Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and cognitive diatheses in unipolar depression

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Abstract

Cognitive vulnerability is a central concept in cognitive theories of unipolar depression. This idea suggests that negative cognitive factors emerge during stressful situations, and that this cognitive reactivity is critical for the onset, relapse, and recurrence of depression. The number of empirical investigations that model the diathesis-stress nature of cognitive reactivity has substantially increased within the last decade. This review examines this literature, with a focus on priming and longitudinal designs. Extant research supports the concept of cognitive vulnerability to depression among adults, and support is accruing for the validity of this concept among children. Research that examines direct links between cognitive vulnerability and depression onset, relapse, and recurrence and the attachment origins of cognitive vulnerability is also accruing, although at a slower pace.

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Empirical examination of the key components of cognitive theories of depression shows widespread consensus that cognition is an important factor in unipolar depression (Haaga, Dyck, & Ernst, 1991; Ingram, Miranda, & Segal, 1998). Among the concepts that are most central to such cognitive theories is the idea of cognitive vulnerability; an idea that is firmly rooted within diathesis-stress perspectives. In its

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broadest sense, this idea suggests that negative cognitive factors emerge during stressful situations. This cognitive reactivity uniquely characterizes individuals who are vulnerable to depression and brings about the processes linked to the onset, relapse, and recurrence of depression (Beck, 1967).

Although research on cognitive processes in depression is voluminous (Ingram et al., 1998), this fundamental position of cognitive models was in large part unexamined until research modeling the diathesis-stress paradigm appeared in the late 1980s. Prior to this period, depression research tended to assess cognitive processes in isolation from the presumed activating features of stress. In so doing, data eventually demonstrated that the ostensibly stable processes featured by cognitive models could not be detected once the depressive episode remitted, which led to claims that these features were correlates or consequences of depression (e.g., Coyne, 1992). The cognitive reactivity data that began appearing in the 1980s, however, suggested the stability of these processes, and has since grown steadily.

The last comprehensive review of literature addressing stress and vulnerability to depression from a cognitive reactivity appeared in 1994 (Segal & Ingram, 1994). In particular, to examine vulnerability, Segal and Ingram (1994) focused on priming studies intended to model the parameters of cognitive diathesis-stress constructs. Although the available data at the time were limited, their review suggested promise for this approach for providing important data on cognitive vulnerability. In the intervening decade since this review appeared, additional empirical data have been reported pertaining to cognitive reactivity that have begun to clarify some matters, and that have also given rise to new issues and questions. Indeed, the body of published data has more than doubled since this earlier review, and has begun to address questions regarding the link between cognitive reactivity and depression relapse, as well as extending findings to provide clues about some of the possible origins of these processes.

Given the substantial body of data that has emerged since the last review of this literature, the goal of this paper is to review current knowledge pertaining to cognitive vulnerability from the cognitive-reactivity/diathesis-stress perspective. To place data on cognitive vulnerability into theoretical context, we begin with a brief review of conceptual issues related to cognitive reactivity constructs and perspectives. We then review studies that empirically examine the concept of a cognitive diathesis-stress vulnerability to depression with a focus on priming designs in which attempts are made to activate the proposed diathesis. We also evaluate longitudinal research that examines the interaction of cognitive reactivity with naturally occurring stressors. We conclude with a discussion of some outstanding issues in the area of cognitive vulnerability to depression, focusing on the relationship of cognitive vulnerability to depression onset versus relapse and recurrence, and on the origins of cognitive vulnerability.

Before examining these issues, it is important to emphasize that although the focus of the current review is on the reactivity perspectives of cognitive depression models, this does not suggest that this is the only approach to cognition and vulnerability. For example, the Temple-Wisconsin Cognitive Vulnerability to Depression Project (Alloy & Abramson, 1999) examines cognitive risk, but does so from a conceptual framework that differs from cognitive reactivity hypotheses. Additionally, theoretical frameworks that focus on ideas of autonomy and sociotropy (e.g., Robins, Bagby, Rector, Lynch, & Kennedy, 1997) or dependency and self-criticism (e.g., Blatt & Zuroff, 1992) also address diathesis-stress perspectives on depression (see Zuroff, Mongrain, & Santor, 2004), but do so from the standpoint of personality variables rather than cognitive reactivity.
1. Cognitive reactivity and vulnerability to depression

Several cognitive theories, each focusing in somewhat different units of analysis, have addressed the problem of unipolar depression (Ingram et al., 1998). Although sharing a common set of assumptions, cognitive theories also vary somewhat in their statements about the nature and function of cognitive vulnerability. Among these various approaches, the premier cognitive model of depression was proposed by Beck nearly four decades ago (i.e., Beck, 1967), and most cognitive theories of depression can trace a substantial part of their ancestry to Beck’s proposals. Because Beck’s model originated the ideas about this process, forms the foundation of most current ideas about cognitive vulnerability, and is currently a viable model, Beck’s proposals serve as the guiding framework for the current review. In brief, Beck’s theory of depression (1963, 1967, 1987; Kovacs & Beck, 1978) emphasizes cognitive structures as the critical elements in the development, maintenance, and relapse/recurrence of depression. These cognitive structures, or schemas, are conceptualized as negatively toned representations of self-referent knowledge and information that guide appraisal and interact with information to influence selective attention, memory search, and cognitions (Segal, 1988; Williams, Watts, MacLeod, & Mathews, 1997).

The content and diathesis-stress nature of these schemas are hypothesized to develop from interactions that occur during early development (Beck, 1967, 1987; Kovacs & Beck, 1978). Thus, for example, if childhood experiences are characterized by chronic negativity, abuse, or stress, schemas may develop that guide attention to negative rather than positive events, that lead to the enhanced recall of negative experiences, and possibly distort information to fit the schema (Ingram et al., 1998). Although all persons possess schemas that develop from life experiences and guide information processing, the schemas of vulnerable individuals are considered dysfunctional in that they embody a constellation of dysfunctional attitudes that lead to negative perspectives about oneself, the world, and the future. Moreover, these schemas are also thought to be rigid and unrealistically pessimistic (Beck, 1967, 1987; Kovacs & Beck, 1978).

Although deemed necessary for the development of depression, the mere presence of a negative self-schema is not sufficient to precipitate depression. An original and fundamental aspect of Beck’s model is a description of depressive schemas that lay dormant until activated by relevant stimuli; “Whether he will ever become depressed depends on whether the necessary conditions are present at a given time to activate the depressive constellation” (emphasis added; Beck, 1967, p. 278). This idea itself, however, had become dormant in many of the early empirical investigations of the role and functioning of cognition in depression. That is, most of the original cognitive work on schemas did not experimentally take into account the activating feature of these proposals. More recently, a variety of investigators have revived and elaborated upon this idea (Ingram et al., 1998; Miranda & Persons, 1988; Segal & Ingram, 1994) and have re-articulated the idea that, once activated in vulnerable individuals, schemas provide access to a complex system of negative personal themes that give rise to a corresponding pattern of negative information processing that eventuates in depression (Ingram, 1984; Kovacs & Beck, 1978; Segal & Shaw, 1986; Teasdale, 1988). For example, Ingram et al. (1998) argued that the “cognitive response to negative events is the activation of a schema containing themes of inadequacy and tendency toward self-blame. Cognitively interpreting life stress or negative events in terms of one’s own inadequacies and inferiority thus turns a ‘normal’ negative affective state into depression” (p. 241).
Beck (1967; Kovacs & Beck, 1978) originally suggested that, in a broad sense, schemas become activated when stressful events occur that resemble the type of events that originally led to the creation of the schema. Segal and Ingram (1994) refined and further elaborated two specific ways that activation can occur. First, as suggested by Beck, activation can occur when a stimulus corresponds to the content of existing negative schemas. In this case, referred to as direct activation, the stimulus corresponds to something of relevance to the person by virtue of resembling the information represented in the schema (e.g., an interpersonal loss). This stimulus provides enough energy to exceed the activation threshold level required for the schema to become fully active and perform its functions. Second, indirect activation occurs when a schema is set in motion through its relationships to other more fully activated schemas. In essence, schemas are linked to various degrees with one another based on similarity of content. When a schema becomes fully activated, activation spreads to associated schemas. If the link between the activated schema and an associated schema is sufficiently strong, the associated schema may become fully activated as well. If the link is weak, the associated schema may become partially activated, but should not exceed the threshold required for full activation in a manner that would guide information processing (see Bower, 1981; Ingram, 1984).

2. Empirical evaluation of the cognitive reactivity diathesis-stress concept

The earliest tests of ideas of presumed cognitive vulnerability mechanisms of depression relied on contrasting depressed versus nondepressed control groups on a theory-specified cognitive variable (e.g., dysfunctional attitudes as assessed by the Dysfunctional Attitudes Scale). The implication of finding that depressed individuals evidence more negative cognition than nondepressed controls was that the particular cognitive variables reflected causal or vulnerability aspects of depression. Although some exceptions are possible, this approach has been largely discredited as it applies to vulnerability (Ingram et al., 1998; Ingram & Siegle, 2002). The main reason for this is reflected in a second generation of studies examining individuals whose depression was in remission. Sometimes these studies compared individuals with remitted depression to currently depressed and nondepressed groups, but more frequently they examined cognitive variables in individuals in episode and then again in remission (e.g., Gotlib & Cane, 1987). The assumption underlying this research is that, to be causal, cognitive variables must be stable in and out of episode. In general, however, these studies found it is difficult to detect negative cognitive variables reflective of the depressive schema at both time points, leading to the suggestion that these cognitive factors were mere correlates or consequences of the depressed state rather than causally-related processes (Barnett & Gotlib, 1988). It is important to note in this regard that a small body of intriguing work is developing that is finding some factors that do appear to characterize the remitted depressed state. For example, Atchley, Ilardi, and Enloe (2003) found evidence to suggest persistent affective sensitivity in the right hemisphere of individuals who had experienced a depressive episode. Other data have shown that remitted depression is characterized by uncertain self-esteem (e.g., Wenzlaff, Rude, & West, 2002). There is little doubt that these factors are important and provide potentially important clues to risk, but they are considerably different from the factors that cognitive models specifically suggest are operating in depression vulnerability.

As noted by several investigators (Gotlib & Krasnoperova, 1998; Gotlib & Neubauer, 2000; Ingram & Siegle, 2002; Segal & Ingram, 1994), remission methodologies can provide information about
vulnerability factors, but remission designs alone are poor methodological choices to test a model in which there is theoretical reason to believe that vulnerability factors may be stable, but not easily accessible. Such assumptions are inherent in cognitive diathesis-stress models of depression, in which the diathesis is only accessed under stress, or at least under conditions that resemble stress. Hence, to test such a theory, studies need to appropriately model the interaction of diatheses and stress. Investigators must therefore either find a way to assess activating features naturalistically, or simulate in the lab the kind of stress that is proposed to activate cognitive vulnerability factors (Hollon, 1992). Laboratory studies, which comprise the majority of this work, have been conducted largely through the use of priming designs.

2.1. Priming designs

Because negative cognition is proposed to recede as remission progresses, a clear implication of the cognitive diathesis-stress hypothesis is that, in the absence of schema activation, persons with and without depressive schemas will appear similar on measures of maladaptive cognitions and information processing. However, under conditions of activation, vulnerable persons should evidence maladaptive cognitions and information processing while nonvulnerable persons should not. Two methodological strategies characterize most, although not all, studies of vulnerability. The first relies on defining vulnerability in terms of depression remission. That is, most research assesses vulnerable groups by studying depressed individuals in remission. As noted, former depression is typically operationalized by following depressed individuals into remission, defined as no longer meeting diagnostic criteria for depression, or by identifying a group of individuals who met criteria for past depression, but who no longer do so. Such persons are at considerably increased risk for subsequent depressive episodes. For example, Kessler (2002) has reported that up to 80% of individuals who have experienced a clinically significant episode of depression will experience additional episodes. Cognitive theories propose that these individuals, or at least a subset of individuals, are at-risk because of cognitive reasons, specifically because they possess latent depressive schemas.

A second methodological choice in most priming studies is a reliance on the use of a negative mood priming procedure. The rationale for such primes is that they rekindle, at a mild level, the negative or sad mood state that is created by stressful life events. Thus, the assumption that guides mood primes is to recreate the affective consequences of life events of the sort that are linked to the onset of depression. It is important to note, however, that although mood primes are most frequently used, they are not universal in priming research. For example, following ideas that self-focused states can initiate or contribute to depression (Ingram, 1990; Smith & Greenberg, 1981), some studies have attempted to heighten self-focus as an activating agent (e.g., Hedlund & Rude, 1995). In principle, any activating feature proposed by a cognitive diathesis-stress model could be empirically examined (Ingram et al., 1998). To illustrate one possibility, to the extent that negative interpersonal interactions are hypothesized to initiate depression (see, e.g., Joiner & Coyne, 1999), modeling such interactions in the laboratory would fulfill the criterion of modeling the interaction between diatheses and stress and would thus represent an appropriate test of such hypotheses.

Studies evaluating the cognitive diathesis-stress hypothesis using priming designs are summarized in Table 1. Generally speaking, priming research views depression vulnerability, and the putative cognitive
<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Cognitive measure(s)</th>
<th>Prime</th>
<th>Findings</th>
</tr>
</thead>
</table>
| Teasdale and Dent (1987)    | 32 RD and 21 ND community women                                              | Self-rating questionnaire; Free recall task  | Sad music                              | Before prime: Rating self-descriptiveness of words: RD = ND for non-depressive words 
                                                                                                                                                    RD > ND for depressive words 
                                                                                                                                                    Free recall: RD < ND for positive words 
                                                                                                                                                    RD = ND for negative words 
                                                                                                                                                    After prime: Rating self-descriptiveness of words: RD = ND for non-depressive words 
                                                                                                                                                    RD = ND for depressive words 
                                                                                                                                                    Free recall: RD < ND for positive words 
                                                                                                                                                    RD = ND for negative words 
                                                                                                                                                    (p = .05)                                                                                                                                   |
| Miranda and Persons (1988)  | 30 FD and 13 ND community women                                              | DAS                                          | Naturally occurring mood               | Mood x depression history related to DAS                                                                                                                                   Study 1: DAS covaried with mood 
                                                                                                                                                    Study 2: Mood x depression history related to DAS                                                                                           |
| Miranda et al. (1990)       | Study 1: 47 treatment-seeking depressed persons 
                          | DAS                                          | Naturally occurring mood               | Study 1: DAS covaried with mood 
                                                                                                                                                    Study 2: Mood x depression history related to DAS                                                                                           |
|                            | Study 2: 14 FD and 27 ND community members                                    | DAS                                          | Naturally occurring mood               | Study 1: DAS covaried with mood 
                                                                                                                                                    Study 2: Mood x depression history related to DAS                                                                                           |
| Ingram et al. (1994)        | 45 FD and 44 ND students                                                     | Dichotic listening task                      | Autobiographical mood induction with sad music | In unprimed condition: FD = ND for all error types 
                                                                                                                                                    In primed condition: Negative stimulus errors: FD > ND 
                                                                                                                                                    Positive stimulus errors: FD = ND 
                                                                                                                                                    Neutral stimulus errors: FD = ND                                                                                                           |
| Hedlund and Rude (1995)      | 53 community participants (CD = 20, FD = 15, and ND = 18)                   | Emotional Stroop task; SST; Incidental recall task | Self-focus induction                   | Stroop: CD = FD = ND on reaction time for positive versus negative words 
                                                                                                                                                    SST: CD > FD > ND for negatively-solved sentences 
                                                                                                                                                    Recall: FD > ND for negative words 
                                                                                                                                                    FD = ND for positive words 
                                                                                                                                                    CD > ND for negative words 
                                                                                                                                                    CD < ND for positive words 
                                                                                                                                                    CD = FD on word recall 
                                                                                                                                                    Intrusions: FD < ND for positive intrusions 
                                                                                                                                                    FD = ND for negative intrusions 
                                                                                                                                                    CD > ND for positive intrusions 
                                                                                                                                                    CD > ND for negative intrusions Group 
                                                                                                                                                    (CD, FD) × Word Valence p = .06                                                                                                               |
<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Measures and Induction</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roberts and Kassel (1996)</td>
<td>88 RDys and 74 NDys college students</td>
<td>Automatic Thoughts Questionnaire; Automatic Thoughts Questionnaire-Positive; DAS; Rosenberg Self-Esteem Scale</td>
<td>Naturally occurring affect - Larger correlations between RDys negative affect and all cognitive variables than between NDys negative affect and cognitive variables, - No group differences in relationships between positive affect and cognitive variables</td>
</tr>
<tr>
<td>Dykman (1997)</td>
<td>60 RD and 60 ND college students</td>
<td>DAS</td>
<td>RD = ND on DAS, regardless of priming</td>
</tr>
<tr>
<td>Solomon et al. (1998)</td>
<td>25 RD and 25 ND community individuals</td>
<td>BS; ATSS</td>
<td>Before prime: FD = ND on DAS Depression history × mood n.s. After prime: FD &gt; ND on DAS (p = .08) Depression history × mood change related to increased DAS change score for FD and decreased DAS change score for ND BS related to mood for RD but not ND RD = ND on ATSS</td>
</tr>
<tr>
<td>Brosse et al. (1999)</td>
<td>30 FD and 33 ND college students</td>
<td>DAS</td>
<td>Before prime: FD = ND on DAS (when controlling for depressive symptoms) FD &gt; ND on DAS (when controlling sadness), Sadness × Depression history n.s. for DAS, After prime: FD = ND on change in DAS Change in Sadness × Depression history n.s. for change in DAS</td>
</tr>
<tr>
<td>Segal et al. (1999)</td>
<td>54 persons treated to remission with either cognitive behavioral therapy or pharmacotherapy</td>
<td>DAS</td>
<td>Cognitive reactivity to the DAS predicted relapse status approximately two years later</td>
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</tbody>
</table>

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### Table 1 (continued)

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Cognitive measure(s)</th>
<th>Prime</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Taylor and Ingram (1999)</strong></td>
<td>40 children of CD mothers (High risk) and 46 children of ND mothers (Low risk)</td>
<td>Self-referent encoding task</td>
<td>Autobiographical mood induction</td>
<td>Rating as self-descriptive: High risk children in negative mood rated fewer positive words as self-descriptive compared to high risk children in neutral mood and low risk children in either condition, Low risk children in neutral mood rated fewer negative words as self-descriptive compared to all other groups, Recall: High risk = low risk for positive words, High risk in negative mood &gt; high risk in a neutral mood for negative words</td>
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<tr>
<td><strong>Ingram and Ritter (2000)</strong></td>
<td>35 FD and 38 ND college students</td>
<td>Dichotic listening task</td>
<td>Autobiographical mood induction with sad music</td>
<td>Following control induction: FD = ND for all error types, Following sad mood induction: FD &gt; ND for negative errors, FD = ND for positive errors, FD = ND for neutral errors, For ND, parenting unrelated to error rates in both conditions, For FD, decreased maternal care related to increased negative errors in sad mood condition; no other relationships between parenting and errors for FD</td>
</tr>
<tr>
<td><strong>McCabe et al. (2000)</strong></td>
<td>40 FD and 40 ND married women</td>
<td>Attention deployment task</td>
<td>Sad music</td>
<td>Neutral condition: FD, ND avoid negative trait words, FD, ND avoid negative state words, Sad condition: FD unbiased in responding to trait words, ND avoid negative trait words when paired with neutral words, FD avoid negative state words when paired with positive words, ND unbiased in responding to state words</td>
</tr>
<tr>
<td>Study Authors and Year</td>
<td>Sample Description</td>
<td>Methodology</td>
<td>Findings</td>
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<td>Gemar et al. (2001)</td>
<td>23 FD persons who had previously received either cognitive behavioral therapy or pharmacotherapy, 27 ND community persons, and 32 treatment-seeking CD persons</td>
<td>IAT; DAS</td>
<td>Autobiographical mood induction with sad music</td>
<td>IAT: Mood induction did not affect “not me” judgments for FD and ND. Mood induction negatively affected “me” judgments for FD but not ND. Before mood induction, FD evaluated themselves more positively than CD. Following mood induction, FD = CD. DAS: Before and after mood induction: FD &gt; ND for DAS. Shift in DAS scores for FD but not ND following induction. Mood induction negatively affected FD but not ND.</td>
</tr>
<tr>
<td>Murray et al. (2001)</td>
<td>94 children, over half of whom had been exposed to maternal depression</td>
<td>Verbal and nonverbal expressions</td>
<td>Card game</td>
<td>When dealt a winning hand, children exposed to maternal depression did not differ from non-exposed children in their expressions. When dealt a losing hand, exposed children made more negative expressions compared to non-exposed children.</td>
</tr>
<tr>
<td>Timbremont and Braet (2004)</td>
<td>19 CD, 10 RemD, and 15 ND inpatient children</td>
<td>Self-referent encoding task with intentional recall</td>
<td>Social rejection film Rating as self-descriptive: RemD, CD &gt; ND for negative words, RemD = CD for negative words, RemD = ND, CD for positive words, CD &lt; ND for positive words, CD rated negative and positive words as self-descriptive equally often, ND, RemD rated positive words as self-descriptive more often. Free recall: RemD = ND, CD on recall of negative self-descriptive words, CD &gt; ND on recall of negative self-descriptive words, RemD, CD &lt; ND on recall of positive self-descriptive words, RemD = CD on recall of positive words, RemD, CD did not differentially recall positive and negative words; ND recalled positive words more often.</td>
<td></td>
</tr>
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</table>

ATSS = Articulated Thoughts in Simulated Situations; BS = Modified Belief Scale; CD = Currently depressed; DAS = Dysfunctional Attitudes Scale; FD = Formerly depressed; FDys = Formerly dysphoric; IAT = Implicit Association Test; ND = Never depressed; NDys = Never dysphoric; RD = Recovered depressed; RemD = Remitted depressed; RDys = Remitted dysphoric; SST = Scrambled Sentences Task.
mechanism responsible for vulnerability, as the independent variables. As noted, owing to the fact that
an episode of depression confers considerable risk for future depression, most studies operationalize
vulnerability on the basis of a previous episode. Dependent variables fall into one of two broad
categories. One category consists of scores on self-report questionnaires. For example, dysfunctional
attitudes, or their close conceptual cousin, irrational beliefs, reflect central features of several cognitive
proposals. Accordingly a number of studies have used measures of such attitudes or beliefs (e.g., the
Dysfunctional Attitudes Scale). Another category of dependent variables relies on performance-based
measures to assess information processing biases. Given these two methodological strategies, we thus
organize our review of priming research according to the kind of variable assessed, starting with self-
report measures.

3. Self-report measures of presumed cognitive vulnerability

3.1. Dysfunctional attitudes and irrational beliefs

Dysfunctional attitudes constitute an important causal element in many cognitive approaches to
depression. It is therefore of little surprise that the Dysfunctional Attitudes Scale is the most common
schema indicator in priming studies. These studies have largely used either mood inductions to activate
vulnerability or have examined reports of dysfunctional attitudes and naturally occurring sad mood.
Cognitive diathesis-stress perspectives suggest that, in these latter studies, sad mood should bear a
positive relationship with reports of dysfunctional attitudes among vulnerable individuals (e.g., formerly
depressed participants), but not among nonvulnerable people (e.g., never depressed participants).

In the first study to examine the relationship between dysfunctional attitudes and mood, Miranda and
Persons (1988) found that naturally occurring negative mood was related to the occurrence of
dysfunctional attitudes, but only in people who had a history of depression. In particular, a linear
relationship was found such that as negative mood increased, these individuals endorsed more
dysfunctional attitudes. In contrast, little evidence of a relationship between mood and dysfunctional
attitude endorsement was found in people without such a history. Similar results were reported by
Miranda, Persons, and Byers (1990) in two studies. Although not testing vulnerability, per se, in the first
study, Miranda et al. (1990) found that endorsement of dysfunctional attitudes varied with diurnal
fluctuations in mood among depressed individuals, with better mood associated with fewer dysfunc-
tional beliefs. In a second study assessing vulnerability, they again found a linear relationship between
naturally occurring sad mood and dysfunctional attitudes for formerly depressed individuals, but no
association between mood and dysfunctional attitudes in never depressed people. Using a film-based
negative mood induction procedure as a prime, Miranda, Gross, Persons, and Hahn (1998) examined
dysfunctional attitudes before and after participants watched the film. As in previous studies, increases in
negative mood were associated with increased endorsement of dysfunctional attitudes in formerly
depressed participants, while in never depressed participants the effect was in the opposite direction; that
is, nonvulnerable individuals reported decreased dysfunctional attitudes in association with increased
negative mood.

Similar results were reported by Roberts and Kassel (1996) and Solomon, Haaga, Brody, Kirk, and
Friedman (1998). For example, Roberts and Kassel (1996) found stronger relationships between
negative affect and positive and negative automatic thoughts, self-esteem, and dysfunctional attitudes for
remitted dysphoric persons than for never dysphoric persons, while no between group differences were found in relationships between cognitions and positive affect. Rather than dysfunctional attitudes, Solomon et al. (1998) examined irrational beliefs, a construct that is arguably the conceptual equivalent of dysfunctional attitudes. They likewise found a stronger relationship between negative mood and irrational beliefs among recovered depressed persons than among never depressed persons. These results are consistent with results reported by Gemar, Segal, Sagrati, and Kennedy (2001), who found that, compared to never depressed persons, formerly depressed individuals evidenced a greater change in dysfunctional beliefs following a negative mood induction.

Some studies have failed to find an association between mood primes and dysfunctional attitudes. For instance, Brosse, Craighead, and Craighead (1999) found that increased endorsement of dysfunctional attitudes following a negative mood induction was unrelated to depression history. Likewise, Dykman (1997) found that dysfunctional attitudes assessed before and after a mood induction were unrelated to depression history. Instead of a mood induction, Solomon et al. (1998) exposed participants to negative sociotropic and autonomous event scenarios. In doing so, they failed to find differences between never depressed and recovered depressed persons, although, as previously discussed, they did find a relationship between depression history, naturally occurring negative mood, and irrational beliefs.

3.2. Self-ratings

Two studies have used ratings of word self-descriptiveness as dependent variables. In the first, Teasdale and Dent (1987) examined word ratings among recovered depressed and never depressed community women before and after a mood prime. Before mood priming, the recovered depressed women rated greater numbers of depressive words as self-descriptive compared to the never depressed women. After mood priming, formerly depressed and never depressed women rated equal numbers of depressive words as self-descriptive. Thus, findings were not supportive of the cognitive diathesis-stress hypothesis. However, a more recent study with children did find some support for this hypothesis (Timbremont & Braet, 2004). Specifically, following a negative mood induction, remitted depressed children rated more negative words as self-descriptive compared to never depressed children. The ratings of remitted depressed children were similar to those of a currently depressed group. Within group analyses revealed that remitted and never depressed children rated more positive than negative words as self-descriptive, while currently depressed children rated word types as self-descriptive equally often.

3.3. Summary

It is unclear why some research has failed to find an association between dysfunctional attitudes or ratings of negative word self-descriptiveness, mood state, and vulnerability. Yet, despite the small number of failures, the bulk of available literature clearly shows such an association. Moreover, this association has been found both for naturally occurring mood states as well as for experimentally induced moods. It thus seems reasonable to conclude that endorsement of dysfunctional attitudes, and perhaps the endorsement of negative words as self-descriptive, is associated with the experience of negative mood for vulnerable individuals, and that this relationship is consistent with the predictions of cognitive models of depression.
4. Information processing biases

4.1. Information recall

Teasdale and Dent (1987) were among the first to conduct a priming study of formerly depressed individuals with an adequate mood induction and found that, following this negative mood induction, recovered depressed persons were more likely to recall negative adjectives endorsed as self-descriptive than were never depressed persons. Gilboa and Gotlib (1997) also found support for biased recall among formerly dysphoric persons following a negative mood induction. Using an incidental recall paradigm, they found that previously dysphoric persons recalled more negative words than never dysphoric persons. There were no group differences in recall of positive or neutral words. Most recently, Timbremont and Braet (2004) examined intentional recall in groups of never depressed and remitted depressed children following a negative mood induction. The never depressed children recalled more positive self-descriptive words compared to the remitted depressed children; recall of positive self-descriptors among the remitted depressed children was comparable to that of a currently depressed group. Remitted and never depressed children did not differ in their recall of negative self-descriptive words. Further analyses revealed that never depressed children recalled more positive than negative self-descriptive words, while remitted depressed children recalled each word type equally often.

Although most priming vulnerability studies rely on mood primes, as previously noted, other activating features can also be studied. Self-focused attention has been proposed to serve a variety of functions in psychopathological states. In depression, one of these functions may be to initiate access to a network of dysfunctional cognitive and affective processes that eventuate in disorder (Ingram, 1990). Following this idea, some studies have attempted to heighten self-focus as an activating agent. For instance, Hedlund and Rude (1995) examined incidental recall and intrusions of negative and positive words following a self-focus manipulation. They found that, similar to currently depressed persons, formerly depressed persons recalled more negative words and had fewer positive intrusions compared to never depressed persons. Overall, the data on recall consistently suggest recall biases that are activated in vulnerability, although the nature of these biases may differ somewhat among adults and children. The study by Hedlund and Rude (1995) also reinforces the notion that dysfunctional cognitive processes can be activated by conditions other than negative mood states.

4.2. Interpretive biases

Two studies have examined interpretive biases among formerly and never depressed persons. In addition to a recall task, Hedlund and Rude (1995) used a scrambled sentences task to assess

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1 It is important to emphasize that both vulnerable and nonvulnerable individuals in mood priming studies should experience negative mood at roughly equivalent levels. If vulnerable individuals experience more negative mood than nonvulnerable individuals, then results are likely to be confounded with group status. Fortunately for research seeking to test cognitive differences, there is no reason to believe that nonvulnerable individuals do not experience negative mood states. Indeed, cognitive models argue that it is not that some people do not experience negative mood states, but rather what individuals “cognitively do” with negative mood that differentiates vulnerable individuals from those who are not vulnerable.
information processing and found that formerly depressed persons constructed more negative sentences than never depressed persons following the self-focus manipulation. Moreover, they found that currently depressed persons constructed more negative sentences than both of the other groups. Gemar et al. (2001) examined self-evaluative bias using a negative mood induction and found that depressed individuals evidenced a negative self-evaluative bias following the induction, but that never depressed individuals did not evidence such a shift. The post-induction negative bias demonstrated by the recovered depressed group was comparable to that evidenced by a currently depressed group. Groups did not differ on judgments of whether information was not relevant to them. Although the data on interpretive biases are not yet extensive, results thus far support cognitive reactivity in vulnerable individuals.

4.3. Attention

In addition to studies examining recall and interpretive biases, several studies have examined attentional processes in never depressed and formerly depressed individuals. Ingram, Bernet, and McLaughlin (1994) used a modified dichotic listening paradigm to assess attention to positive and negative stimuli. Subjects were asked to track a story that they heard in one ear while in the other ear they heard distracter words that were neutral, negative, or positive in nature. One of the presumed functions of a schema is to draw attention to relevant information. Errors in tracking the story were thus considered a measure of the extent to which attention is diverted from the primary task to emotionally laden stimuli. No differences between individuals in a normal mood control condition were found, but when induced into a sad mood, formerly depressed individuals made more tracking errors for negative stimuli than did never depressed individuals. The number of tracking errors for the never depressed group, however, was quite similar in both the normal and sad mood conditions.

These results were replicated in a subsequent study by Ingram and Ritter (2000), who found that vulnerable, but not nonvulnerable, individuals diverted attention toward negative stimuli, but only when they were in a negative mood. McCabe, Gotlib, and Martin (2000) also examined the attentional biases of previously depressed and never depressed persons following neutral or sad mood inductions. Following a neutral mood induction, both groups tended to avoid negative state and trait words. Following a sad mood induction, the responding pattern was more variable. In support of the cognitive diathesis-stress hypothesis, the previously depressed group no longer demonstrated biased responding to trait words, while the never depressed group continued to avoid negative words to some extent. However, this pattern of results was reversed for state words, with the previously depressed group avoiding negative words and the never depressed group no longer demonstrating biased responding. Such findings suggest the importance of stimuli characteristics when examining the cognitive diathesis-stress hypothesis.

Additional findings have been reported that are contrary to the attention findings of Ingram et al. (1994), Ingram and Ritter (2000) and McCabe et al. (2000). Using an emotional Stroop paradigm, Hedlund and Rude (1995), did not find evidence of an attentional bias following a self-focus induction. Similarly, Gilboa and Gotlib (1997) did not find evidence of an attentional bias following a negative mood induction, although as previously noted they did find that previously dysphoric individuals in a negative mood state had significantly better recall of the negative Stroop stimuli than did never dysphoric participants.
4.4. Summary

The weight of available data consistently show priming effects in vulnerable individuals. Using self-report measures to indicate vulnerability processes, with but a small number of exceptions, data show that vulnerable individuals uniquely report more dysfunctional attitudes when they experience negative mood states. There is also some modest evidence that vulnerable children demonstrate a bias to rate negative words as self-descriptive. Studies relying on performance-based measures also support the diathesis-stress proposals of cognitive depression models, and do so using both mood and self-focus primes. Studies assessing memory processes are uniform in finding a bias for negative stimuli for vulnerable adults who have been primed, and some data (i.e., Timbremont & Braet, 2004) have found memory bias in remitted depressed children as young as 8 years old.

Studies assessing attentional functioning as a risk mechanism indicator have produced somewhat more variable results, although here again the bulk of studies support cognitive diathesis-stress perspectives. Interestingly, most of the failures to find priming effects have relied on the Stroop task, while other measures of attentional function, such as dichotic listening, have produced more reliable effects. It is unclear if these different measures of attention assess roughly the same attentional processes, which would tend to suggest that the Stroop is a less sensitive measure, or if different facets of attention are being assessed by different tasks. The latter possibility is supported by the fact that the Stroop assesses lexical or semantic interference in attention, while dichotic listening tasks are probably more “pure” measures of attention. Deployment of attention tasks such as those used by McCabe et al. (2000) also provide support for cognitive diathesis-stress perspectives, although suggesting that the mechanisms responsible may be the erosion of positive biases that characterize normal functioning individuals and that presumably allow the activation of depressotypic schemas.

Even though the number of studies examining interpretive biases is quite small in comparison to studies assessing other presumed risk mechanisms, the data that are available again support cognitive diathesis-stress models. When considering all of the various dependent measures (i.e., self-report, memory, attention, and interpretive biases), the bulk of the data clearly and consistently point to activation processes that characterize vulnerability, and do so in ways that are generally consistent with cognitive diathesis-stress models of depression. Moreover, the available data have grown considerably since the first review of this literature appeared in 1994. The restricted database at that time limited conclusions to some degree, but it seems safe to conclude at this point that there are sufficient data that are sufficiently supportive to clearly argue for the veracity of the activation processes that are central to cognitive models of depression. Assessing activation processes in laboratory settings, however, is not the only way to evaluate the diathesis-stress component of cognitive models of depression. Longitudinal research has also examined these processes.

5. Longitudinal research

Longitudinal research is a second approach to evaluating cognitive diathesis-stress hypotheses. In a typical study falling within this paradigm, individuals who are not selected beforehand for cognitive risk are examined over time to evaluate whether levels of cognitive vulnerability interact with life events to predict depression. In an ipsative diathesis-stress model (Ingram & Luxton, in press), high levels of risk combined with high levels of stress should be predictive of symptomatology whereas high levels of risk...
and low levels of stress should not be related. Although such studies are limited in that levels of cognitive vulnerability are likely to have a low base-rate in unselected samples, these designs do have several advantages compared to priming designs with remitted depressed persons, including the ability to potentially demonstrate temporal antecedence of cognitive vulnerability and the ability to examine naturally occurring stressors (Table 2).

Several studies have examined the ability of dysfunctional attitudes to predict depressive symptoms within a week following a discrete stressor such as learning a college admissions decision or the outcome of an examination. For example, Brown, Hammen, Craske, and Wickens (1995) examined whether dysfunctional attitudes interacted with discrepancies between anticipated and actual exam performance to predict depressive symptoms. As expected, dysfunctional attitudes interacted with poorer-than-expected performance to predict depressive symptoms shortly after receipt of the exam grade. Additional analyses suggested that this effect might largely be due to the interaction between poorer-than-expected performance and dysfunctional attitudes regarding achievement. Similar findings were reported by Joiner, Metalsky, Lew, and Klocek (1999) and Hankin, Abramson, Miller, and Haefeli (2004); both groups found that dysfunctional attitudes interacted with the discrepancy between acceptable and actual exam scores to predict depressive symptoms within days of receipt of the exam grade. However, a fourth study examining the interaction of dysfunctional attitudes and college admissions outcomes found only partial support for the vulnerability hypothesis. In this study, dysfunctional attitudes interacted with negative admissions outcomes to predict immediate depressed mood, but not depressed mood days later (Abela & D’Alessandro, 2002).

Another group of longitudinal studies has examined the ability of dysfunctional attitudes in interaction with a wider range of stressors to predict depression. These studies differ from those previously reviewed in that they typically use longer intervals (i.e., weeks or months rather than days) between assessment of attitudes and assessment of symptoms. One of the first studies of this type was conducted by Kwon and Oei (1992). They found that dysfunctional attitudes in interaction with negative life events predicted depressive symptoms three months later. Dykman and Johll (1998) found that high levels of dysfunctional attitudes interacted with stress to predict depressive symptoms 14 weeks later, but only for initially nondysphoric women. Similarly, Hankin et al. (2004) examined the interaction of dysfunctional attitudes and negative life events at both 5 weeks and 2 years following initial assessment. At both time points, the interaction of dysfunctional attitudes with negative life events predicted depressive symptoms. Moreover, this interaction also predicted occurrence of depressive disorders over the 2-year follow-up interval. However, Barnett and Gotlib (1988, 1990) did not find interactions between dysfunctional attitudes and stress.

The schema-activation hypothesis has also been examined among youths, using similar designs. Using a longitudinal design, Lewinsohn, Joiner, and Rohde (2001) provided perhaps the most stringent test of the schema activation hypothesis. Using a high school sample, they examined the ability of dysfunctional attitudes in interaction with stress to predict the onset of major depressive disorder (MDD) during a 1-year interval. Analyses controlled for a number of potential depression risk factors, including sex of participant, family history of both depressive and non-depressive psychological disorders, and participants’ own histories of non-depressive disorder. Dysfunctional attitudes interacted with stress to predict the onset of MDD at a trend level, and the analysis suggested that this finding applied to both first onsets of MDD and recurrences. However, contrary to the findings of several other studies, dysfunctional attitudes did not interact with stress to predict depressive symptoms. One of these studies was reported by Shirk, Boergers, Eason, and Van Horn (1998), who focused on the transition to high
<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Cognitive measure</th>
<th>Stress measure</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barnett and Gotlib (1988)</td>
<td>57 female college students</td>
<td>DAS</td>
<td>LES</td>
<td>DAS did not interact with LES to predict depressive symptoms at 3 months</td>
</tr>
<tr>
<td>Barnett and Gotlib (1990)</td>
<td>240 college students</td>
<td>DAS</td>
<td>LES</td>
<td>For both men and women: DAS did not interact with LES to predict depressive symptoms at 3 months</td>
</tr>
<tr>
<td>Kwon and Oei (1992)</td>
<td>Study 2: 200 college students</td>
<td>DAS</td>
<td>LES</td>
<td>DAS × LES predicted depressive symptoms at 3 mos</td>
</tr>
<tr>
<td>Brown et al. (1995)</td>
<td>72 college students</td>
<td>DAS</td>
<td>Difference between expected and actual exam grade</td>
<td>DAS × difference variable predicted depressive symptoms 2–6 days after receipt of grade</td>
</tr>
<tr>
<td>Dykman and Johll (1998)</td>
<td>276 college students</td>
<td>DAS</td>
<td>NLEQ</td>
<td>Across all students, DAS did not interact with NLEQ to predict depressive symptoms 14 weeks later. DAS × NLEQ predicted depressive symptoms for initially asymptomatic, but not initially symptomatic, individuals; this relationship held only for women</td>
</tr>
<tr>
<td>Shirk et al. (1998)</td>
<td>Study 4: 91 8th graders</td>
<td>DISS</td>
<td>Questionnaire assessing school transitional stress</td>
<td>DISS × stress predicted depressive symptoms following transition to high school</td>
</tr>
<tr>
<td>Joiner et al. (1999)</td>
<td>119 college students</td>
<td>DAS</td>
<td>Difference between acceptable and actual exam grade</td>
<td>DAS × difference variable predicted depressive symptoms 2 days after grade receipt</td>
</tr>
<tr>
<td>Lewinsohn et al. (2001)</td>
<td>1507 high school students</td>
<td>DAS</td>
<td>Self-report of 11 negative events</td>
<td>DAS × life events predicted 1-year onset of Major Depressive Disorder (p &lt; 0.09). DAS × life events did not predict depressive symptoms at 1 year</td>
</tr>
<tr>
<td>Abela and D’Alessandro (2002)</td>
<td>136 high school seniors</td>
<td>DAS</td>
<td>College application outcome</td>
<td>DAS × outcome predicted depressive symptoms immediately but not four days after notification of outcome Cognitive reactivity × AEQ for negative, but not positive, words predicted depressive symptoms</td>
</tr>
<tr>
<td>Beevers and Carver (2003)</td>
<td>70 college students</td>
<td>Dot probe attentional task</td>
<td>AEQ</td>
<td>Studies 1 and 2: NLEQ, Study 3: Difference between acceptable and actual exam performance</td>
</tr>
<tr>
<td>Hankin et al. (2004)</td>
<td>Study 1: 216 college students, Study 2: 233 college students, Study 3: 110 college students</td>
<td>DAS</td>
<td>NLEQ, Study 3: Difference between acceptable and actual exam performance</td>
<td>Study 1: DAS × NLEQ predicted depressive symptoms at 5 weeks, Study 2: DAS × NLEQ predicted depressive symptoms and depressive disorder diagnoses at 2 years, Study 3: DAS × difference variable predicted depressive symptoms 5 days after receipt of exam grade</td>
</tr>
</tbody>
</table>

AEQ = Adverse Events Questionnaire; DAS = Dysfunctional Attitudes Scale; DISS = Dysphoric Interpersonal Schema Scale; LES = Life Experiences Survey; NLEQ = Negative Life Events Questionnaire.
school. These investigators found that maladaptive interpersonal schemas assessed before the transition interacted with interpersonal stress to predict depressive symptoms during the first weeks of high school.

Finally, Beevers and Carver (2003) extended the findings of previous longitudinal studies by using an information-processing task, the dot probe, to assess cognitive vulnerability. They examined whether cognitive reactivity interacted with negative life events to predict depressive symptoms seven weeks later. All participants were euthymic at the time of initial testing; the recruitment strategy ensured inclusion of both remitted depressed and never depressed persons. Change in attentional bias for negative, but not positive, words following a negative mood induction interacted with life events to predict depressive symptoms.

6. Conclusions regarding the cognitive diathesis-stress hypothesis

Both priming and longitudinal studies evaluating the cognitive diathesis-stress hypothesis are largely supportive of the idea that depressive schemas do not affect information-processing and cognition until activated. This finding cuts across several different levels of cognitive analysis, including cognitive operations such as attention and free recall and cognitive products such as self-reported cognitions. Moreover, effects are found when the stressor is an experimental prime, such as a sad mood or self-focus induction, or a naturally occurring stressor. Additionally, evidence supporting the cognitive reactivity hypothesis appears to cut across age groups, with children, adolescents, and adults demonstrating greater levels of depressive symptoms following activation of dysfunctional cognitions. Effects are also found when predicting both diagnoses of depressive disorders and depressive symptoms. These latter findings do not confirm the continuity of depression although they are consistent with such assumptions.

The extent of empirical data evaluating the cognitive diathesis-stress hypothesis has grown substantially in the decade since the last review of this literature was published (Segal & Ingram, 1994). The conclusions from that review were that studies accounting for activation of cognitive diatheses tended to support the vulnerability formulation of depression. These conclusions not only remain accurate, but the additional data now available even more clearly support the diathesis-stress proposals of cognitive models. This is the case despite a few studies that did not find support for these ideas; the clear bulk of data appearing since the earlier review are supportive. As evidence continues to mount in support of the basic cognitive diathesis-stress hypothesis, additional issues regarding cognitive reactivity and vulnerability have also emerged. How cognitive vulnerability may contribute to depression onset versus relapse and recurrence and how cognitive vulnerability originates are among the most pressing of these issues.

7. The contribution of cognitive vulnerability to depression onset, relapse, and recurrence

As previously noted, one outstanding issue in the area of cognitive vulnerability to depression concerns whether such vulnerability is related to depression onset, relapse, and recurrence. Indeed,

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2 One direction for future research may be to conduct a meta-analysis of the reviewed literature with the goal of identifying study-specific attributes (e.g., assessment strategies for dependent variables) that affect results.
implicit in the notion of cognitive vulnerability is the idea that such vulnerability may lead to the onset as well as to the relapse and recurrence of depression. The studies reviewed were largely conducted either cross-sectionally, comparing formerly and never depressed persons, or longitudinally, examining persons whose depression histories were unknown. Although these studies are supportive of the notion of cognitive diathesis-stress vulnerability, they do not directly examine whether presumed vulnerability actually contributes to depressive onset, relapse, recurrence, or all three. Empirical studies that directly examine these issues have been slow to emerge.

Indeed, to our knowledge only one study has directly addressed whether cognitive vulnerability is related to onset, relapse, or recurrence. In this study, Segal, Gemar, and Williams (1999) examined the ability of cognitive reactivity to predict relapse in a group of formerly depressed patients \( N = 30 \) who had been treated to remission with either cognitive behavioral therapy or antidepressants. These patients participated in a mood induction task in which changes in dysfunctional attitudes were examined in both euthymic and induced transient dysphoric moods. They were re-contacted up to 4 years later to assess relapse status. Logistic regression indicated that the magnitude of mood-linked cognitive reactivity significantly predicted relapse over this interval, with 70% of participants correctly classified as relapers or non-relapers. Thus, the one study to date that directly examines cognitive reactivity as a predictor of depressive relapse supports the idea that cognitive vulnerability increases risk for future episodes of disorder, and buttresses the idea that the various processes that have been studied (e.g., attention, interpretive biases) may in fact play a meaningful role in vulnerability.

Although research to date is supportive, the limited amount of available data suggest that there is a strong need for additional studies to examine whether cognitive vulnerability predicts depression onsets, relapses, and recurrences. For example, to our knowledge no research has examined mood reactivity processes in first onsets. There are some data that point in this direction, such as the research showing reactivity in children of depressed mothers (Murray, Woolgar, Cooper, & Hipwell, 2001; Taylor & Ingram, 1999), but such results obviously do not directly test whether reactivity is associated with later depression. No doubt such research has yet to be conducted because it is difficult and costly to undertake. Even though cognitive vulnerability research is arguably ahead of at least some paradigms that are mainly focused on describing processes that differentiate depressed from nondepressed people, vulnerability research that examines diathesis-stress processes in first onsets and replicates the functioning of these processes in relapses and recurrences is at least one of the next needed steps.

Another important step is examining the possible origins of these presumed cognitive vulnerability processes.

8. Origins of cognitive vulnerability

As supportive evidence has mounted for vulnerability ideas, a number of researchers have directed their attention to identifying the developmental origins of maladaptive schemas and the associated negative information processing that is thought to create risk (Ingram, 2003). At first glance, such attention may seem misplaced because most theoretical discussions and empirical evaluations of cognitive models are directed toward the development and course of depression in adults (see Engel & DeRubeis, 1993; Haaga et al., 1991; Ingram & Holle, 1992; Ingram, Scott, & Siegle, 1999; Sacco & Beck, 1995). Nonetheless, as suggested by Beck (1967, 1987; Kovacs & Beck, 1978), vulnerability to depression develops through the acquisition of cognitive schemas concerning stressful or traumatic
events in childhood and adolescence. Specifically, Beck (1967) suggests that when such events occur relatively early in the individual’s development, these individuals become sensitized to just these types of events. The corresponding generation of negative schemas to process information about these events leads to the subsequent activation of these schemas, and corresponding depression, if and when similar events occur in the future:

In childhood and adolescence, the depression-prone individual becomes sensitized to certain types of life situations. The traumatic situations initially responsible for embedding or reinforcing the negative attitudes that comprise the depressive constellation are the prototypes of the specific stresses that may later activate these constellations. When a person is subjected to situations reminiscent of the original traumatic experiences, he may then become depressed. The process may be likened to conditioning in which a particular response is linked to a specific stimulus; once the chain has been formed, stimuli similar to the original stimulus may evoke the conditioned response (p. 278).

Thus, while most commonly viewed as a theory of adult depression, Beck’s model specifically and centrally incorporates the idea that vulnerability to depression develops early in life. Although stressful early interactions with any number of people may be linked to the development of cognitive vulnerability to depression, both theory and data focus on interactions with attachment figures and suggest that problematic interactions with such figures may have an especially pernicious effect on schema development. We therefore now turn to a discussion of attachment relationships and their implications for the development of maladaptive schemas.

8.1. Attachment theory

Many of Beck’s ideas are paralleled by Bowlby’s (1969/1982) attachment theory. Bowlby suggested that an attachment relationship consisting of proximity-maintaining behaviors by the human infant and care-giving behaviors of his or her primary caregivers typically forms during the first year of an infant’s life. The result of this attachment is the security and protection of the infant. These attachment relationships differ from many other types of social relationships in significant ways. First, attachment relationships are thought to be enduring. Attachment relationships formed during childhood appear to persist throughout the lifespan (Bartholomew & Horowitz, 1991; Bowlby, 1980). Second, although a child may form more than one early attachment relationship, attachment figures are not interchangeable. For example, an attachment relationship with a deceased parent cannot be replaced by an attachment relationship with a surviving parent. Third, a child desires physical and emotional closeness with his or her attachment figure(s), and may become distressed upon uncontrollable separation. Finally, a child seeks security and comfort in attachment relationships; this latter component distinguishes attachment relationships from other social relationships (Ainsworth, 1989; Bowlby, 1969/1982). In sum, attachment relationships are unique and, according to attachment theorists, long-lasting parts of most people’s lives.

The longevity and uniqueness of attachment relationships suggest that they are fertile ground for the development of schemas of the type that Beck has proposed lead to cognitive vulnerability. By their very nature, attachment relationships consist of repeated interactions, often over the course of a lifetime. During the course of a healthy (or secure) early attachment relationship, a child experiences consistently
accessible and responsive care giving on the part of the attachment figure (Bowlby, 1973, 1977). Through such experiences, a child may surmise that he or she is loved and valued. During the course of an unhealthy (or insecure) attachment relationship, however, a child finds his or her caregivers to be inaccessible or unresponsive; such caregiving may include hostility and rejection directed towards a child and threats used as a means of control (Bowlby, 1973, 1977, 1980). Within the context of such relationships, a child may surmise that he or she is unlovable and unwanted. Moreover, the messages provided in the context of these relationships may be especially meaningful in that they are coming from someone who is uniquely valued by the child and who functions as one of only a few sources of security and comfort. Thus, the messages, whether positive or negative, are likely to be quite well-established in a child’s developing schemas.

8.2. Empirical evaluations of attachment-based origins of cognitive vulnerability

Investigation of the potential relationship between early attachment and cognitive vulnerability to depression is relatively new. Several studies (e.g., Ingram, Overbey, & Fortier, 2001; Randolph & Dykman, 1998; Whisman & Kwon, 1992; Whisman & McGarvey, 1995), while providing support for relationships between attachment experiences and dysfunctional cognitions, did not attempt to activate presumed diatheses, and thus did not adequately model cognitive vulnerability to depression as suggested by Beck et al. (Beck, 1967, 1987; Ingram et al., 1998; Kovacs & Beck, 1978; Segal & Ingram, 1994). Nonetheless, studies are also emerging that do model the hypothesized diathesis-stress nature of cognitive vulnerability.

Two studies have examined the origins of cognitive vulnerability in children at risk for depression. In both studies (Murray et al., 2001; Taylor & Ingram, 1999), children were defined as at-risk by virtue of having depressed mothers. Taylor and Ingram (1999) examined identification of positive and negative adjectives as self-descriptive and recall of self-descriptive adjectives following neutral or negative mood inductions in a group of 8- to 12-year-old nondepressed children. In support of cognitive vulnerability ideas, positive words were endorsed as self-descriptive less often by high risk children in a negative mood than by high risk children in a neutral mood or low risk children regardless of mood. Moreover, high risk children in a negative mood recalled a higher proportion of negative self-descriptors than high risk children in a neutral mood. Murray et al. (2001) found conceptually similar results in a group of 5-year-old children. These investigators examined negative and positive verbal and nonverbal expressions during a card game in which children were alternately winning and losing. When winning, both high risk and low risk children tended toward positive expressions. When losing, however, high-risk children demonstrated more negative expressions than low-risk children. Further analyses suggested that parental hostility may have accounted for some of the effects of maternal depression on high risk children’s negative expressions.

Finally, a previously discussed study by Ingram and Ritter (2000) also examined relationships between attachment experiences and cognitive vulnerability. In addition to examining information processing as a function of depression history, these authors examined processing as a function of childhood care and overprotection provided by parents. Among formerly depressed individuals in a sad mood, they found that low levels of perceived maternal care were related to increased attention to negative information. Never depressed individuals did not evidence similar relationships between early experiences and attention. No relationships were found between depression history, parenting by fathers, and attention, regardless of depression history. They interpreted these results to suggest that low levels of
maternal care may be a critical factor in the development of negative schemas that emerge during sad mood states.

Overall, the growing literature on the origins of cognitive vulnerability suggests that experiences with parents may play a pivotal role in the development of such vulnerability. Indeed, high-risk children as young as 5 years old demonstrate reactivity to negative information that, if cognitive models are correct, may predispose them to depression later on. Nonetheless, the extant body of literature leaves many questions unanswered. First, the role of attachment figures other than mothers in the development of vulnerability is unclear. Certainly, Bowlby’s attachment theory (Ainsworth, 1989; Bowlby, 1969/1982) suggests that fathers and extended family members might each play a part in schema development, although mothers are most often the focus of research examining depressive risk. More contemporary ideas have also suggested the importance of considering fathers’ roles in the creation of psychological health or disorder (i.e., Phares, Duhig, & Watkins, 2002). Additionally, the precise parenting behaviors that lead to cognitive vulnerability merit further investigation. Both Murray et al. (2001) and Ingram and Ritter (2000) provide direction in this regard, but behaviors other than hostility or lack of care may be important as well. For example, Bowlby (1980) suggests that parental rejection as well as hostility may play roles in the development of depression. Finally, maltreatment experiences, both within and outside the family, warrant investigation as possible factors in the development of cognitive vulnerability to depression (Gibb, Abramson, & Alloy, 2004). Indeed, a great deal of literature suggests that experiences of childhood maltreatment lead to depression in adulthood (Hammen & Garber, 2001). Continued attention to identifying the origins of cognitive vulnerability are thus warranted, both in terms of understanding the basic processes that give rise to vulnerability, but also for the purpose of preventive interventions (Dozois & Dobson, 2004).

9. Concluding comments

This paper serves as a review of empirical knowledge regarding cognitive vulnerability to depression from a diathesis-stress perspective. As such, we have reviewed the concept of cognitive vulnerability, using the work of Beck (1963, 1967, 1987; Kovacs & Beck, 1978) as a guiding framework, and examined empirical studies evaluating the cognitive diathesis-stress vulnerability concept, focusing on priming and longitudinal designs. What is clear from this review is that extant research largely supports the notion of a cognitive diathesis-stress vulnerability to depression. Moreover, although most of the literature to date examines vulnerability in adults, there is increasing data to suggest that the same sort of vulnerability exists in children.

Additionally, we identified two outstanding issues in the area of cognitive vulnerability that may inform depression prevention efforts: the contribution of cognitive vulnerability to depression onset, relapse, and recurrence, and the origins of cognitive vulnerability. Relevant empirical data has been slower to accrue in these two areas. Nonetheless, the extant data suggest that cognitive vulnerability may contribute to depression relapse and that vulnerability may originate in part from interactions that occur within the context of childhood attachment relationships. The next step for investigators is to utilize this knowledge and build upon this data with the goal of preventing depression onset, relapse, and recurrence across the lifespan. Indeed, given that unipolar depression currently imposes the fourth greatest burden of ill health for diseases worldwide (Murray & Lopez, 1996), prevention is sorely needed.
References


