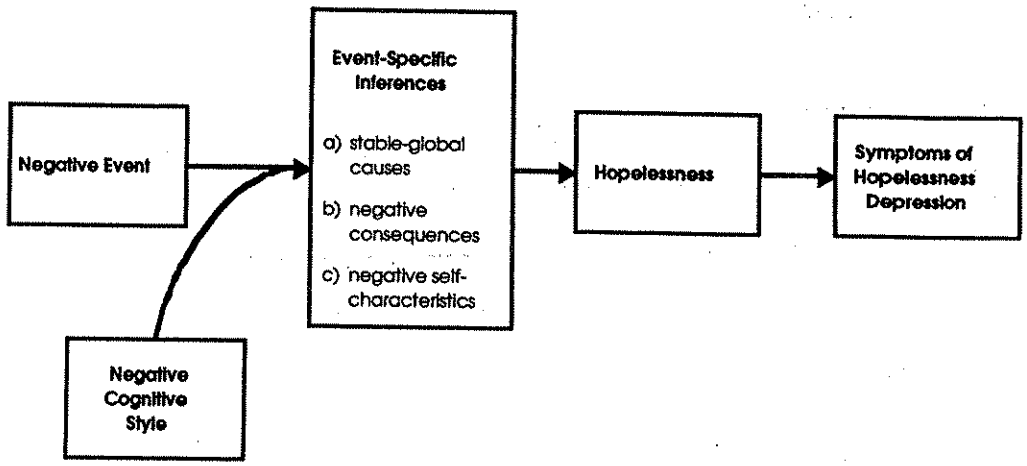

FIGURE 11.1. Causal chain in the hopelessness theory.

to other negative consequences; and (3) construed as implying that the person is unworthy or deficient. (When the causal attribution for a negative event is internal, stable, and global, hopelessness will be accompanied by lowered self-esteem and dependency as well as the other symptoms of HD.)

For example, suppose a student fails a test. According to the theory, the student should be likely to become depressed if she believes that the failure (1) was due to her low intelligence; (2) will prevent her from getting into medical school; and (3) means that she is worthless. In contrast, another student who fails the same test will be protected from becoming depressed if he believes that the failure (1) was due to not studying hard enough; (2) will motivate him to do especially well on the next test; and (3) has no implications for his self-worth.

In the hopelessness theory, informational cues in the situation (e.g., consensus, consistency, and distinctiveness information; Kelley, 1967), as well as individual differences in cognitive style, influence the content of people's inferences about cause, consequence, and self when negative life events occur. Individuals who exhibit a general style to attribute negative events to stable and global causes, to infer that current negative events will lead to further negative consequences, and to infer that the occurrence of negative events means that they are deficient or unworthy, should be more likely to make these depressogenic inferences about a given negative event than should individuals who do not exhibit this cognitive style. However, in the absence of negative life events, people exhibiting the depressogenic inferential style should be no more likely to develop hopelessness and, in turn, depressive symptoms than people not exhibiting this style. This aspect of the theory is a cognitive vulnerability–stress component: negative cognitive styles are the cognitive vulnerability and negative life events are the stress. A cognitive vulnerability in a particular content domain (e.g., for interpersonal events) provides "specific vulnerability" when a person is confronted with negative events in that same domain (e.g., social rejection).

Additional environmental factors also have been hypothesized to moderate the cognitive vulnerability–stress interaction. Much evidence indicates that social support buffers against depression when people experience stressful events (Barnett & Gotlib, 1988; Monroe & Hadjiyannakis, Chapter 13, this volume). Material, emotional, and informational support from others may buffer against depression by preventing the development of hopelessness. In particular, other people may provide "adaptive inferential feedback" that pro-

motes benign, rather than depressogenic, inferences about the causes, consequences, and meaning of negative events (Panzarella, Alloy, & Whitehouse, 2002).

Beck's Theory

Beck's cognitive theory of depression has been interpreted in several different ways (e.g., Abramson & Alloy, 1990; Abramson et al., 1989; Haaga, Dyck, & Ernst, 1991). However, in all accounts, the etiological hypotheses of Beck's (1967, 1987; Clark et al., 1999) theory are conceptually similar to those of the hopelessness theory. As Figure 11.2 shows, in Beck's theory, maladaptive self-schemata containing dysfunctional attitudes involving themes of loss, inadequacy, failure, and worthlessness constitute the cognitive vulnerability for depression. Such dysfunctional attitudes often involve the theme that one's happiness and worth depend on being perfect or on other people's approval. Examples of dysfunctional attitudes include "If I fail partly, it is as bad as being a complete failure" or "I am nothing if a person I love doesn't love me." When these hypothesized depressogenic self-schemata are activated by the occurrence of negative life events (the stress), they generate specific negative cognitions (automatic thoughts) that take the form of overly pessimistic views of oneself, one's world, and one's future (the *negative cognitive triad*) that, in turn, lead to sadness and the other symptoms of depression. In the absence of activation by negative events, however, the depressogenic self-schemata remain latent, less accessible to awareness, and do not directly lead to negative automatic thoughts or depressive mood and symptoms (Haaga et al., 1991). Beck (1987) has hypothesized that his cognitive vulnerability–stress model applies to only some forms of depression, particularly nonendogenous, unipolar depressions.

In Beck's (1983, 1987) theory, individual differences in the value people place on different kinds of experiences influence whether or not particular negative events will activate the cognitive vulnerability (depressogenic self-schemata) for depression. People who value social relationships, intimacy, and acceptance from others are high in "sociotropy" and are likely to become depressed when they experience social rejection or interpersonal losses. In contrast, people who are high in "autonomy" and value independence, freedom, and achievement will be more likely to become depressed when they experience failures or threats to their personal control.

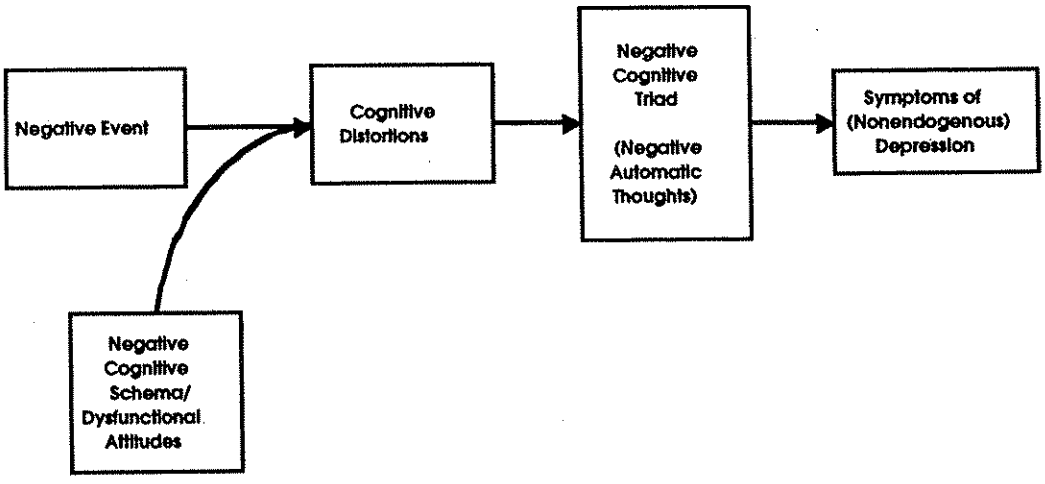

FIGURE 11.2. Causal chain in Beck's theory.

Comparison of the Hopelessness Theory and Beck Theory: Similarities and Differences

Although differing in some specifics, the hopelessness and Beck theories share many important features. At the most basic level, both theories emphasize the role of cognition in the origins and maintenance of depression. In addition, both theories contain a cognitive vulnerability hypothesis in which negative cognitive patterns increase people's vulnerability to depression when they experience negative life events in congruent or highly valued content domains. Moreover, both theories also propose a mediating sequence of negative inferences that influence whether or not negative events will lead to depressive symptoms. Finally, both theories recognize the heterogeneity of depression and either explicitly (hopelessness theory) or implicitly (Beck's theory) propose the existence of a cognitively mediated subtype of depression. This subtype may not map neatly on to any currently diagnosed category of depression. Instead, the hypothesized subtype may cut across currently diagnosed categories of clinical depression, or even some anxiety or personality disorders, and may be found in subsyndromal form in nonclinical populations as well. Of course, the possibility exists that this "subtype" may not be a bona fide subtype with characteristic cause, symptoms, course, treatment, and prevention. Instead, the etiological chains featured in the hopelessness and Beck theories may be two of many pathways to a final common outcome of depressive symptoms.

Despite their similarities, there is one striking difference between the hopelessness and Beck theories. To understand this difference, it is useful to distinguish between cognitive *processes* and cognitive *products* (Ingram, Miranda, & Segal, 1998). Cognitive processes involve the operations of the cognitive system such as informational encoding, retrieval, and attentional allocation. Cognitive products are the end result of the cognitive system's information-processing operations and consist of the cognitions and thoughts that the individual experiences. Inferences about cause, consequences, and self, as featured in the hopelessness theory, are examples of cognitive products. According to hopelessness theory, depressive and nondepressive cognition differ in content (e.g., stable, global vs. unstable, specific causal attributions for negative events) but not in process. In contrast, Beck's (1967) original theory emphasized that depressive and nondepressive cognition differs not only in content but also in process. Beck (1987) suggested that the inference process is "schema-driven" among depressives and "data-driven" among nondepressives. Thus, although both Abramson et al. (1989) and Beck (1967, 1987) emphasized that depressed people's inferences are negative, Beck further proposed that depressed people's inferences are unwarranted given current information. Specifically, Beck suggested that depressed individuals ignore positive situational information and are unduly influenced by current negative situational information in making their negative inferences. Nondepressed individuals, in contrast, appropriately utilize current information in making inferences. In short, Beck's (1967, 1987) original theory emphasized that depressive cognition is distorted whereas hopelessness theory is silent on the distortion issue.

Empirical Evaluation of the Theories

There are five central predictions from hopelessness theory and Beck's theory. First, both theories posit an interaction between cognitive vulnerability and stress that culminates in depressive symptoms. That is, cognitive vulnerability is hypothesized to moderate the effects of a negative life event on depression. When faced with a negative life event, individuals who exhibit cognitive vulnerability are hypothesized to be at higher risk for developing

depression than are individuals who do not exhibit cognitive vulnerability. Second, both theories include mediating links (e.g., hopelessness, negative cognitive triad) between the occurrence of a negative life event and the formation of depressive symptoms. For example, in the hopelessness theory, when cognitively vulnerable individuals encounter a negative life event, they are hypothesized to make depressogenic inferences about the event's cause, consequences, and implications for their self-concept. According to the hopelessness theory, these depressogenic inferences contribute to hopelessness, which in turn contributes to depression. Third, both theories posit causal chains that culminate in a particular subtype of depression (e.g., HD). Fourth, both theories hypothesize that a "match" in a particular domain (e.g., interpersonal or achievement) between the cognitive vulnerability and the negative event increases the likelihood of the development of depressive symptoms relative to a domain "mismatch." Finally, Beck's original theory emphasizes that depressive cognition is distorted; hopelessness theory is silent on this issue.

Research Designs for Testing the Etiological Hypotheses of the Cognitive Theories

What is the most powerful way to test the etiological hypotheses of the cognitive theories of depression? One research design that has figured prominently in testing the cognitive vulnerability hypotheses featured in Beck's theory and hopelessness theory is the *remitted* depression paradigm (see Ingram & Siegle, Chapter 4, this volume, and Just, Abramson, & Alloy, 2001, for reviews). In this design, the cognitive patterns of depressed individuals are examined during the depressed state as well as later, when the depression has remitted. The key assumption underlying the remitted design is that if negative cognitive patterns provide vulnerability for depression, they must be trait-like and persist beyond the remission of a current episode. According to this logic, any cognitive pattern not exhibited by previously depressed individuals cannot qualify as a vulnerability for depression. However, recent researchers have challenged the theoretical fidelity of remitted depression studies, as they are typically conducted, as potent tests of the cognitive theories (e.g., Just et al., 2001; Persons & Miranda, 1992). In particular, Just et al. (2001) argued that the conclusions based on the typical remitted depression studies are not justified because they are based on an erroneous assumption—namely, that cognitive vulnerability should be an immutable trait (see also Ingram et al., 1998). Making an analogy to the immune system and biological vulnerability, Just et al. (2001) showed that a factor need not be immutable to qualify as a vulnerability factor. If the remitted depression design is not adequate for testing the etiological hypotheses of the cognitive theories, what is?

A more powerful strategy for testing the cognitive vulnerability and other etiological hypotheses of the cognitive theories of depression is the behavioral ***high-risk design*** (e.g., Depue et al., 1981). As with the genetic high-risk paradigm, the behavioral high-risk design involves studying participants who do not currently have the disorder of interest but who are hypothesized to be at high or low risk for developing it. In contrast to the genetic high-risk paradigm, however, individuals in the behavioral high-risk study are selected on the basis of hypothesized psychological, rather than genetic, vulnerability or invulnerability to the disorder. Thus, to test the cognitive vulnerability hypothesis of depression, one would select currently nondepressed people who are at high versus low risk for depression based on the presence versus absence of the hypothesized depressogenic cognitive patterns. One would then compare these cognitively high- and low-risk groups on their likelihood of exhibiting depression both in the past (retrospective version of the design) and in the future (prospective version of the design). The prospective version of the design is superior to the retrospective version because the cognitive "vulnerability" that is assessed in the latter might actually

be a *scar* of a prior depressive episode (Rohde, Lewinsohn, & Seeley, 1991) rather than a *causal* factor in that prior episode (Gotlib & Abramson, 1999).

Etiological Hypotheses Featured in Hopelessness Theory

Many cross-sectional studies have established that cognitive vulnerability (typically attributional style and dysfunctional attitudes) is associated positively with depression in adults (Sweeney, Anderson, & Bailey, 1986; Haaga et al., 1991) and in children/adolescents (Gladstone & Kaslow, 1995; Joiner & Wagner, 1995). However, such cross-sectional studies do not provide strong tests of the cognitive vulnerability hypothesis because they cannot distinguish between the possibility that the cognitive vulnerability came first and contributed to the occurrence of depression as hypothesized in the cognitive theories and the alternative possibility that cognitive vulnerability does not contribute to depression and, instead, is a correlate or consequence of depression. Prospective behavioral high-risk studies are needed to establish temporal precedence. The prospective studies described below used a variant of a behavioral high-risk design (conceptualizing cognitive vulnerability as a continuous factor) to test the etiological hypotheses of the hopelessness theory for the development of depressive symptoms.

Recent prospective studies with children (Hilsman & Garber, 1995; Nolen-Hoeksema, Girgus, & Seligman, 1992; Robinson, Garber, & Hilsman, 1995), adolescents (Hankin, Abramson, & Siler, 2001), and adults (Alloy & Clements, 1998; Alloy, Just, & Panzarella, 1997; Hankin, Abramson, Miller, & Haeffel, 2002; Metalsky, Halberstadt, & Abramson, 1987; Metalsky & Joiner, 1992; Metalsky, Joiner, Hardin, & Abramson, 1993; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999) consistently have found that individuals who exhibit the hypothesized cognitive vulnerability featured in the hopelessness theory are more likely to develop depressive moods and/or depressive symptoms when they experience negative life events than are individuals who do not show this vulnerability. The few studies examining whether hopelessness mediates the relation between the cognitive vulnerability × stress interaction and increases in depression have provided partial support in adolescents (Hankin et al., 2001) and adults (Alloy & Clements, 1998; Metalsky & Joiner, 1992). Further work is needed to more fully test the hopelessness-as-mediator hypothesis of the hopelessness theory.

Work has only just begun to test the specific vulnerability or domain-match hypothesis of hopelessness theory. Supporting this hypothesis, in their academic midterm study Metalsky et al. (1987) reported that attributional vulnerability in the academic, but not the interpersonal, domain interacted with receipt of a low grade to predict sustained increases in depressed mood. Also consistent with the domain-match hypothesis, Joiner and Rudd (1995) found that negative attributional style for interpersonal events interacted with severe interpersonal disruptions to predict increases in suicidal ideation.

Hopelessness Depression Subtype Hypothesis

Preliminary evidence supports the HD subtype hypothesis. Five prospective studies have demonstrated that the cognitive vulnerability × stress interaction featured in hopelessness theory predicts changes in HD symptoms (Alloy & Clements, 1998; Alloy, Just, & Panzarella, 1997; Hankin et al., 2001, 2002; Metalsky & Joiner, 1997). Moreover, using structural equation modeling in clinical and nonclinical samples, Joiner et al. (2001) found support for the HD symptom cluster as a cohesive and distinct syndrome that stood out statistically even from closely related depressive symptoms.

Etiological Hypotheses and Nonendogenous Subtype Hypothesis Featured in Beck's Theory

Testing Beck's theory, recent prospective studies have found that the cognitive vulnerability × stress interaction (more specifically, dysfunctional attitudes × negative events) predicts increases in depressive symptoms over time (Brown, Hammen, Craske, & Wickens, 1995; Dykman & Johll, 1998; Hankin et al., 2002; Joiner, Metalsky, Lew, & Klocek, 1999; Klocek, Oliver, & Ross, 1997; Kwon & Oei, 1992). Regarding the mediational component, two prospective studies found that depression-specific negative thoughts mediated the association between the cognitive vulnerability × stress interaction and subsequent changes in depression (Joiner et al., 1999; Kwon & Oei, 1992). A number of studies have examined whether a domain match in Beck's theory (sociotropy/interpersonal and autonomy/achievement) is associated with depression. However, many of these studies have been cross-sectional and have produced inconsistent findings (see Coyne & Whiffen, 1995, for a critical review). Although a number of studies found that sociotropic individuals are particularly likely to become depressed following negative interpersonal events, some studies found that these individuals also become depressed following negative achievement events. Support is even less strong for the prediction that autonomous individuals are particularly likely to become depressed following negative achievement, but not interpersonal, events. The one study to date (Gibb et al., 2001) testing whether the cognitive vulnerability–stress and mediational components in Beck's theory lead to the hypothesized nonendogenous subtype found support for this formulation.

The Cognitive Vulnerability to Depression Project

The Cognitive Vulnerability to Depression (CVD) Project is a collaborative, two-site (Temple University and the University of Wisconsin) study that used both a retrospective and prospective behavioral high-risk design to test the cognitive vulnerability and other etiological hypotheses of hopelessness and Beck's theories of depression for both depressive symptoms and clinically significant depressive episodes. In the CVD Project, first-year college students at either high or low cognitive risk for depression who were nondepressed and had no other current Axis I psychopathology at the outset of the study were followed for 2.5 years with self-report and structured interview assessments every 6 weeks. The participants then were followed for an additional 3 years, with assessments occurring every 4 months.

Because the cognitively high-risk (HR) participants were required to score in the highest quartile on measures of the cognitive vulnerabilities featured in both the hopelessness theory (Cognitive Style Questionnaire [CSQ]; Abramson, Metalsky, & Alloy, 2000) and Beck's theory (expanded Dysfunctional Attitude Scale [DAS]; Weissman & Beck, 1978), and the cognitively low-risk (LR) participants were required to score in the lowest quartile on both of these measures, the CVD Project provides a broad test of a "generic" or "pooled" cognitive vulnerability hypothesis. A strength of this study is that the two sites permit a built-in assessment of generalizability of results.

The initial findings of the CVD Project provided direct and compelling support for the cognitive theories of depression. With their retrospective behavioral high-risk design, Alloy et al. (2000) found that, consistent with the cognitive vulnerability hypothesis, the HR group showed greater lifetime prevalence of DSM-III-R/RDC major depressive disorder, RDC minor depressive disorder, and HD than did the LR group (see also Alloy, Lipman, & Abramson, 1992, for similar findings). Moreover, the prospective results (Abramson et al., 1999; Alloy, Abramson, et al., 1999) also were consistent with the cognitive vulnerability hypothesis. Among participants with no prior history of depression, HR participants

showed a greater likelihood than LR participants of a *first onset* of DSM-III-R/RDC majc depressive disorder, RDC minor depressive disorder, and HD during the initial 2.5 yea prospective follow-up. These findings provide especially important support for the cognitiv vulnerability hypothesis because they are based on a truly prospective test, uncontaminate by prior history of depression.

What about those participants who, though nondepressed at the outset of the CVI Project, did have a prior history of clinically significant depression? This subsample allow a test of whether the cognitive vulnerability hypothesis holds for recurrences of depression which is particularly important given that depression often is recurrent (Belsher & Costello 1988; see Boland & Keller, Chapter 2, this volume, for a review of this literature). Consis tent with the cognitive vulnerability hypothesis, HR participants with a past history of clin ically significant depression were more likely than LR participants with previous depressior to develop recurrences of depressive episodes during the follow-up.

Finally, HR participants also were more likely than LR participants to exhibit suicidal ity, on a continuum from suicidal ideation to suicidal behavior, during the initial prospec tive follow-up (Abramson et al., 1998). Fortunately, no participants died by suicide.

These findings provide the first demonstration that the hypothesized cognitive vulnera bilities do indeed confer risk for both first onsets and recurrences of full-blown, clinically significant depressive disorders, the hypothesized subtype of HD, and suicidality. These results refute the criticism that the cognitive theories of depression apply only to mild depression. Moreover, the results also provide support for the hypothesis that the specific subtype of HD exists in nature and conforms to theoretical description. Complementing findings from the prospective portion of the CVD Project, Hankin et al. (2002) found that cognitive vulnerability (both negative cognitive styles from hopelessness theory and dysfunctional attitudes from Beck's theory) interacted with negative life events occurring over a 2-year prospective interval to predict onsets of clinically significant depressive episodes.

Recently, Lewinsohn, Joiner, and Rohde (2001) reported that both dysfunctional attitudes (at the level of a statistical trend) and attributional styles interacted with stress to predict major depressive disorder (MDD) among adolescents over a 1-year interval. Interestingly, the form of the interaction between cognitive vulnerability and stress differed for attributional styles and dysfunctional attitudes. Specifically, attributional style had its largest effect on depression under lower levels of stress, whereas it had little impact on depression under higher levels of stress. This result for attributional style is intriguing because it supports the *titration* model of vulnerability–stress relations featured in the original statement of the hopelessness theory (Abramson et al., 1989; Abramson, Alloy, & Hogan, 1997; see also Monroe & Simons, 1991). According to the titration model of the cognitive vulnerability–stress interaction, lower "doses" of stress are sufficient to trigger depression in cognitively vulnerable individuals, whereas higher doses of stress are required to precipitate depression in nonvulnerable individuals. At lower levels of stress, only cognitively vulnerable individuals may become depressed, whereas at very high levels of stress both cognitively vulnerable and nonvulnerable individuals alike may become depressed. In contrast to the titration model, it typically has been assumed that the vulnerability–stress model featured in both hopelessness theory and Beck's theory is a *synergistic* interaction in which only cognitively vulnerable individuals who experience a high level of stress develop depression. Lewinsohn et al.'s (2001) results for dysfunctional attitudes, as well as the results of the prior studies reviewed above, conform to this synergistic interaction model. However, Lewinsohn et al. (2001) acknowledged limitations in their assessment of attributional styles and stress that preclude definitive conclusions from their study about the relations of attributional style versus dysfunctional attitudes with stress in precipitating depression among adolescents. An exciting direction for future re-

search is to specify more precisely how cognitive vulnerability and stress combine to produce depression.

Summary of Empirical Tests of the Etiological Hypotheses in the Hopelessness and Beck Theories

Results of prior studies using methods less adequate than the behavioral high-risk design (e.g., remitted depression design) to test the etiological hypotheses of the cognitive theories of depression have been equivocal (see Barnett & Gotlib, 1988, and Just et al., 2001, for reviews). In contrast, the more recent studies reviewed above using or approximating a behavioral high-risk design have obtained considerable support for the etiological hypotheses of these theories. Indeed, we are especially excited by the findings from the CVD Project demonstrating that the hypothesized cognitive vulnerabilities confer risk for full-blown clinically significant depressive disorders. Although this newer work is very promising, future studies are needed to test more fully the role of cognition in the etiology of depression. Future work should target evaluations of the relations between cognitive vulnerability and stress in predicting clinically significant depression, the hypothesized mediating links, and the specific vulnerability hypothesis. Finally, it will be important to further explore whether HD actually is a bona fide subtype of depression.

Beck's Cognitive Distortion Hypothesis

In contrast to Beck's (1967, 1987) original characterization of "depressive cognitive distortion" and "nondepressive accuracy," research has demonstrated pervasive optimistic biases among nondepressed people and a "depressive realism effect" in which depressed individuals actually are more accurate than nondepressed individuals (see Alloy, Albright, Abramson, & Dykman, 1990, for a review). For example, nondepressed individuals often exhibit an "illusion of control" in which they believe that they control outcomes over which they objectively have no control, whereas depressed individuals seem less susceptible to this illusion (e.g., Alloy et al., 1990). Research on optimistic biases among "normal" people suggests that in formulating theories of depressive cognition, clinical researchers may have been wrong in assuming accuracy as the baseline of normal cognitive functioning. Consistent with Coyne and Gotlib's (1983) conceptual analysis, laboratory work has demonstrated that *both* depressed and nondepressed people show cognitive biases and illusions that are consistent with their preconceived beliefs or schemata (Dykman, Abramson, Alloy, & Hartlage, 1989; Gotlib, McLachlan, & Katz, 1988; McCabe & Gotlib, 1995). Thus, although work on depressive realism and nondepressive optimistic illusions has not established that depressed people always are more accurate than nondepressed people, these studies nevertheless have posed an important challenge to Beck's original portrayal of depressed people as either impervious to information in their environments or hopelessly biased by pervasive negative schemata and of nondepressed people as completely data-driven and free of the influence of biasing schemata (Gotlib & Abramson, 1999). In response to this work, modifications of Beck's theory have been proposed that emphasize differences in the content, rather than in the process, of depressive and nondepressive cognition (e.g., Dykman et al., 1989; Haack, Metalsky, Dykman, & Abramson, 1996; Haaga & Beck, 1995; Hollon & Garber, 1988).

How Well Can the Cognitive Theories Explain the "Big Facts" of Depression?

Descriptive research has produced some big, undisputed facts about depression. We consider seven of these facts and evaluate how well the cognitive theories can explain them.

First, depression is *recurrent* (Belsher & Costello, 1988; Boland & Keller, Chapter 2, this volume). One plausible explanation for this finding is that some individuals are especially depression-prone and likely to experience multiple episodes of depression. This possibility is consistent with the cognitive vulnerability hypothesis and the findings from the CVD Project that negative cognitive styles/dysfunctional attitudes provide vulnerability to both first onsets and recurrences of depression. Of interest and consistent with Teasdale's (1988) differential activation hypothesis, Lewinsohn, Allen, Seeley, and Gotlib (1999) reported that dysphoria, in combination with dysfunctional attitudes, was a stronger predictor of recurrence than of first onset of depression. These results, combined with those from the CVD Project, suggest that the processes underlying first onsets and recurrences are not completely identical, although cognitive vulnerability contributes importantly to both first onsets and recurrences.

Second, *life events* play a role in the development of depression (Brown & Harris, 1989; Monroe & Hadjiyannakis, Chapter 13, this volume). This fact is incorporated directly into the cognitive vulnerability–stress component of the cognitive theories (i.e., life events moderate the role of cognitive vulnerability).

Third, depression can be lethal as it clearly increases risk for *suicide* (e.g., Davila & Daley, 2000). Research has suggested that hopelessness is the key factor contributing to suicide among depressed individuals (e.g., Minkoff, Bergman, Beck, & Beck, 1973). Drawing on this work, Abramson et al. (1989) hypothesized that suicidality, on a continuum from suicidal ideation to completed suicide, is a core symptom of HD. Consistent with this hypothesis, Abramson et al. (1998) reported results from the CVD Project indicating that cognitively vulnerable individuals were more likely to show suicidality, mediated by hopelessness, over the prospective follow-up than were cognitively nonvulnerable individuals.

Fourth, depression is a *common* disorder (Kessler et al., 1994). This fact suggests that typical, as opposed to rare, factors cause depression. Negative life events, featured in the causal chains of the cognitive theories, are, unfortunately, all too common. Furthermore, as we described above, depressed individuals do not appear to exhibit rare or aberrant cognitive processes. Instead, depressed and nondepressed people likely rely on much the same cognitive processes but differ mainly in cognitive content (e.g., Dykman et al., 1989).

Fifth, the rates of depression *surge* during middle to late *adolescence* (Hankin et al., 1998). A rise in negative life events, the stressor in the cognitive vulnerability × stress interaction depicted in the cognitive theories, has been observed throughout adolescence, and this developmental trajectory parallels the increase in depression rates (Ge, Lorenz, Conger, Elder, & Simons, 1994). Furthermore, cognitive vulnerability appears to have developed and been consolidated by late childhood (Nolen-Hoeksema et al., 1992; Turner & Cole, 1994), and thus has become accessible for the cognitive vulnerability × stress interaction. Finally, the mediating link of hopelessness is within cognitive capability and available for participation in the causal chain culminating in depression by early adolescence (e.g., Kazdin, French, Unis, Esveldt-Dawson, & Sherick, 1983; Hankin et al., 2001). Thus, the cognitive theories can explain the surge in depression among adolescence through a rise in negative events that can interact with consolidated cognitive vulnerability which, in turn, can contribute to hopelessness and thereby eventuate in depression.

Sixth, *gender differences* in depression exist among adults, with twice as many women as men experiencing depression (Nolen-Hoeksema, Chapter 21, this volume). Consistent with this gender difference in depression, women show elevations on two of the factors featured in the causal chain of hopelessness theory: negative life events, especially in the interpersonal domain (see Hankin & Abramson, 2001, for a review) and negative cognitive styles (Angell et al., 1999; Hankin & Abramson, 2002). Holding everything else equal, then, women should be at greater risk than men for the depressogenic cognitive vulnerabili-

ty × stress interaction, and therefore for depression. Surprisingly, men actually exhibit *greater* dysfunctional attitudes, the cognitive vulnerability featured in Beck's theory, than do women (e.g., Angell et al., 1999; Gotlib, 1984; Haeffel et al., 2002). It is surprising that opposite patterns of gender differences are found on measures of cognitive vulnerability for the two cognitive theories. However, as we argue below, a growing body of empirical work indicates that the cognitive vulnerabilities defined in Beck's theory and in hopelessness theory, respectively, represent somewhat distinct constructs. Indeed, negative cognitive styles, as defined in hopelessness theory, may be more generally maladaptive whereas some "dysfunctional" attitudes such as perfectionism may be adaptive when paired with other characteristics such as high ability and/or high self-efficacy but maladaptive when paired with low ability and/or low self-efficacy. Perhaps males show elevations on the more "adaptive" dysfunctional attitudes. Future work is necessary to determine why men's elevations on dysfunctional attitudes do not appear to put them at heightened risk for depression.

Finally, depression long has been viewed as *heterogeneous* with multiple *causes*. The cognitive theories recognize this heterogeneity by postulating hypothesized depressive subtypes.

In showing that the cognitive theories can explain, or at least are consistent with, these big facts about depression, we don't mean to imply that other theories cannot also explain these facts. We simply are suggesting that the cognitive theories meet this explanatory requirement.

ADDITIONAL CONSIDERATIONS IN THE CONCEPTUALIZATION AND ASSESSMENT OF COGNITIVE VULNERABILITY

Cognitive vulnerability is a core construct in the cognitive theories of depression that provides the mechanism by which, given the same negative life event, some individuals become depressed whereas others do not. This construct, more so than any other in the theories, has captured research attention. Indeed, despite the fact that Abramson et al.'s (1989) theory is termed the hopelessness theory and features hopelessness as a proximal sufficient cause of depressive symptoms, far more work testing the theory has examined predictions about cognitive vulnerability than about hopelessness. Perhaps researchers have focused on cognitive vulnerability because it addresses the intuition of both clinical scientists and laypeople that some individuals seem to be highly susceptible to depression, whereas others appear to be highly resistant, even under the most dire circumstances.

A Comparison of the Cognitive Vulnerability Construct in Beck's Theory and Hopelessness Theory

Up to this point, we have focused largely on the similarities of the cognitive vulnerabilities featured in hopelessness theory and Beck's theory. Despite these conceptual similarities, however, the two theories' respective hypothesized cognitive vulnerability factors to depression are not identical. For Beck, cognitive vulnerability consists of negative schemata containing dysfunctional attitudes. According to Beck, Rush, Shaw, and Emery (1979), dysfunctional attitudes differ from more adaptive attitudes in that the former are inappropriate, rigid, and excessive. Beck (1976) provided examples of dysfunctional attitudes hypothesized to provide vulnerability to depression (pp. 255–256): "In order to be happy, I have to be successful in whatever I undertake," "If I make a mistake, it means that I am inept," and "My value as a person depends on what others think of me." As these examples illustrate, dysfunctional attitudes often are expressed in absolute and extreme terms.

Indeed, Beck et al. (1979) described dysfunctional attitudes as often incorporating phrases such as "should," "have to," and "must." Additionally, Beck contended that many dysfunctional attitudes have an "*if, then*" contingency as in one of the examples above. These rigid, extreme, and idealized standards (i.e., dysfunctional attitudes) often involve themes regarding approval and love from others, performance, perfectionistic standards, and self-worth.

In the hopelessness theory (Abramson et al., 1989), cognitive vulnerability is defined as the tendency to make negative inferences about the causes, consequences, and self-implications of a negative life event. Some of the dysfunctional attitudes in Beck's theory clearly overlap with the negative inferential tendencies featured in hopelessness theory. For example, the dysfunctional attitude "If I make a mistake, it means that I am inept" is an instance of making a negative inference about the self (I'm inept) given the occurrence of a negative life event (making a mistake). Given this overlap, it is not surprising that positive correlations between the CSQ and the DAS routinely are obtained (e.g., Haeffel et al., 2002). However, other aspects of dysfunctional attitudes, such as their focus on an individual's rigid and extreme rules for happiness, have no apparent counterpart in the hopelessness theory.

Consistent with these theoretical distinctions, a growing body of empirical evidence indicates that the cognitive vulnerabilities defined in Beck's theory and hopelessness theory, respectively, represent *distinct* constructs. Cross-sectional studies investigating the factor structures of measures of dysfunctional attitudes and attributional style have found that they load on two distinct factors labeled Self-Regard and Attributional Generality, respectively (e.g., Joiner & Rudd, 1996). In addition, Spangler, Simons, Monroe, and Thase (1997) reported that depressed patients with a cognitive vulnerability–stress match, according to Beck's theory, were largely distinct from patients with a cognitive vulnerability–stress match according to hopelessness theory. Moreover, Hollon, DeRubeis, and Evans (1996) reported that attributional styles significantly mediated the relapse prevention effect of cognitive therapy whereas dysfunctional attitudes did not. Similarly, Alloy, Reilly-Harrington, et al. (1999) found that attributional styles interacted with life events to prospectively predict both depressed and hypomanic symptoms, whereas dysfunctional attitudes did not. Furthermore, using an unselected sample of college students to begin to "unpack" the generic cognitive vulnerability of the CVD Project (Alloy & Abramson, 1999), Haeffel et al. (2002) reported that negative cognitive styles, but not dysfunctional attitudes, uniquely predicted lifetime history of clinically significant depression and anxiety comorbid with depression. Preliminary investigations of a link between cognitive and biological vulnerability to depression have revealed yet another difference: the depressogenic attributional style was related to hemispheric asymmetry (Davidson, Abramson, Tomarken, & Wheeler, 1991), whereas dysfunctional attitudes were not (Gotlib, Ranganath, & Rosenfeld, 1998). Finally, *opposite* patterns of gender differences are found for the two cognitive vulnerabilities. High school and college females exhibit more negative cognitive styles than do their male counterparts (Angell et al., 1999; Hankin & Abramson, 2002). In contrast, college males exhibit higher levels of dysfunctional attitudes than do college females (Angell et al., 1999; Gotlib, 1984; Haeffel et al., 2002). Thus, although overlapping, the vulnerability factors featured in Beck's theory and hopelessness theory are distinct in important ways. We caution, however, that the empirical differences between the two vulnerabilites might reflect measurement, as opposed to true conceptual, differences. While the intent of both the CSQ and the DAS is to identify cognitively vulnerable people, the creators of these two questionnaires have adopted different measurement approaches.

There are two noteworthy differences between the CSQ and the DAS. First, the CSQ provides the participant with hypothetical situations that serve as references from which

questions are to be answered whereas the DAS does not. Some researchers contend that priming is required for adequate measurement of cognitive vulnerability (Persons & Miranda, 1992; see Just et al., 2001, for further discussion). The CSQ is unique in that it provides a "built-in" event priming mechanism (Hollon, 1992). For each hypothetical situation, participants are asked to vividly imagine the situation happening to them (i.e., they prime themselves) and make inferences about its cause, consequences, and self-concept implications. The DAS does not provide this built-in priming mechanism, but rather asks participants to make ratings about statements without a contextual situation upon which to rely.

The second major difference between the CSQ and the DAS is the level of self-awareness needed by participants to complete the questionnaire accurately. The CSQ, compared to the DAS, requires a much lower level of self-awareness on the part of the participant. The CSQ simply asks questions about the cause, consequence, and self-concept implications of specific events and does not directly inquire whether or not the participant "thinks" he or she has a negative cognitive style. In essence, participants provide a "cognitive sample" on the CSQ that is thought to reveal their general cognitive style. Conversely, the DAS *does* require the participant to possess a relatively high level of self-awareness. The DAS directly asks participants to make global judgments about themselves. For example, an item on the DAS reads, "My value as a person depends greatly on what others think of me." To rate accurately how much he or she agrees or disagrees with this statement, the participant must have insight into his or her own concept of self-worth. Moreover, as this example illustrates, items composing the DAS are very general statements (unlike the specific events on the CSQ). Answering questions about general beliefs, rather than specific events, requires a greater degree of self-awareness on the part of the participant. Indeed, Beck et al. (1979) stated that dysfunctional attitudes are "not articulated by the patient without considerable introspection" (p. 247).

These two differences in measurement may explain some of the obtained empirical differences between the cognitive vulnerabilities featured in Beck's theory and in the hopelessness theory. However, even with equal accuracy of measurement, dysfunctional attitudes and negative cognitive styles, although overlapping, seem to be somewhat distinct constructs. Indeed, we speculate that negative cognitive styles may be more generally maladaptive, whereas some "dysfunctional" attitudes such as perfectionism may be adaptive when paired with other characteristics such as high ability and/or high self-efficacy but maladaptive when paired with low ability and/or low self-efficacy (see also Gibb, Zhu, Alloy, & Abramson, in press).

An important future task will be to compare the CSQ and the DAS to other operationalizations of cognitive vulnerability. In this regard, Alloy, Abramson, Murray, Whitehouse, and Hogan (1997) reported that cognitively HR participants in the CVD project exhibited negative biases on self-referent information-processing tasks. The recent debate in social psychology about implicit (automatic, overlearned, and nonconscious) versus explicit (more controlled, conscious) attitudes (see Bosson, Swann, & Pennebaker, 2000) has a parallel in work on cognition and depression. Relying on "network" theories of cognition, some investigators have developed implicit measures of depressive self-schemata such as the "emotional" Stroop task (e.g., Gotlib & McCann, 1984). In contrast, both the CSQ and the DAS can be construed as more explicit measures. Currently, little direct evidence exists that the implicit measures of cognitive vulnerability prospectively predict the development of clinically significant depression. In contrast, the CVD results demonstrate that a more explicit measure (i.e., pooled CSQ and DAS) of cognitive vulnerability prospectively predicts both first onsets and recurrences of clinically significant depression in a behavioral high-risk design. We are excited about the possibility of developing implicit measures of negative cog-

nitive styles and dysfunctional attitudes to see if they predict as well in a behavioral high-risk design as do the current explicit measures of these constructs.

THE RELATION BETWEEN COGNITIVE VULNERABILITY AND RUMINATION: INSIGHTS FROM A SELF-REGULATORY PERSPECTIVE

In parallel with work on the hopelessness theory and Beck's theory, investigators have demonstrated that rumination also plays an important role in many aspects of depression. Work by Martin and Tesser (1996) and Nolen-Hoeksema (1991) suggests that rumination represents a process of perseverative attention directed to specific (often internal) content. For example, in depression, attention may be focused on an individual's negative affect and the causes and consequences of their depressed mood (Nolen-Hoeksema, 1991). The tendency to ruminate is associated with vulnerability to depressed mood (Nolen-Hoeksema & Morrow, 1991), onsets of depressive episodes (Just & Alloy, 1997), longer (Nolen-Hoeksemsa, Morrow, & Fredrickson, 1993) and more severe (Nolen-Hoeksema, Parker, & Larson, 1994) episodes of depression, gender differences in vulnerability to depression (Nolen-Hoeksema, Larson, & Grayson, 1999), and various symptoms of depression (Gotlib, Roberts, & Gilboa, 1996).

What is the relation between cognitive vulnerability, as defined in hopelessness theory and Beck's theory, and rumination? Although little has been done to integrate these constructs (but see Alloy et al., 2000, and Spasojevic & Alloy, 2001, for exceptions), we take a self-regulatory perspective and suggest that cognitive vulnerability, by its very nature, should lead to rumination. Thus, we argue that cognitively vulnerable individuals should be at especially high risk for engaging in rumination. To make this case, we present a four-part argument that describes rumination as a particular type of self-regulatory problem.

The Four-Part Argument

Attention Increases Salience and Influence

Attention increases the salience and influence of whatever content is the subject of focus (see also Williams, Watts, MacLeod, & Matthews, 1997). For example, self-focus increases the intensity of whatever affective state is most salient at the time (Ingram, 1990). When attention is focused on the self, people are more responsive to whatever mood state has been induced, regardless of whether that mood state was positive or negative (Scheier & Carver, 1977). From this perspective, attention is a "spotlight" that selects the "subject" that will influence behavior and mood at a given moment.

Discrepancies Initiate a Self-Regulatory Shift of Attention

Self-regulatory theories converge on the conclusion that a negative event, or "discrepancy," initiates a shift of attention to evaluation of the current situation. For example, Gray (1994) has posited a behavioral inhibition system (BIS) that responds to punishment cues and negative events (as well as unexpected events) by initiating a self-regulatory process that involves the interruption or inhibition of ongoing behavior to "check out" or evaluate the undesired or unexpected situation. Thus, the individual switches attention to addressing the current discrepancy. Similarly, Carver and Scheier (1998) specified that a self-regulatory cycle begins with attention directed internally (e.g., self-focus), allowing individuals to compare their current versus their desired state (e.g., goal) and to initiate behavior to reduce any discrepancy (see also Higgins, 1987).

So far, so good. Self-regulatory theories emphasize that when confronted with a negative event, it is adaptive for people to switch their attention to the problem (presumably to try to remedy it). Of course, as Figures 11.1 and 11.2 show, in the cognitive theories of depression, the occurrence of a negative event is the first step in the causal chain culminating in depression. The self-regulatory theories suggest that at this point, people, cognitively vulnerable and nonvulnerable alike, shift their attention to an evaluation of the unfortunate situation. Consistent with the self-regulatory perspective, the cognitive theories of depression emphasize that when confronted with a negative event, individuals make inferences about the cause, consequences, and implications for self-concept of the event (part of the "checking" process). Recall the example presented earlier in the chapter of the two students who failed the test. According to the self-regulatory perspective, upon discovering the failing grade, both students will begin self-regulation by shifting attention to consideration of the "discrepancy" between the desired grade and the actual "F." This attentional switch to negative cognitive content (i.e., a grade of "F") is likely to produce initial negative affect in *both* students regardless of their respective levels of cognitive vulnerability. Consistent with this prediction, the academic midterm studies (e.g., Metalsky et al., 1987, 1993) have shown that both cognitively vulnerable and cognitively nonvulnerable students experience initial depressive affect after receiving a low grade. Thus, it appears that the crucial difference between cognitively vulnerable and nonvulnerable individuals lies in what happens after the initial self-regulatory step.

Cognitively Vulnerable Individuals Have Difficulty Disengaging Their Attention from This Evaluation ("Sticky Attention")

Drawing on work by self-regulation theorists (e.g., Carver & Scheier, 1998; Pyszczynski & Greenberg, 1987), we contend that cognitively vulnerable individuals find it more difficult to disengage from this "checking" process (i.e., focusing attention on the discrepancy), once initiated, than do nonvulnerable individuals. Cognitively vulnerable individuals become stuck in the self-regulatory cycle and perseverate in this checking. This self-regulatory perseveration (Pyszczynski & Greenberg, 1987) constitutes rumination.

Why do cognitively vulnerable individuals persist in a ruminative checking process whereas nonvulnerable individuals disengage? There are three hypothesized way to disengage from rumination: (1) resume progress toward the goal (i.e., a solution for resolving the discrepancy, remedying the problem, or overcoming the obstacle, etc., is found); (2) relinquish desire for the goal (Pyszczynski & Greenberg, 1987); or (3) distract attention away from the discrepancy or problem (Carver & Scheier, 1998; Martin & Tesser, 1996; Nolen-Hoeksema, 1987). We suggest that cognitively vulnerable individuals have difficulty passing through any of these three exits.

1. *Resume progress toward a goal (i.e., a solution is found).* Cognitively vulnerable individuals are not likely to find a solution to their discrepancy or problem. Indeed, their stable and global attributions encourage the perception that problems cannot be solved. In our example of the student who attributed her failing grade to low intelligence, there is no easy solution for doing better in the future. A problem without a solution is described by Mandler (1972) as helplessness, one consequence of which is "the repetitive circling through of previous behavior patterns" (p. 260). If we view attention as a kind of cognitive behavior, then this is a good description of rumination. Moreover, the globality of the student's attribution suggests that other negative events will befall her outside of school and thus suggests new discrepancies about which to ruminate. The inferred negative consequence that the failing grade will prevent her from getting into medical school suggests an even more major discrepancy to ponder.

In contrast, unstable and specific attributions for negative events suggest reasonable solutions. For the other student in the example who attributed the same failing grade to not studying hard enough, the solution is to study harder. He can exit from the self-regulatory cycle and move on to preparing for the next test. Moreover, because he did not infer that negative consequences will result from the failing grade, he does not have any additional negative events to "check out."

2. *Relinquish desire for the goal.* We suggest that cognitively vulnerable individuals are also less likely than are nonvulnerable individuals to relinquish desire for the goal, even when a solution appears impossible or unlikely. Thus, cognitively vulnerable individuals may be stuck wanting something they are convinced they cannot have (Pyszczynski & Greenberg, 1987).

Both hopelessness theory and Beck's theory are compatible with this point of view. First, the stable, global causal attributions featured in hopelessness theory are likely to increase the importance of negative outcomes by *linking* (see Martin & Tesser, 1996) the current negative situation to other, more important goals. Following Carver and Scheier (1998), we suggest that goals and behaviors are structured hierarchically. For example, at the top of a student's hierarchy is a globalized sense of idealized self, who she really wants to be. At the next subordinate level in the hierarchy are guiding principles that specify the qualities required to achieve her idealized self, such as intelligence. The type of behaviors needed to be intelligent are embodied at the next level down in the hierarchy and might include achieving good test grades. Our first student attributed her failing grade to low intelligence, a stable, global attribution that links her lower order behavior (failing the test) to her higher order goal of being intelligent. Given the hierarchical organization of goals, being intelligent is more important to her than the test itself. Indeed, one might argue that the student's failure is important to the extent that it is linked to her higher order goals (e.g., being intelligent). It is easy to see that the same logic applies for this student's negative inferences about consequences ("The low grade will prevent me from getting into medical school") and self ("I'm worthless") linking to higher order goals.

According to Carver and Scheier (1998), higher order goals such as these (being intelligent and worthy, getting into medical school) are much harder to relinquish because doing so necessitates a reorganization of one's value system. In contrast, the inferences of the cognitively nonvulnerable person do not have such negative implications for higher order goals. Thus, cognitively vulnerable individuals may be less likely than cognitively nonvulnerable individuals to relinquish their currently thwarted goal because it is vital to their self-concept.

Linking also is related to the perfectionism and rigid standards embodied in Beck's concept of cognitive vulnerability. Items on the DAS such as "If I don't set the highest standards for myself, I am likely to end up a second-rate person" reveal evidence of linking lower to higher order goals. If a person *must* achieve a certain standard (rigidity) and must achieve it perfectly, it is unlikely that the person will achieve it (unless he or she is exceptionally competent) or relinquish desire for it.

3. *Distraction.* Distraction involves shifting attention to content unrelated to the current discrepancy—thinking about something else. Distraction can be construed as the *temporary* relinquishing of a goal. As we argued above, cognitively vulnerable individuals should have difficulty relinquishing a goal, even temporarily.

Although distraction is only a temporary solution because it does nothing to directly relieve the conditions that led to rumination in the first place, it may be viewed as a form of problem solving, either strategic or automatic. Such a solution will be especially difficult to implement for a cognitively vulnerable individual, whose attention already is strongly focused on an important, threatened goal in his or her hierarchy. As already suggested, the

more important the goal of current focus, the more difficult it should be to move attention elsewhere. The student who believed that low intelligence caused her to fail the exam, that she never will get into medical school, and that she is worthless would seem to have an especially hard time becoming *engrossed* in a new goal or activity (a requirement for effective distraction). Distraction can also take the form of overt behavior (e.g., exercising). However, the hopelessness engendered by cognitive vulnerability likely suggests that such behaviors will not reduce negative feelings or lead to positive outcomes. Moreover, the response initiation deficits hypothesized to result from hopelessness (Abramson et al., 1989) should decrease the hopeless person's likelihood of initiating voluntary distracting behaviors.

In summary, discrepancy checking may be a relatively universal self-regulatory response following a negative life event. However, cognitively nonvulnerable individuals may quickly disengage their attention from their discrepancy by finding a solution, relinquishing their current thwarted goal, or distracting themselves. In contrast, these exits may be blocked for cognitively vulnerable individuals, such that they get "stuck" ruminating about a goal they can neither attain nor relinquish. In short, following a negative event, the cognitively vulnerable individual is left with the spotlight of attention fixed on relatively negative content (e.g., expectations of hopelessness, stable and global attributions for negative events, anticipated future negative events, negative views of the self; see also Teasdale, 1988). In turn, this attentional spotlighting of negative content allows it to strongly influence affect and behavior (see below). Whereas cognitively vulnerable and nonvulnerable individuals alike may experience initial negative affect when first encountering a negative event/discrepancy, the nonvulnerable are protected from the development of more severe or prolonged depression whereas the vulnerable are not (see also Teasdale, 1988). Consistent with this hypothesis, in the academic midterm studies (e.g., Metalsky et al., 1987, 1993) cognitively vulnerable students experienced persistent depressive mood for several days after receiving a low grade whereas nonvulnerable students recovered more quickly.

Increased Activation of Negative Content and Resulting Symptoms

The inability to disengage attention from negative cognitive content is a plausible mechanism underlying several symptoms of HD. Most obviously, rumination should increase negative affect because it provides a constant reminder of just how bad things appear to be. Difficulty in disengaging attention from a discrepancy and its associates also may account for the poor concentration characteristic of many depressed individuals (Pyszczynski & Greenberg, 1987). Simply put, if attention is directed to task-irrelevant cues (e.g., negative content related to the discrepancy, failure, or negative event), it is not available to activate and enhance task-relevant cues. In addition, rumination may contribute to insomnia, another hypothesized symptom of HD, by interfering with the calm state associated with falling asleep. Finally, rumination may increase the risk of suicidality, a core symptom of HD. A person not focused currently on negative cognitions is unlikely to be suicidal. Providing clinical support for the intuitive notion that suicide is related to focusing attention on negative thoughts, one common intervention in preventing suicide is to remind the suicidal individual of reasons to live.

Summary

Our integration of self-regulatory perspectives with hopelessness theory and Beck's theory highlights the important association between cognitive vulnerability and rumination. Namely, a negative event causes an interruption of ongoing behavior and calls for a shift of

attention to monitor the discrepancy between the actual event and the desired event. A person is ruminating when his or her attention is "stuck" on this discrepancy and its associates. The three exits from rumination are less available to the cognitively vulnerable individual than to the nonvulnerable individual. For the cognitively vulnerable person, attention continues to activate discrepancies and other negative content, a process that may account for some of the symptoms composing HD. Thus, the negative cognitive content associated with cognitive vulnerability helps instigate the process of rumination, which in turn contributes to depressive symptoms.

Empirically supporting this view, both stress-reactive (Alloy et al., 2000) and mood-related (Spasojevic & Alloy, 2001) rumination have been found to be positively related to cognitive vulnerability. Furthermore, in the CVD Project, rumination was found to mediate the association between cognitive vulnerability and the prospective development of episodes of major depressive disorder, suggesting that rumination is a proximal mechanism through which cognitive vulnerability contributes to depression (Spasojevic & Alloy, 2001).

FUTURE DIRECTIONS: PLACING THE COGNITIVE THEORIES OF DEPRESSION IN A PSYCHOBIOLOGICAL CONTEXT

We close by suggesting an important future direction for the cognitive theories of depression. Given the empirical support for these theories and the success of cognitive therapy for depression, it is critical to integrate the cognitive approach with other successful approaches to depression. Much important work has been conducted demonstrating the fruitfulness of integrating cognitive and interpersonal approaches to depression (e.g., Gotlib & Hammen, 1992; Haines, Metalsky, Cardamone, & Joiner, 1999). However, little has been done to integrate the cognitive perspective with powerful biological approaches to depression and, more generally, with major advances in understanding the psychobiology of motivation. Below we speculate about how we may move toward placing the cognitive theories of depression in a psychobiological context.

Over the past 25 years, experimental psychologists have made major advances in understanding the motivational bases of behavior. Coming from diverse perspectives including animal learning and behavioral pharmacology (e.g., Gray, 1994), the neural substrates of emotion (e.g., Davidson, 1994; Davidson, Pizzagalli, & Nitschke, Chapter 9, this volume), personality (e.g., Cloninger, 1987), and psychopathology (e.g., Depue & Iacono, 1989), investigators have converged on the conclusion that two (sometimes more) fundamental psychobiological systems are critical in regulating behavior. One of these systems regulates approach behavior to attain rewards and goals, which the other regulates withdrawal and/or inhibition of behavior in response to threat and punishment. The former variously has been called the Behavioral Approach System (BAS; Gray, 1994), Behavioral Activation System (BAS; Cloninger, 1987; Fowles, 1980), and the Behavioral Facilitation System (BFS; Depue & Iacono, 1989). We will call this system the BAS. The latter system typically is called the Behavioral Inhibition System (BIS; Gray, 1994), although Davidson (1994) has referred to it as a withdrawal system. We will term this system the BIS.

In its regulation of approach behavior to attain rewards and goals, the BAS is sensitive to signals of reward, nonpunishment, and escape from punishment. These signals can be external (e.g., environmental events such as the presence of an attractive goal object) or internal (e.g, expectations of goal attainment). Activation of the BAS by these signals causes the person to begin (or to increase) movement toward goals. Activation of the BAS is hypothesized to be associated with hope, elation, and happiness (e.g., Carver & White, 1994).

In contrast, in its regulation of withdrawal and/or inhibition of behavior in response to

threat or punishment, the BIS is sensitive to signals of punishment and nonreward (e.g., failure). Again, as with the BAS, the signals activating the BIS can be external environmental events or internal cognitions. Activation of the BIS inhibits behavior that may lead to negative or painful outcomes in response to signals of punishment, nonreward, and failure. Activation of the BIS is hypothesized to be associated with negative affect such as anxiety (e.g., Carver & White, 1994). Investigators have suggested that BAS and BIS stand in reciprocal relation to each other. As one becomes active, the other becomes inactive (e.g., Fowles, 1993).

Within this psychobiological motivational perspective, depression is viewed as associated with the activation of aversive motivation (increased BIS; e.g., Fowles, 1993) and/or a disruption or "shutdown" of appetitive motivation (decreased BAS; Davidson, 1994; Fowles, 1993), with the greater emphasis being on the latter. Indeed, examination of the hypothesized symptoms of HD reveals that some (e.g., brooding, difficulty in concentration, insomnia) appear to reflect an active BIS, whereas other symptoms (e.g., retarded initiation of voluntary responses, lack of energy, apathy) likely reflect an inactive BAS.

It is useful to examine, from a BIS/BAS perspective, the hypothesized causal pathways that culminate in depression in the cognitive theories. The causal chain starts with the occurrence of a negative event, as in our example of the student failing a test. The receipt of the failing grade should activate the BIS, which entails the interruption or inhibition of ongoing behavior as the student "evaluates" the failing grade (e.g., makes inferences about the grade's cause, likely consequences, and self-concept implications). As we argued above, initial negative affect should occur at this point in the causal chain. However, if the student is cognitively nonvulnerable, he or she will make inferences that facilitate exit from this self-regulatory process (and the associated negative affect), and he or she will resume progress toward goals. This resumption of goal-seeking activity should be reflected in deactivation of the BIS and reactivation of the BAS. Thus, the cognitively nonvulnerable student is protected from a large-scale deactivation of the BAS. In contrast, the cognitively vulnerable student makes inferences (e.g., I failed the test because I'm stupid) that lead to hopelessness about achieving important current and future goals and, in turn, the symptoms of HD. Fowles (1993) has argued that as expectancies move in the direction of hopelessness, appetitive motivation (e.g., BAS) will decrease. From this perspective, then, hopelessness and the associated symptoms of HD together may represent the cognitive, affective, and behavioral manifestations of an inactive BAS (and possibly of a relatively more active BIS).

Although much important work has been conducted on cognitive and biological vulnerability to depression, these two lines of research have proceeded in relative isolation from each other (Gotlib & Abramson, 1999). However, placing the cognitive theories in a psychobiological motivational context facilitates their integration with powerful biological theories of depression. Enabling such an integration, recent research (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997) has begun to elucidate the neural circuitry involved in implementing the approach and inhibition/withdrawal systems. Specifically, the BAS, as measured by Carver and White's (1994) BIS/BAS scale, is associated with increased left frontal cortical activity during resting baseline (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). Relating depression to frontal cortical activity, Davidson and colleagues (see Davidson, 1994) have demonstrated that depressed individuals exhibit greater relative right-sided frontal activation (i.e., lesser relative left-sided frontal activation) of their cerebral hemispheres than do nondepressed individuals. In Davidson's scheme, this pattern is consistent with withdrawal from the environment (active BIS) and decreased approach behavior (inactive BAS). These cerebral activation differences appear to be, at least in part, state-independent. Asymptomatic remitted depressed individuals exhibit greater relative right-sided frontal activation than never-depressed individuals (e.g., Gotlib et al., 1998;

Henriques & Davidson, 1990). Based on these and other findings, Davidson and colleagues (e.g., Davidson, 1994) and Gotlib et al. (1998) have suggested that relative right frontal hemispheric activation may represent a vulnerability to depression.

What is the nature of the relationship between cognitive vulnerability to depression and biological vulnerability to this disorder as indexed by patterns of cerebral asymmetry? To the extent that hopelessness, the expectation to which cognitively vulnerable individuals are hypothesized to be predisposed, may be particularly powerful in signaling a shutdown of approach motivation (inactive BAS), we suggest that cognitively vulnerable individuals may be characterized by relative right-sided frontal activation of their cerebral hemispheres, the biological vulnerability featured in Davidson's theory. Consistent with this hypothesis, Davidson et al. (1991) reported that attributionally vulnerable college students exhibited relative right frontal hemispheric activation. In contrast, Gotlib et al. (1998) failed to obtain a relationship between cognitive vulnerability as measured by DAS scores and hemispheric asymmetry. Consistent with our earlier comments, dysfunctional attitudes, as currently conceptualized and operationalized in the DAS, may include some adaptive components such as perfectionism. It is intriguing to consider the possibility that individuals characterized by the combination of perfectionism and high self-efficacy (e.g., Arnold Schwarzenegger!) may show relatively high approach motivation and low withdrawal motivation (relative left frontal hemispheric activation), the pattern opposite to the biological vulnerability to depression featured in Davidson's theory. The presence of such individuals might have diluted the relationship between DAS scores and cerebral asymmetry in Gotlib et al.'s study.

Future research that "unpacks" the cognitive vulnerability featured in Beck's theory and that utilizes various measures of cognitive vulnerability (e.g., explicit vs. implicit) will facilitate the empirical comparison of biological and cognitive vulnerability. Davidson et al.'s (1991) results provide preliminary evidence that attributional vulnerability and right frontal hemispheric asymmetry may share a common biocognitive process that increases people's risk for depression. We speculate that those cognitive styles that predispose an individual to hopelessness and to a generalized loss of self-efficacy should be especially likely to be related to biological vulnerabilities associated with "shutdowns" of the behavioral approach system. Integrating the cognitive and biological theories of depression promises to be an exciting and important direction for future theory and research.

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