



# REMITTED DEPRESSION STUDIES AS TESTS OF THE COGNITIVE VULNERABILITY HYPOTHESES OF DEPRESSION ONSET: A CRITIQUE AND CONCEPTUAL ANALYSIS

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**ABSTRACT.** *Investigations of cognitive patterns among individuals who have recovered from a depressive episode (i.e., remitted depressives) have figured importantly in evaluations of the validity of the vulnerability hypotheses of the cognitive theories of depression. However, we suggest that remitted depression studies as typically conducted and interpreted are inadequate tests of the cognitive vulnerability hypotheses of depression onset for four reasons: (1) remitted depression studies are based on the erroneous assumption that cognitive vulnerability should be an immutable trait; (2) remitted depression studies use a logically “backward” participant selection strategy in which participants are selected on the basis of the “dependent” variable (depression) and then compared on the “independent” variable (cognitive vulnerability), which is likely to result in heterogeneity of cognitive vulnerability among both the remitted depressed as well as the nondepressed groups given the causal relations specified in the cognitive theories of depression; (3) many remitted depression studies have ignored the possible activating role of stress in the cognitive vulnerability-stress theories, particularly Beck’s theory, and thus, may attempt to assess cognitive vulnerability at a time when it is not operative (i.e., priming hypothesis); and (4) remitted depression studies inappropriately use postmorbid participants to test causal hypotheses, and therefore, are ambiguous about whether negative cognitive styles observed in remitted depressed persons are vul-*

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nerabilities as opposed to consequences of depression (i.e., scar hypothesis). As a remedy, we advocate the use of a theory-guided behavioral high-risk strategy to more adequately test the cognitive vulnerability hypotheses of depression onset. © 2000 Elsevier Science Ltd.

**KEY WORDS.** Cognitive vulnerability, Remitted depression, Scar hypothesis, Priming.

COGNITIVE THEORIES OF depression have generated enthusiasm as well as controversy. Both Beck's (1967, 1987) cognitive model of depression and the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989), as well as its precursor, the reformulated helplessness theory of depression (Abramson, Seligman, & Teasdale, 1978), contain a "cognitive vulnerability hypothesis." According to these hypotheses, particular maladaptive cognitive patterns increase people's risk for onset of depression when they experience negative life events. In this view, cognitive vulnerability is an individual difference variable which contributes causally to the development of depressive episodes (see Bootzin & McKnight, 1998). Consistent with other conceptualizations (e.g., Ingram, Miranda, & Segal, 1998), our definition of cognitive vulnerability includes the idea that cognitive vulnerability need not be invariant over a person's lifespan. In addition, we emphasize that cognitive vulnerability is hypothesized to contribute to first episodes as well as recurrences of depression. We use the term *onset*, then, to refer to the occurrence of each episode, regardless of whether it is the first or a subsequent episode of depression.

Based largely on the cognitive vulnerability hypotheses, psychological research on depression over the past 15 years has focused on evaluating the potential causal role of dysfunctional thinking patterns in depression. The body of empirical evidence generated by this research has led to highly conflictual interpretations of the status of cognitive patterns as causes, concomitants, or consequences of depressive states (Abramson et al., 1989; Abramson, Alloy, & Metalsky, 1988; Barnett & Gotlib, 1988; Brewin, 1985; Coyne & Gotlib, 1983; Haaga, Dyck, & Ernst, 1991; Persons & Miranda, 1992; Peterson & Seligman, 1984).

One research design that has figured importantly in the search for cognitive vulnerability factors for depression is the *remitted* depression paradigm, of which there are two major types. In the cross-sectional version, the cognitive patterns of formerly depressed persons are compared to those of currently symptomatic depressives and non-depressed controls. In longitudinal versions of the design, depressed persons' cognitive patterns may be compared to those of nondepressed controls both when the depressives are symptomatic and when their symptoms have remitted. In addition, longitudinal within-subject comparisons are sometimes made of depressed participants' cognitive patterns in the symptomatic versus the remitted states. Alternatively, depressed persons who subsequently remit may be compared to those who do not, on cognitive patterns measured when all were depressed.

A vast number of "remitted depression studies" have been conducted because many investigators (e.g., Schreiber, 1978; Silverman, Silverman, & Eardley, 1984) believed that this design afforded a powerful opportunity to untangle the symptoms or concomitants of depression from vulnerability factors for this disorder. In fact, such remitted depression designs have been touted by some investigators as having the potential to provide the "proof of the pudding" that particular cognitive patterns actually are vulnerability factors for depression (e.g., Schreiber, 1978; Kovacs & Beck, 1978). In this vein, one research team (Silverman et al., 1984) titled an article, "Do

maladaptive attitudes cause depression?" and then conducted a remitted depression study to answer the question. The idea was that if dysfunctional attitudes or negative cognitive styles actually are vulnerability factors for depression, then these cognitive patterns should be highly stable and persist beyond remission of a current episode. On the other hand, the guiding logic went, if such cognitive patterns are symptoms or concomitants of depression, then they should be present during the depressive episode but dissipate upon remission of the episode. Thus, the key assumption underlying the remitted depression design was that cognitive vulnerability must be traitlike. According to this assumption, to qualify as a vulnerability factor, a cognitive pattern exhibited by depressed people must continue to be exhibited by them when they have recovered from their depression. On this view, any cognitive pattern that is not exhibited by formerly depressed people cannot qualify as a vulnerability factor for depression. If this assumption is incorrect, as we argue below, then the typical remitted depression studies do not provide valid tests of the cognitive vulnerability hypotheses.

Although some studies have found that remitted depressives continued to evidence cognitive patterns that were more negative than those of normal controls (e.g., Eaves & Rush, 1984; Teasdale & Dent, 1987), the majority of remitted depression studies have found that when previously depressed participants are in remission, they no longer evidence the distinctive maladaptive cognitive styles hypothesized by cognitive theorists to contribute vulnerability to onset of depression (see Barnett & Gotlib, 1988; Ingram et al., 1998; Persons & Miranda, 1992 for reviews). These findings have led to great controversy. Based in substantial part on findings from remitted depression studies, some reviewers (e.g., Barnett & Gotlib, 1988; Wilson, Nathan, O'Leary, & Clark, 1996) have concluded that the vulnerability hypotheses of the cognitive theories of depression are not well supported and that negative cognitive patterns may be viewed more appropriately as concomitants rather than as contributory causes of depression onset.

For example, based on their finding that depressed patients' dysfunctional attitudes, as measured by the Dysfunctional Attitude Scale (DAS), decreased significantly as they recovered and, moreover, at remission, did not differ significantly from those of a comparison nondepressed group, Silverman et al. (1984) answered the question posed in the title of their paper ("Do maladaptive attitudes cause depression?") with a resounding "No." Similarly, a review article in 1988 (Barnett & Gotlib, 1988) concluded ". . . there is little evidence in adults of a cognitive vulnerability to depression" (p. 97) based, in substantial part, on findings that negative attributional styles and dysfunctional attitudes decreased significantly as depressive symptoms remitted, and remitted depressives, as a group, did not differ from nondepressed controls with respect to dysfunctional attitudes or negative attributional styles. Finally, in an evaluation of the cognitive vulnerability hypotheses of depression onset, the authors (Wilson et al., 1996) of a current textbook on abnormal psychology concluded, "Beck's idea that negative cognitions *cause* depression onset has not been confirmed, however. Although there are contrary findings . . . , most studies find that when people have recovered from depression, their negative thinking disappears, as well . . ." (p. 213). The authors further concluded, "Data regarding the important question of whether this attributional style *causes* depression are again largely negative . . . For example, attributional style . . . doesn't remain after the depression disappears" (p. 214).

The negative appraisals of the validity of the cognitive vulnerability hypotheses provoked by the results of remitted depression studies have led some researchers to challenge the adequacy of remitted depression studies as tests of these hypotheses (e.g.,

Abramson et al., 1988). Alternatively, these negative evaluations have galvanized other investigators to develop “priming” versions of the remitted depression paradigm, in which there is an attempt to activate the cognitive patterns prior to assessing them in the remitted state (e.g., Ingram et al., 1998; Miranda & Persons, 1988; Segal & Ingram, 1994). Given the importance afforded remitted depression studies in many investigators’ evaluations of the validity of the cognitive theories of depression, we believe a careful analysis of these studies, both regular and priming versions, as tests of the cognitive vulnerability hypotheses of depression onset, is warranted.

Consequently, in this article, we analyze and critique remitted depression studies, as typically conducted and interpreted, with the goal of examining the adequacy of these studies in providing tests of the vulnerability hypotheses of two of the major cognitive theories of depression: the hopelessness theory and its predecessor (Abramson et al., 1978, 1989), as well as Beck’s theory (Beck, 1967, 1976, 1987). With respect to all of the conceptual points we make in the sections to follow, the more recent hopelessness theory of depression (Abramson et al., 1989) and its predecessor, the reformulated helplessness theory (Abramson et al., 1978), are equivalent. We argue that negative conclusions about the cognitive vulnerability hypotheses based on remitted depression studies conducted to date are not justified because these studies do not have sufficient theoretical fidelity to provide adequate tests of the cognitive vulnerability hypotheses. Specifically, we suggest that typical remitted depression studies: (1) are based on the erroneous assumption that cognitive vulnerability should be an immutable trait; (2) have used a logically “backward” participant selection strategy in which participants are selected on the basis of the “dependent” variable (depression) and then compared on the “independent” variable (cognitive vulnerability) which is likely to result in heterogeneity of cognitive vulnerability among both the remitted depressed as well as the nondepressed groups given the causal relations specified in the cognitive theories of depression; (3) have ignored the possible activating role of stress in the cognitive vulnerability-stress theories, particularly Beck’s theory, and thus, may attempt to assess cognitive vulnerability at a time when it is not operative (i.e., priming hypothesis); and (4) inappropriately have used postmorbidity participants to test causal hypotheses, and therefore, are ambiguous about whether negative cognitive patterns observed in formerly depressed persons are vulnerabilities which actually contributed causally to their prior depressive episodes as opposed to consequences of depression which did not contribute causally to their prior episodes (i.e., scar hypothesis).

As a remedy to the problems we raise with typical remitted depression designs, we will advocate the use of a theory-guided behavioral high-risk strategy to more adequately test the cognitive vulnerability hypotheses of depression. Before turning to these conceptual and methodological issues, however, we first briefly describe the hopelessness theory and Beck’s theory of depression.

## THE COGNITIVE VULNERABILITY-STRESS THEORIES OF DEPRESSION

The hopelessness theory of depression (Abramson et al., 1989) is a cognitive vulnerability-stress model. This theory specifies a sequence of events in a causal chain hypothesized to culminate in hopelessness, a proximal sufficient cause of the symptoms of depression, in particular, the symptoms of the hypothesized subtype of “hopelessness depression.” The cognitive vulnerabilities featured in this theory are a set of three hypothesized “depressogenic inferential styles”: a tendency to attribute negative events to stable and global causes; to infer that negative consequences will ensue from the oc-

currence of negative life events; and to infer negative characteristics about oneself from the occurrence of negative life events. According to the cognitive vulnerability-stress component of the hopelessness theory, individuals who exhibit these depressogenic inferential styles (the cognitive vulnerability) should be especially likely to develop symptoms of depression, in particular, the symptoms of hopelessness depression, when they experience negative life events (the stress). In contrast, individuals who do not exhibit these styles should be less likely to become depressed when confronted with negative life events. However, in the absence of negative life events, individuals exhibiting depressogenic inferential styles should be no more likely to develop symptoms of depression than individuals who do not exhibit these styles. We emphasize that the hopelessness theory explicitly recognizes the likely etiologic heterogeneity of depression by featuring a hypothesized sufficient, rather than necessary, cause of a hypothesized subtype of depression, hopelessness depression. Hopelessness depression may cut across current diagnostic categories of depression (Abramson et al., 1989).

Although less well articulated, the cognitive vulnerability-stress logic of the hopelessness theory was already present in the reformulated theory of helplessness and depression (Abramson et al., 1978). Also similar to the hopelessness theory, the reformulation explicitly recognized the probable etiologic heterogeneity of depression. Thus, both the hopelessness theory and its predecessor, the reformulated helplessness theory, feature a vulnerability-stress component as well as postulate a sufficient, rather than necessary, cause of depressive symptoms.

Beck's (1967, 1976) cognitive theory of depression is also a cognitive vulnerability-stress model. Beck hypothesizes that cognitive vulnerability for depression is provided by maladaptive self-schemata containing dysfunctional attitudes revolving around themes of loss, inadequacy, failure, and so on. Such dysfunctional attitudes include beliefs such as that one's happiness depends on being perfect or on other people's approval. When these hypothesized depressogenic self-schemata are activated by the occurrence of life stresses, they lead to the appearance of specific negative cognitions (automatic thoughts) that take the form of overly negative beliefs about oneself, one's world, and one's future (the negative cognitive triad). Thus, in Beck's theory, dysfunctional attitudes are a manifestation of cognitive vulnerability to depression, whereas negative automatic thoughts are more proximal causes of depression that are usually observed as part of the depressive clinical state. Therefore, similar to the cognitive vulnerability-stress component of the hopelessness theory, in Beck's theory, people who exhibit depressogenic self-schemata containing dysfunctional attitudes (the cognitive vulnerability) are at greater risk for becoming depressed when they experience negative life events (the stress) than are people who do not possess these self-schemata.

In Beck's theory, negative self-schemata are characterized as latent until activated, primarily by the occurrence of a life event that is relevant to the content embodied in the self-schema (Beck, 1967; Beck, Rush, Shaw, & Emery, 1979). For example, Beck (1967) described a woman with a core schema of "I am stupid" which was evoked "repetitively and inappropriately in situations relevant to her intellectual ability" (p. 284). Finally, similarly to the hopelessness theory, Beck explicitly acknowledged the heterogeneity of depression by postulating that the cognitive vulnerability-stress pathway featured in his model contributes to the causation of nonendogenous unipolar, but not other, depressions (e.g., Beck, 1987; Haaga et al., 1991). For example, Beck (1987) wrote, "The longitudinal cognitive model should probably be restricted to the so-called reactive depressions; that is, those that are brought about by socially relevant events. We would then postulate that although the *negative cognitive processing* is similar

for all types of depression, the factors precipitating the various disorders vary widely.” (p. 24, italics in original). Thus, as Haaga et al. (1991) summarized, according to Beck’s theory, cognitive vulnerability contributes to the causation of only some depressions but, once depressed, all depressed individuals should develop negative cognitions, regardless of the cause of their disorder.

## ANALYSIS AND CRITIQUE OF REMITTED DEPRESSION STUDIES

We now will argue that remitted depression studies, as typically conducted and interpreted, do not have sufficient theoretical fidelity to provide adequate tests of the cognitive vulnerability hypotheses of depression featured in the hopelessness theory (as well as the 1978 reformulation) and Beck’s theory (see also Hollon, 1992; Ingram et al., 1998).

### ***Remitted Depression Studies Are Based on the Erroneous Assumption that Cognitive Vulnerability Should Be an Immutable Trait***

The first problem with the typical remitted depression studies as tests of cognitive vulnerability hypotheses of depression onset is that they are based on the erroneous assumption that cognitive vulnerability should be an immutable trait and, thus, be exhibited by remitted depressives as well as by current depressives. While it is unclear whether Beck’s theory requires depressogenic self-schemata and dysfunctional attitudes, the constituents of cognitive vulnerability in his theory, to be highly stable or traitlike, the hopelessness theory of depression allows for the possibility that the negative cognitive styles hypothesized to confer cognitive vulnerability do not possess traitlike stability. For example, in anticipating the questions to be addressed by further expansions of the theory, Abramson et al. (1989) asked, “How stable are cognitive diatheses?” (p. 365). All that is required by the hopelessness theory is that at any given moment, a person’s current cognitive style confers the predicted amount of vulnerability to depressive symptoms, specifically the symptoms of hopelessness depression.

Historically, investigators of the cognitive vulnerability hypotheses of depression seem to have inferred, incorrectly we would argue, that to qualify as a vulnerability factor for depression, a variable must possess traitlike stability. Perhaps in making this inference, depression researchers were influenced by prior theories of schizophrenia which often did feature traitlike vulnerability factors (e.g., Meehl, 1962). In addition, in referring to cognitive vulnerability, we and our colleagues (e.g., Abramson et al., 1989) have used the phrase “cognitive diathesis” which nicely denotes an individual vulnerability variable but, unfortunately, also may have connoted immutability given that this term sometimes is used in medicine to refer to a fixed characteristic providing risk for a disorder. We regret our choice of the term “diathesis” if it suggested immutability because we never intended that cognitive vulnerability should be conceptualized as immutable.

An example from medicine illuminates that an individual difference vulnerability factor for disease need not necessarily show traitlike stability and, instead, may show transient or long-term fluctuations. We emphasize that we use this analogy from medicine to help depression researchers “break set” and see that it is possible to have vulnerability wax and wane. The analogy, of course, cannot prove that cognitive vulnerability for depression actually does wax and wane. Instead, the purpose of the analogy is to undermine the guiding assumption of remitted depression studies—namely, that a vulnerability factor for a disorder must be invariant over a person’s lifetime.

Consider the role the immune system plays in vulnerability to disease. A person's vulnerability to various diseases is in part a function of the integrity of the various arms of his or her immune system (e.g., Coe, 1994; Schindler, 1991). Marked immune suppression increases vulnerability for a host of diseases such as influenza at the mild end, to cancer at the severe end. Some cases of compromised immune functioning do show relative permanence. For example, in severe combined immunodeficiency disease (SCID), infants are born with a congenital lack of all the major immune defenses. Until the advent of recent breakthroughs in treatment, children with SCID would have to live for years in germ-free rooms or "bubbles" to avoid developing the infections for which they have "immune vulnerability." An example of a change in immune functioning with subsequent traitlike stability is AIDS, or acquired immunodeficiency syndrome, in which an individual shows a massive long-term decrement in immune functioning caused by a virus. In contrast, the integrity of the various branches of a person's immune system also can show transient changes over time such as those induced by stressful life events.

The point here is that medical researchers conceptualize compromised immune functioning as a critical individual difference vulnerability factor for a host of diseases. Yet, no such researchers ever would suggest that people's immune functioning necessarily should be immutable. On the contrary, much work focuses on factors that produce changes in immune functioning and, in turn, accompanying changes in vulnerability to disorder. In our reading on this topic, we have not encountered any debates about whether compromised immune functioning is a trait or a state. Apparently, immune vulnerability is "traitlike" in some cases (e.g., SCID) and "statelike" in others (e.g., compromised immune functioning caused by stressful life events). Once the processes underlying immune vulnerability are well understood, it should be possible to predict when immune vulnerability will be a trait and when it will be a state.

This analogy from medicine illustrates that it is inappropriate to use immutability or traitlike stability as a criterion for evaluating whether or not a given variable is a vulnerability factor for a particular disorder. Immutability or traitlike stability is not required for a factor to confer vulnerability to a given disorder. Consistent with this view, some schizophrenia researchers have begun to suggest that the diathesis for schizophrenia may vary over time within a given individual as well as across individuals (see Gooding & Iacono, 1995, for a discussion of the evolution of the concept of vulnerability in the field of schizophrenia). For example, Prescott and Gottesman (1993) recently have entertained the possibility that schizogene(s) can switch on and off in a given person over time as a function of various factors, quite possibly including internal biochemical states induced by momentary stressors. Thus, given existing knowledge about individual differences in vulnerability to medical disorders as well as current theorizing about other forms of psychopathology, there is no a priori reason to require immutability or traitlike stability of any individual difference factor hypothesized to confer vulnerability to depression. Consistent with our view, Barnett and Gotlib (1988) and Coyne and Whiffen (1995) emphasized the importance of understanding change in cognitive vulnerability to depression. Similarly, Ingram et al. (1998) recently have suggested that vulnerability levels may fluctuate over time.<sup>1</sup>

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<sup>1</sup>Although Ingram et al. (1998) suggested that vulnerability levels need not be immutable, they do conceptualize them as stable.

Although a discussion of possible mechanisms giving rise to change in cognitive vulnerability to depression is beyond the scope of this paper, it is useful to highlight a few candidates. In this regard, we (Dykman & Abramson, 1990; Metalsky & Abramson, 1981) have derived the prediction from Kelley's (1967) theory of causal attribution that if a person is confronted with repeated occurrences of negative life events in a wide variety of life areas that do not happen to other people, the person should develop a more negative attributional style over time, and, hence, increased cognitive vulnerability to depression. In Kelley's theory, this would represent a situation of repeated high consistency, low distinctiveness, low consensus information that should lead to internal, stable, global attributions for negative events—the hypothesized depressogenic attributional vulnerability. Coyne and Whiffen (1995) similarly argued that people's current social contexts may exert a powerful influence on their cognitive vulnerability to depression.

Other investigators have suggested that severe mood changes (mood congruence effect; e.g., Blaney, 1986) or a history of depressive episodes (scar hypothesis; Zeiss & Lewinsohn, 1988) also may worsen cognitive patterns hypothesized to confer vulnerability to depression. According to the mood congruence hypothesis (Blaney, 1986), cognitive processes may be biased in the direction of current mood state. Although transient changes in cognition due to mood state have been demonstrated (see Blaney, 1986, for a review), it is not known whether or not cognitive patterns remain more negative once severe depressive mood states remit. According to the scar hypothesis (Zeiss & Lewinsohn, 1988; Rohde, Lewinsohn, & Seeley, 1990), individuals may be changed permanently as a consequence of a depressive episode. Recently, Steinberg, Alloy, and Abramson (1998) reported preliminary results that college students did not show worsening of cognitive vulnerability to depression following episodes of major and minor depression. Additional studies are needed that follow initially nondepressed individuals through a depressive episode and remission to determine if increased negativity in cognitions is a "scar" left by depression. Moreover, it will be important to determine if any increases in negativity of cognitions following an episode of depression actually do increase vulnerability to subsequent recurrences of depression.

Finally, treatment may lessen cognitive vulnerability. Indeed, Beck et al. (1979) suggested that a goal for the later sessions of a course of cognitive therapy should be reduction of cognitive patterns that provide vulnerability for depression. Moreover, such cognitive style changes may not be limited to individuals treated with cognitive therapy, as has sometimes been assumed. Other types of psychotherapy not represented as cognitive therapy (e.g., milieu therapy, supportive therapy, psychodynamic therapy, group therapy, behavior therapy) often include therapeutic elements geared at altering cognitions. For example, an important aspect of milieu therapy involves motivating patients to speak positively of themselves and others, and often in supportive therapy, therapists encourage patients to conceptualize themselves as more competent and worthy than they had done previously. Additionally, many of the published remitted depression studies included samples of remitted depressives who were treated with antidepressants while in episode, or who were currently on antidepressants at the time of the remission assessment of cognitive patterns, or both. Although the immediate and long-term effects of pharmacotherapy on cognitions and cognitive styles is uncertain (Simons, Garfield, & Murphy, 1984), some therapy outcome studies have reported changes in hypothesized cognitive vulnerability factors associated with remission following tricyclic antidepressant treatment equal to that after cognitive

therapy (e.g., Rehm, Kaslow, & Rabin, 1987; Rush, Beck, Kovacs, Weissenburger, & Hollon, 1982; Simons et al., 1984; Zeiss, Lewinsohn, & Munoz, 1979). In contrast, DeRubeis and Hollon (1995) reported that following treatment, depressed patients treated successfully with cognitive therapy showed improvements in attributional style, whereas patients treated successfully with pharmacotherapy did not.

Second, even if psychotherapy or pharmacotherapy does not fundamentally change depressives' cognitive styles, such treatment could deactivate maladaptive cognitive patterns or otherwise make such patterns less accessible in memory (Blaney, 1986; Miranda & Persons, 1988; Persons & Miranda, 1992; Teasdale, 1983). For example, when a depressed patient's mood improves (a requirement for remission status), he or she may be less able to retrieve dysfunctional cognitions (see Persons & Miranda, 1992 for further discussion of the mood state dependency hypothesis). A third way in which treatment could have an impact is by reducing the likelihood that treated patients would report negative cognitive styles, even if they still possess such styles (Dykman, 1992). This effect could occur due to demand characteristics such as the patient's desire to appear well in order to be released from the hospital or to gain the therapist's approval. In sum, remitted depressives who have been treated may no longer exhibit negative cognitive patterns because treatment has either remediated, deactivated, or reduced the likelihood of reporting such patterns. Insofar as the great majority of remitted depression studies examining hypothesized cognitive vulnerabilities have utilized inpatient or outpatient samples which received some form of treatment (e.g., Blackburn, Jones, & Lewin, 1986; Hamilton & Abramson, 1983; Hollon, Kendall, & Lumry, 1986), the implications of these studies for the vulnerability hypotheses of the cognitive theories of depression are unclear.

A final issue is the conceptualization and operationalization of stability of cognitive vulnerability. Drawing on work by Santor, Bagby, and Joffe (1997), Zuroff, Blatt, Sanislow, Bondi, & Pilkonis, (1999) suggested that researchers can use analysis of variance (ANOVA) to assess changes in mean scores on measures of cognitive vulnerability, or they can examine relative stability (stability in people's relative standing on a trait) with correlational techniques. Using data from the National Institute of Mental Health Treatment of Depression Collaborative Research Program, Zuroff et al. (1999) reported that this sample displayed decreases in DAS scores following treatment, consistent with the majority of remitted depression studies. However, of great interest, test-retest correlations showed that DAS scores displayed considerable relative stability in this sample. Thus, despite decreases in absolute levels of cognitive vulnerability following treatment, remitted depressives maintained their relative standing on measures of cognitive vulnerability over the treatment period. Cognitive vulnerability showed both state and trait properties.

### ***Backward Logic***

To date, all published remitted depression studies have used a logically "backward" participant selection strategy in which participants are selected on the basis of what would be defined as the "dependent variable" in the cognitive theories (i.e., depression) and then compared on what would be considered the "independent variable" in these theories (i.e., cognitive vulnerability), rather than the reverse. That is, in cross-sectional remitted depression studies, participants are selected on the basis of the presence versus absence of past depression. In longitudinal remitted depression studies, participants are selected on the basis of the presence versus absence of current de-

pression and, then, the depressed participants are followed until their symptoms remit. The remitted depressed and nondepressed groups are then compared on the hypothesized cognitive vulnerability factors. This backward participant selection strategy leads to two major problems, given the causal relations postulated in the cognitive theories of depression.

The first problem is heterogeneity of cognitive vulnerability among the remitted depressed group (see also Bootzin & McKnight, 1998; Ingram et al., 1998). The notion that the phenomenon commonly referred to as “depression” may in fact be a heterogeneous group of disorders has been maintained since the early 20th century (Depue & Monroe, 1978; Gillespie, 1929; Kendell, 1968; Kraepelin, 1913). Recall that both the hopelessness theory and Beck’s theory explicitly recognize the likely etiologic heterogeneity of depression by postulating that cognitive vulnerability may contribute only to particular subtypes of depression (hopelessness depression in the hopelessness theory and “reactive” depression in Beck’s theory) rather than all depressions. It is likely that among a group of depressives, only a subset would exhibit the hypothesized subtypes mediated by cognitive vulnerability (i.e., “negative cognition depression”).<sup>2</sup> Thus, it may be only this subset of the selected depressed group who, when their depression remits, would continue to exhibit negative cognitive styles.

Despite the theoretical emphasis on a negative cognition subtype of depression in the cognitive vulnerability models, most remitted depression studies select their remitted depression sample on the basis of the presence of a past depressive episode as defined by Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978), *DSM* criteria, and/or the presence in the past of a minimum number of general depressive symptoms as measured by self-report instruments (e.g., the Beck Depression Inventory (BDI); Beck et al., 1967). Such a selection process treats depression as a unitary phenomenon and may obscure the persistence of negative cognitive patterns in a subgroup of depressives (those with negative cognition depression).

Consistent with this idea, three remitted depression studies provide some evidence for the possible existence of a subgroup of depressives whose negative cognitive patterns persist following remission of depressive symptoms. In a longitudinal study of an inpatient sample, Hamilton and Abramson (1983) found that half of their participants who were depressed at admission to the hospital exhibited cognitive styles similar to those of their normal control participants, while the other half of their depressed sample exhibited very negative cognitive styles, well out of the range of those shown by the normal controls. Those depressed patients who exhibited very negative styles did not differ from the depressed patients who evidenced more normal styles in terms of severity of depressive symptoms at admission. Thus, the presence or absence of the hypothesized depressogenic cognitive patterns did not merely reflect the severity of the depressive syndrome. This finding suggests that the presence of depressive mood and symptoms is not sufficient to cause a person to exhibit negative cognitive patterns and that cognitive styles may be important in subtyping individuals with depressive syndromes. In addition, Hamilton and Abramson (1983) found that part of

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<sup>2</sup>A note on terms is in order. Because we (Abramson & Alloy, 1990) believe that hopelessness depression and the hypothesized subtype of depression implied in Beck’s model (what Beck [1987] termed “reactive” depression) are similar, overlapping, and, for the most part, refer to the same group of individuals, we use the phrase “negative cognition depression” to refer to the cognitively-mediated subtype of depression postulated jointly by hopelessness (Abramson et al., 1989) and Beck’s (1967, 1987) theories.

their subgroup of depressed patients with very negative cognitive styles continued to show dysfunctional attitudes and depressogenic attributional styles following remission of their symptoms.

Miller and Norman (1986) also were able to divide their sample of depressed inpatients at admission to the hospital into a group with negative cognitive biases and a group with more normal scores on the Cognitive Bias Questionnaire (Krantz & Hammen, 1979). Miller and Norman (1986) found that the subgroup of depressives with the negative cognitive biases at admission continued to demonstrate these maladaptive patterns after clinical improvement.

Finally, Ilardi and Craighead (1999) examined the relationship between personality dysfunction and cognitive vulnerability to depression (dysfunctional attitudes and negative attributional style) in a follow-up study of formerly depressed inpatients. Results indicated that Axis II personality dysfunction was related to cognitive vulnerability even after controlling statistically for subsyndromal depressive symptoms on the BDI. Based on these findings, Ilardi and Craighead speculated that previously depressed individuals with marked personality dysfunction may exhibit negative cognitive styles in truly traitlike fashion and thereby may be vulnerable to subsequent recurrences of depression.

These three studies suggest that some depressives (presumably those with negative cognition depression) may show persistence of dysfunctional cognitive patterns beyond remission of symptoms. However, it is important to note that these studies, as well as the other remitted depression studies, did not directly test the vulnerability hypotheses of Beck's theory and the hopelessness theory because cognitive styles were initially assessed while subjects were in episode rather than prior to the onset of the depressive episode. This issue is examined further in the section below entitled *Scar Hypothesis*.

The second problem resulting from the backward subject selection strategy is possible heterogeneity of cognitive vulnerability among the nondepressed comparison group. Insofar as the hopelessness and Beck's theories are cognitive vulnerability-stress theories, a significant portion of nondepressed participants (and even, never-depressed participants) may exhibit cognitive vulnerability, but not yet have experienced a depressive episode because they have not encountered the relevant or activating negative life events.

Thus, given that the typical participant selection strategy in remitted depression studies fails to take into account the subtype and cognitive vulnerability-stress components of the cognitive theories, it's easy to see how a heterogeneous group of remitted depressives (some of whom previously exhibited a negative cognition depression and some of whom did not) could fail to differ from a heterogeneous group of nondepressed controls (some of whom are vulnerable to negative cognition depression and some of whom are not) on cognitive vulnerability even if the cognitive vulnerability hypotheses are correct.

### ***Activation or "Priming" of Cognitive Styles***

***Stress activation.*** In Beck's cognitive theory of depression, dysfunctional attitudes and negative self-schemata are characterized as latent until activated (Beck et al., 1979). The primary way in which these dysfunctional cognitive patterns become activated is through the occurrence of a life event that is relevant to the content embodied in the attitudes and self-schemata. For example, a person who maintains the dysfunctional

attitude, "I am nothing if a person I love doesn't love me" has a high probability of becoming depressed after encountering a negative life event of sufficient magnitude and relevance to activate this attitude (e.g., the breakup of a significant relationship). If, as hypothesized in Beck's theory, dysfunctional attitudes and negative self-schemata are latent and not always easily accessible to conscious awareness, these cognitive patterns may be difficult to measure when not activated (Ingram et al., 1998). Some depression researchers (e.g., Ingram et al., 1998; Persons & Miranda, 1992; Riskind & Rholes, 1984; Segal & Dobson, 1992; Segal & Ingram, 1994; Segal & Shaw, 1986; Teasdale, 1988) have suggested that the failure to use explicit activation or priming procedures when measuring dysfunctional attitudes and negative self-schemata may explain why most remitted depression studies have not found persistence of these patterns in nonsymptomatic remitted depressives. Thus, just as researchers of physical health problems have discovered that some biological vulnerabilities to physical disorders cannot be detected easily when a person is in a baseline or resting state, dysfunctional attitudes and negative self-schemata may lie latent until activated by appropriate stimuli. On this view, adequate measurement of these hypothesized cognitive vulnerability factors requires a challenge protocol in which they are primed.

In the hopelessness theory of depression, depressogenic inferential styles are not necessarily hypothesized to be latent or to require explicit activation paradigms to measure them (Abramson & Alloy, 1992). Rather, in the hopelessness theory, the hypothesized depressogenic inferential styles are defined as propensities to make certain kinds of inferences about causes, consequences, and self when a negative life event occurs. Therefore, the context of a negative life event is necessary when measuring these inferential styles. This is why instruments developed to assess the kinds of attributional styles (e.g., the Attributional Style Questionnaire [ASQ]; Seligman, Abramson, Semmel, & von Baeyer, 1979; the Expanded Attributional Style Questionnaire [EASQ]; Peterson & Villanova, 1988) and other inferential styles (e.g., the Cognitive Style Questionnaire [CSQ]; Abramson, Metalsky, & Alloy, 1990) featured in the hopelessness theory present respondents with hypothetical events for which causal attributions and inferences about consequences and self are elicited.

Given the role negative life events may play as activators of maladaptive thinking patterns in Beck's theory or as context-setters for the occurrence of depressogenic inferences in the hopelessness theory, the presence of such stressors may be needed for negative cognitive styles to be operative and therefore, adequately measured in remitted depressed persons. However, many of the previous remitted depression studies involved hospitalized samples who were removed from the stresses in their usual environments (e.g., Bowers, 1990; Dobson & Shaw, 1987; Eaves & Rush, 1984; Fennell & Campbell, 1984; Fogarty & Hemsley, 1983; Hamilton & Abramson, 1983; Miller & Norman, 1986; Persons & Rao, 1985; Reda, Carpinello, Secchiaroli, & Blanco, 1985). In the relative absence of stressors that may serve to activate or make accessible negative cognitive patterns, hospitalized depressed patients whose symptoms remit may report cognitive styles that also appear to have normalized. Likewise, even nonhospitalized depressives' level of stressors may be low in the remitted state; indeed, reduced stress in the environment may be a major factor contributing to the remission of depressive symptoms. Thus, the failure of previous remitted depression studies to consider the role of stress as an activator of the hypothesized cognitive diatheses may leave them inadequate for testing the cognitive theories' vulnerability hypotheses.

Consistent with this line of thinking, Miranda (1992) reported a study designed to directly test the stress-activation hypothesis. Miranda examined levels of dysfunctional

beliefs and negative automatic thinking as a function of the number of stressful life events subjects experienced over the past 6 months in never-depressed people versus people who had recovered from a past episode of depression. She found that whereas subjects with no history of depression exhibited low levels of dysfunctional cognitions irrespective of the number of stressful events they experienced, subjects who had histories of depression reported increasingly greater maladaptive beliefs and automatic thoughts as the number of stressful events they experienced increased. These results are consistent with the hypothesis that negative cognitive patterns continue to be present in remitted depressed persons in latent form and may become more accessible when activated by stressful life events.

**Mood activation.** Although stressful life events are hypothesized to serve the activating or context-setting role in Beck's theory and the hopelessness theory of depression, several theorists, pursuing Beck's idea that dysfunctional attitudes and negative self-schemata are latent, have proposed that current mood state may also serve this role (Ingram, 1984; Ingram et al., 1998; Persons & Miranda, 1992; Riskind & Rholes, 1984; Segal, 1988; Segal & Ingram, 1994; Segal & Shaw, 1986; Teasdale, 1988). Based on associative network models of memory (e.g., Bower, 1981) and the role of affect in memory retrieval (e.g., Blaney, 1986), Persons and Miranda (1992) proposed that people's ability to report their cognitions is dependent on their current mood state; thus, negative cognitions may be accessible only during negative mood states. They suggest that this "mood-state hypothesis" would explain why remitted depressives, who are no longer in a depressed mood, typically do not evidence more negative cognitive styles than never-depressed persons.

Based on the mood-state hypothesis, mood priming procedures have been proposed as a technique by which negative cognitive styles can be accessed. These techniques typically involve mood-induction procedures in which participants read positive or negative statements (Velten, 1968) or listen to particular musical selections chosen specifically to influence mood (Clark, 1983). Three studies that utilized a mood-priming version of the remitted depression design reported evidence supportive of the presence of latent negative cognitive patterns in remitted depressives. Miranda and Persons (1988) found that recovered depressives demonstrated more dysfunctional attitudes than never-depressed subjects following a depressive mood induction with a modified Velten (1968) procedure. Similarly, Teasdale and Dent (1987) found that remitted depressed women showed greater evidence of a negative self-schema (as measured by recall of negative self-referent trait adjectives) than did never-depressed women following a depressive mood induction via sad music. Finally, Ingram, Bernet, and McLaughlin (1994) reported that recovered depressives showed more tracking errors on a dichotic listening task in response to both positive and negative distractor words than did never-depressed participants following a depressed mood induction via sad music and recall of sad autobiographical events. Two other remitted depression studies that used a mood-priming procedure (Blackburn & Smyth, 1985; Gotlib & Cane, 1987) did not find evidence of latent negative cognitive patterns in recovered depressives; however, there was also evidence that the mood-priming procedures did not work. Thus, the implications of these latter two studies are unclear.

Two studies that examined the mood-state hypothesis without the aid of specific priming procedures were conducted by Miranda, Persons, and Byers (1990). In Study 1, Miranda et al. found that reports of dysfunctional beliefs in a sample of depressed

psychiatric patients were correlated with spontaneous diurnal mood fluctuations. In Study 2, they obtained similar results in a sample of remitted depressives but not in a sample of never-depressed subjects, consistent with the hypothesis that dysfunctional beliefs are activated by negative mood in individuals who are vulnerable to depression. Further indirect evidence relevant to the mood-state hypothesis is reviewed in Persons and Miranda (1992).

Priming studies have played an important role in revitalizing the remitted depression paradigm. Although we find studies that utilize priming or activation procedures very exciting and worthy of further exploration, we believe that priming methodology does not fully address the problem of measuring cognitive vulnerabilities. This is because the finding of negative cognitive patterns among remitted depressives who have been primed with a depressed mood does not necessarily mean that these negative patterns were present before, and contributed to, onset of depression in these individuals. Indeed, results with mood priming may be more relevant to understanding maintenance or exacerbation, rather than onset, of depression because the negative cognitive patterns are activated subsequent to the onset of depressed mood. In addition, mood-priming findings among remitted depressives are equally compatible with the interpretation that formerly depressed persons develop negative cognitive styles as a result of their past depression (Ingram et al., 1998; see the section on the *Scar Hypothesis* below). Thus, results from priming versions of remitted depression studies suggest that formerly depressed individuals are likely to exhibit negative cognitive patterns when depressed, but are ambiguous with respect to whether these patterns are vulnerabilities instrumental in the onset of the prior depression (Abramson & Alloy, 1992). A further question is whether or not negative cognitive patterns among remitted depressives who have been primed with a depressed mood predict future episodes of depression. Of great interest, Segal, Gemar, and Williams (1999) recently reported that remitted depressed patients' cognitive response to a mood challenge did predict subsequent depressive relapse.

Moreover, controversy currently surrounds Beck's priming hypothesis because some researchers (e.g., Coyne, 1992; Coyne & Gotlib, 1983) have voiced the concern that the hypothesis of "latent" cognitive vulnerability underlying the priming paradigm may not be falsifiable. In addition, prospective (e.g., Abramson et al., in press; Alloy, Abramson, Whitehouse, et al., in press; Brown, Hammen, Craske, & Wickens, 1995) and laboratory (Dykman, 1997) studies have found that even when measured in an unprimed state, cognitive vulnerabilities still predict depression onset or increases or both (see the section on the *Behavioral high-risk paradigm* below). Future research is needed to determine which hypothesized vulnerabilities for depression actually do require priming for adequate measurement.

### ***Scar Hypothesis***

The final, and perhaps, most telling reason why remitted depression studies, with or without priming, are inadequate to test the cognitive vulnerability hypotheses of depression onset is that they use postmorbidity subjects and, thus, cannot distinguish between cognitive styles as vulnerability factors versus consequences of depression (Alloy, Abramson, Raniere, & Dyller, in press; Ingram et al., 1998). Even if most remitted depression studies did find negative cognitive styles during the remitted state, such results still would leave uncertain whether these cognitive patterns were present before the episode and contributed to its onset or, instead, developed as a result of the de-

pression, as in Lewinsohn's "scar" hypothesis (Rohde et al., 1990). According to the scar hypothesis, depression may remit but leave psychological scars such as negative cognitive patterns which were not present prior to the episode and therefore could not have caused it. Of course, subsequent behavioral high-risk studies, as described below, may show that such cognitive scars contribute to the recurrence of depression (see also Ingram et al., 1998; Segal et al., 1999). Thus, no matter what the outcome of the typically conducted remitted depression studies, whether obtaining evidence of negative cognitive styles in the remitted state or not, such studies, by themselves, do not allow inferences about the causal role of the hypothesized cognitive vulnerabilities for depression. Therefore, remitted depression studies, as typically conducted and interpreted, are not adequate designs for testing the cognitive vulnerability hypotheses.

## A REMEDY: THEORY-GUIDED BEHAVIORAL HIGH-RISK DESIGN

### *Behavioral High-Risk Paradigm*

If typical remitted depression studies are not optimal for testing the cognitive vulnerability hypotheses, how can these hypotheses be tested more adequately? A powerful strategy for testing the cognitive vulnerability hypotheses is the "behavioral high-risk design" (e.g., Depue et al., 1981). Similar to 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, in the behavioral high-risk study individuals are selected on the basis of hypothesized psychological, rather than genetic, vulnerability or invulnerability to the disorder. Thus, to test the cognitive vulnerability hypotheses of depression, one would want to select nondepressed people who were at high versus low risk for depression based on the presence versus absence of the hypothesized depressogenic cognitive styles. One would then compare these cognitively high and low risk groups on their likelihood of exhibiting depression in the future.

In contrast to remitted depression studies, studies utilizing the more logically appropriate high-risk design or variants of the high-risk design to test the cognitive theories' vulnerability hypotheses have obtained greater support for these hypotheses (See Abramson et al., in press and Alloy, Abramson, Whitehouse, et al., in press for reviews). Given that it is not the aim of this article to provide a comprehensive review and critique of these high-risk studies, we provide only one example here, our Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project (Alloy & Abramson, 1998). The CVD Project is a collaborative, two-site study that uses a prospective behavioral high-risk design to test the cognitive vulnerability and other etiological hypotheses of hopelessness and Beck's theories. We discuss the implications of each of our criticisms of remitted depression designs for the design of the CVD Project.

Recall that the first problem with the typical remitted depression study design for testing the cognitive vulnerability hypotheses of depression onset was that cognitive vulnerabilities need not necessarily be immutable over time. A critical implication of this point for the design of a high-risk study is that one cannot assume that a participant at hypothesized high risk for depression at the outset of the study will also be at high risk later in the study or that a low-risk participant will remain at low risk. Intervening life events, treatment, or changes in social support, among other factors, may lead to a change in vulnerability status. Therefore, an ideal high-risk study would include assessment of cognitive vulnerabilities multiple times throughout the longitudi-

nal follow-up period. As an example, in our CVD Project, we selected nondepressed college freshmen at high versus low cognitive risk for depression based on their inferential styles and dysfunctional attitudes. However, we measure these cognitive patterns once a year during the 5-year follow-up in order to examine any changes in these patterns and whether such changes are accompanied by corresponding changes in likelihood of developing subsequent depressions.

The second problem with the typically conducted remitted depression study concerned the potential heterogeneity of the remitted depressed and nondepressed comparison groups. From the perspective of the cognitive theories, maladaptive cognitive styles may only contribute vulnerability to a subtype of depression (e.g., hopelessness depression) but not to other forms of depression. By lumping all depressed participants together, one may obscure the relation between cognitive styles and this subtype. Moreover, some nondepressives may be cognitively vulnerable to this subtype, yet not become depressed, because they have experienced a low rate of negative life events.

The implication of the heterogeneity problem for high-risk studies is that one needs to test whether cognitive vulnerability leads to the theoretically predicted outcome in particular, namely the hypothesized symptoms of hopelessness depression, rather than depressive symptoms in general. For example, in our CVD Project, we are examining whether high and low cognitive risk participants differ in prospective onset of the hypothesized syndrome of hopelessness depression in particular as well as major or minor depression in general. Moreover, given that the cognitive theories are vulnerability-stress theories, one must test the cognitive vulnerability hypotheses in the context of assessment of negative life events. Given the causal relations specified in the theories, cognitive vulnerability should best predict future onsets of the hopelessness depression syndrome in interaction with negative life events.

The third problem was the possible need to activate the cognitive vulnerabilities with stress or negative mood primes to accurately measure them (Ingram et al., 1998). We are open to the possibility that behavioral high-risk designs also may need to select nondepressed high and low cognitive vulnerability groups based on a measurement of their cognitive styles following some type of priming procedure. However, we are not convinced that one needs to prime the cognitive vulnerabilities before measuring them because several prospective studies (e.g., Brown et al., 1995; Metalsky, Halberstadt, & Abramson, 1987; Metalsky & Joiner, 1992; Metalsky, Joiner, Hardin, & Abramson, 1993; Roberts, Gotlib, & Kassel, 1996), including our CVD Project have not used priming and still find that the cognitive vulnerabilities predict future depression onset.

The final problem with typical remitted depression studies was that they used post-morbid participants and, therefore, cannot readily distinguish between cognitive styles as vulnerabilities or consequences of the past depression. The implication of this problem for the design of high-risk studies is that a preferred strategy is to select initially nondepressed participants who vary in cognitive vulnerability and then examine the likelihood of depression onset, particularly the cognitive or hopelessness subtype, in a prospective fashion. By using initially nondepressed participants, there are fewer alternative interpretations of findings than if initially depressed participants who varied on cognitive vulnerability were used.

A further issue is whether high-risk designs should use currently nondepressed or, even more stringently, never-depressed participants. Never-depressed participants provide perhaps the cleanest test of the cognitive vulnerability hypotheses of initial de-

pression onset, but may not be informative about vulnerability to depression recurrence. Moreover, by selecting participants who are high on hypothesized cognitive vulnerability but have no prior history of depression, one might be left with an unrepresentative high-risk group whose members, despite being vulnerable to depression, have been protected by unknown factors from actually becoming depressed. This would lead to an unfair, overly conservative test of the cognitive vulnerability hypotheses. Whether to select out or control for past depression in high-risk studies is a very thorny question. Meehl (1971) wrestled with a similar problem in his discussion of appropriate participant selection for schizophrenia research. There are no easy solutions. In our CVD Project, we are testing the cognitive vulnerability hypotheses both with never-depressed participants as well as with participants who are currently nondepressed at the outset of the study but who have a history of depression. We then can see if converging evidence is obtained for predictions about initial (using never-depressed participants) and recurrent (using nondepressed participants with a history of depression) episodes of depression.

At this point, it is useful to note that in contrast to results obtained with the typical remitted depression studies, results from the CVD Project behavioral high-risk design provided strong support for the cognitive vulnerability hypotheses for both first onsets and recurrences of depression, including the hypothesized subtype of hopelessness depression (see Abramson et al., in press and Alloy, Abramson, Whitehouse, et al., in press for a more complete description of these findings). These results are exciting because they provide the first demonstration that negative cognitive styles, or for that matter, any hypothesized psychological vulnerability factor, indeed appear to confer vulnerability to clinically significant depressive episodes. In further analyses, we will test the vulnerability-stress prediction of the cognitive theories.

## CONCLUSION

In summary, we have argued that remitted depression studies, as typically conducted and interpreted, are not adequate for testing the cognitive vulnerability hypotheses of depression onset for four reasons: (1) remitted depression studies are based on the erroneous assumption that cognitive vulnerability should be an immutable trait; (2) remitted depression studies use a logically “backward” subject selection strategy in which subjects are selected on the basis of the “dependent” variable (depression) and then compared on the “independent” variable (cognitive vulnerability) which is likely to result in heterogeneity of cognitive vulnerability among both the remitted depressed as well as the nondepressed groups given the causal relations specified in the cognitive theories of depression; (3) many remitted depression studies have ignored the possible activating role of stress in the cognitive vulnerability-stress theories, particularly Beck’s theory, and thus, may attempt to assess cognitive vulnerability at a time when it is not operative (i.e., priming hypothesis); and (4) remitted depression studies inappropriately use postmorbidity participants to test causal hypotheses, and therefore, are ambiguous about whether negative cognitive styles observed in formerly depressed persons are vulnerabilities that contributed to the onset of their prior depressive episode as opposed to consequences of that depressive episode (i.e., scar hypothesis). Moreover, we have advocated the use of a behavioral high-risk design that is carefully guided by theory as a better strategy for testing these hypotheses. We do not wish to suggest that remitted depression studies are worthless or uninformative

with respect to cognitive processes in depression. Indeed, remitted depression designs, particularly those involving priming of negative cognitive patterns, may be one of the research strategies of choice for testing certain hypotheses, such as the long-term cognitive functioning of depressed people (Ingram et al., 1998). Moreover, one can even imagine some variants of cognitive vulnerability hypotheses that would be powerfully tested with the typical remitted design. For example, if there were a cognitive theory of depression that proposed a low base-rate, immutable, traitlike cognitive vulnerability that did not interact with stress and was a necessary cause of depression, the typical remitted depression design would provide an excellent test of this hypothetical theory. In conclusion, then, our research designs must be true to our theories, and the more precisely we can specify our theories, the more powerfully we can design studies to test them.

*Acknowledgment*—Preparation of this article was supported by NIMH grants MH43866 to Lyn Y. Abramson and MH48216 to Lauren B. Alloy. Please note that all authors contributed equally to this manuscript.

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