

COGNITIVE VULNERABILITY TO UNIPOLAR AND BIPOLAR MOOD DISORDERS

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In this article, we review findings on the role of negative cognitive styles and information processing biases as cognitive vulnerabilities to unipolar and bipolar mood disorders. We first briefly describe the cognitive theories of unipolar depression and the logic of their extension to bipolar disorders, as well as methodological issues involved in conducting vulnerability research, particularly with bipolar samples. We then review the evidence for negative cognitive styles, information processing, and rumination, alone and in combination with life events, as vulnerabilities to unipolar depression and bipolar disorder, as well as the developmental antecedents of cognitive vulnerability to both forms of mood disorder. In our review, we give particular attention to the findings from the Temple–Wisconsin Cognitive Vulnerability to Depression Project and the Wisconsin–Temple Longitudinal Investigation of Bipolar Spectrum Project. Our review suggests that similar cognitive vulnerabilities may increase individuals' risk for both forms of mood disorder.

Cognitive vulnerability–stress theories of depression have been the subject of intensive investigation over the past three decades. Empirical

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studies based on these models have yielded much valuable information regarding the role of cognitive processes in the onset, maintenance, and treatment of depression. Specifically, cognitive models of depression (e.g., Abramson, Metalsky, & Alloy, 1989; Beck, 1967; 1987; Ingram, Miranda, & Segal, 1998; Nolen-Hoeksema, 1991) emphasize the role of negative inferential styles, dysfunctional beliefs, information-processing biases, and maladaptive emotion-regulation strategies (e.g., rumination) as vulnerabilities for depression when individuals experience stressful life events. Moreover, a growing body of evidence suggests that such cognitive vulnerabilities do, in fact, increase risk for depression (e.g., Abramson et al., 1999; Alloy, Abramson, Whitehouse et al., 1999, Clark, Beck, & Alford, 1999; Ingram et al., 1998).

As a result of the success of cognitive models in contributing to the understanding of the etiology, course, and treatment of unipolar depression, cognitive vulnerability-stress models recently have been extended to bipolar spectrum disorders (e.g., Alloy, Abramson et al., 2006; Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999; Hammen, Ellicott, & Gitlin, 1992; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999). In this article, we review research on the role of cognitive vulnerability to unipolar and bipolar mood disorders. We begin by presenting the cognitive models of unipolar depression and their logical extension to bipolar spectrum disorders (Bipolar I, Bipolar II, Cyclothymia). Next, we briefly discuss methodological issues in vulnerability research, particularly challenges posed by bipolar spectrum disorders. We then review studies of cognitive styles and information processing associated with and predictive of unipolar and bipolar disorders, evidence for cognitive vulnerability-stress interactions as predictors of unipolar and bipolar disorders, and developmental antecedents of cognitive vulnerability to unipolar and bipolar disorders. We note that our review is not intended to be comprehensive; rather, it focuses primarily on our own findings from the Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project (Alloy & Abramson, 1999) and the Wisconsin-Temple Longitudinal Investigation of Bipolar Spectrum (LIBS) Project (Abramson & Alloy, 2002) and related studies. Moreover, although our understanding of cognitive vulnerability to unipolar depression is relatively well developed, the exploration of cognitive vulnerability approaches to bipolar disorders is only at its beginning. We hope that future investigators will be inspired by the present review to conduct further, more sophisticated studies of cognitive vulnerability to the onset and course of bipolar disorders.

COGNITIVE THEORIES OF DEPRESSION

The cognitive theories of unipolar depression have sought to explain why some people are vulnerable to depression when confronted with negative events, whereas others suffer only mild, short-lived dysphoria. From the cognitive perspective, the interpretation people give to their life experiences influences their vulnerability to depression. In the Hopelessness theory of depression (Abramson et al., 1989), individuals who tend to attribute negative life events to stable (enduring) and global (general) causes, catastrophize about the consequences of a current negative event, and infer that the occurrence of a negative event in their lives means that they are deficient or unworthy are hypothesized to be more likely to experience depression when confronted with stressors than are individuals who don't exhibit these negative inferential styles. Individuals who exhibit such negative inferential styles should be more likely to make negative inferences about the causes, consequences, and self-implications of any negative event they encounter, thereby incrementing the likelihood of becoming hopeless, the proximal cause of the symptoms of depression (particularly, the subtype of hopelessness depression [HD]).

In Beck's (1967, 1987) model, negative self-schemata, organized around themes of failure, inadequacy, loss, and worthlessness, serve as vulnerabilities for onset and exacerbation of depression. Such negative self-schemata are often represented by dysfunctional attitudes in which the person believes that his or her happiness and worth depend on being perfect or on others' approval. Beck (1967) hypothesized that depressive self-schemata influence the perception, interpretation, and recall of personal experiences, thereby leading to a negatively biased construal of one's self, personal world, and future (hopelessness). When activated by the occurrence of negative events, depressive self-schemata lead to the onset or exacerbation of depressive symptoms by way of preferential encoding and retrieval of negative self-referent information. In Beck's (1987) theory, individual differences in two cognitive/motivational orientations serve as additional vulnerabilities for depression. People high in sociotropy place great importance on intimacy, relationships, and acceptance from others and are vulnerable to depression when they experience interpersonal rejections, losses, or disappointments. In contrast, people high in autonomy value achievement, independence, and control and are at risk for depression when they experience failures or events that impinge on their personal choice.

According to another cognitive theory of depression, the response styles theory (Nolen-Hoeksema, 1991), individuals who tend to ruminate in response to depressed mood are at greater risk for experiencing

prolonged and severe depressive episodes than are individuals who tend to distract themselves from dysphoria. Rumination can be conceptualized as an emotion-regulation strategy involving perseverative self-focus that is recursive and persistent. Nolen-Hoeksema (1991, p. 569) defined depressive rumination as "repetitively focusing on the fact that one is depressed; on one's symptoms of depression; and on the causes, meanings, and consequences of depressive symptoms." Thus, a ruminative response style is hypothesized to provide vulnerability to full-blown depressive episodes when individuals become dysphoric.

The logic of the cognitive vulnerability-stress models of unipolar depression may be extended to bipolar spectrum disorders (Alloy, Abramson et al., 2006; Alloy, Reilly-Harrington, Fresco, & Flannery-Schroeder, 2006; Alloy, Reilly-Harrington et al., 1999; Hammen et al., 1992; Newman, Leahy, Beck, Reilly-Harrington, & Gyulai, 2002; Reilly-Harrington et al., 1999). The same cognitive processes that contribute vulnerability to unipolar depressive episodes may also confer risk to the depressive episodes experienced by bipolar individuals after negative events.

With respect to vulnerability to the manic and hypomanic episodes of bipolar individuals, two types of predictions may be derived from a logical extension of the cognitive theories of unipolar depression. On the one hand, based on the hopelessness theory, individuals who exhibit positive inferential styles for positive life events (stable, global attributions for positive events, positive consequences and self-implications of positive events) should become hopeful and, in turn, develop euphoria and hypomanic/manic symptoms when they experience positive events. Similarly, Beck (1976) suggested that mania-prone individuals possess a set of positive self-schemata, containing unrealistically positive attitudes about self, world, and future. When these positive self-schemata are activated by the occurrence of positive events, these individuals develop manic symptomatology. On the other hand, given that negative life events have been found to trigger manic episodes as well as depressive episodes among bipolar individuals (Alloy, Abramson et al., 2006; Alloy, Reilly-Harrington et al., 2006; Johnson & Roberts, 1995), bipolar individuals' cognitive styles for construing negative events, rather than their styles for interpreting positive events, may be more important in affecting their vulnerability to manic and hypomanic episodes. The potential relevance of cognitive styles for interpreting negative events to mania/hypomania is consistent with psychodynamic formulations suggesting that the grandiosity of manic states is a "defense" or counterreaction to underlying depressive tendencies (Freeman, 1971; Klein, 1994). In our review of cognitive vulnerability to bipolar disorders below, there is evidence provided for both of

these alternatives. We will also address the conditions under which cognitive styles for interpreting negative events may promote risk for hypomanic/manic episodes versus depressive episodes at different times in the same individual.

METHODOLOGICAL ISSUES IN VULNERABILITY RESEARCH

From a research design perspective, a hypothesized vulnerability factor for a disorder must meet at least two criteria (Alloy, Abramson, Ranieri, & Dyller, 1999; Ingram et al., 1998): (1) it must temporally precede the initial onset of the disorder, or, in the case of a vulnerability for the course of a disorder, precede episodes or exacerbations of the disorder; and (2) it must exhibit some degree of stability independent of the symptoms of the disorder (but does not need to be immutable; see Just, Abramson, & Alloy, 2001). Some research designs are more appropriate than others for demonstrating these criteria (Alloy, Abramson, Ranieri et al., 1999). For example, cross-sectional studies that compare a group with the disorder of interest to a normal group on various characteristics can establish associations between potential vulnerabilities and the disorder. But, they are inadequate for establishing temporal precedence or stability independent of symptoms of the disorder. Designs that compare individuals who have remitted from the disorder to a normal group on the potential vulnerabilities or that longitudinally compare individuals with the disorder in symptomatic versus remitted states are an improvement because they can establish independence of the vulnerabilities from the disorder symptoms. However, such "remitted designs" cannot distinguish between the alternative possibilities that the characteristics are vulnerabilities or consequences ("scars") of the disorder (Just et al., 2001; Lewinsohn, Steinmetz, Larson, & Franklin, 1981). Ideally, prospective, longitudinal designs are needed in which the putative vulnerability is assessed prior to the initial onset of the disorder or prior to recurrent episodes of the disorder. Such prospective designs can establish both the vulnerability factor's temporal precedence and independence from symptoms, although they still cannot demonstrate a causal role for the vulnerability (Alloy, Abramson, Ranieri et al., 1999).

Cognitive vulnerability to unipolar depression has been demonstrated with the preferred prospective designs (see below). However, few studies exploring the role of cognitive vulnerabilities for bipolar disorders have used prospective designs. Indeed, as a group, the bipolar disorders pose especially difficult challenges for vulnerability research. First, these disorders are highly recurrent with significant interepisode symptomatology and impairment. Thus, it is difficult to establish independence of potential vulnerabilities from symptoms of depression and

hypomania/mania. Second, bipolar disorders have their initial onset at an early age (mean onset age of 14; Goodwin & Jamison, 1990), with childhood onsets not uncommon (Geller & DelBello, 2003). Thus, to truly establish temporal precedence for initial onset of bipolar disorder, one should assess cognitive vulnerabilities in childhood or early adolescence. No study to date has done this. Given these methodological challenges, investigations of cognitive vulnerability to bipolar disorders are in their infancy and much work remains to be done.

NEGATIVE COGNITIVE STYLES, INFORMATION PROCESSING, AND RUMINATION AS COGNITIVE VULNERABILITIES

In this section, we review empirical findings regarding negative cognitive styles, negatively biased information processing, and ruminative response styles that are associated with and predictive of mood disorders. We consider evidence for these cognitive factors as vulnerabilities to unipolar depression and bipolar spectrum disorders, respectively.

UNIPOLAR DEPRESSION

Cross-sectional studies have demonstrated associations between negative inferential styles, dysfunctional attitudes, information processing biases, and ruminative response styles and depression in adults, adolescents, and children (see Abramson et al., 2002; Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997; Ingram et al., 1998; Spasojevic, Alloy, Abramson, MacCoon, & Robinson, 2003, for reviews). Studies using the remitted depression design have been mixed. Although some studies have found that individuals remitted from a depressive episode show negative cognitive styles, the majority find that remitted depressives do not exhibit negative cognitive patterns, unless the cognitive styles are "activated" by a depressive mood induction (see Ingram et al., 1998; Just et al., 2001; and Persons & Miranda, 1992, for reviews). In contrast, recent studies using the more ideal prospective designs with children, adolescents, and adults have found that negative inferential styles and dysfunctional attitudes do increase risk for depressive symptoms (see Abramson et al., 2002, for a review). However, the strongest evidence that the hypothesized cognitive vulnerabilities actually increase risk for clinically significant depressive disorders comes from the Temple—Wisconsin CVD Project (Alloy & Abramson, 1999).

In the CVD Project, university freshmen with no Axis I psychiatric disorders at the study outset were selected to be at hypothesized high risk (HR) or low risk (LR) for depression based on the presence versus absence of negative cognitive styles, as assessed by the Dysfunctional Atti-

tudes Scale (DAS; Weissman & Beck, 1978) and Cognitive Style Questionnaire (CSQ; Alloy et al., 2000). The HR and LR participants were followed prospectively every 6 weeks for 2.5 years and then every 16 weeks for an additional 3 years with self-report and structured interview measures of stressful life events, cognitions, and symptoms and diagnosable episodes of depression and other disorders (see Alloy & Abramson, 1999; Alloy et al., 2000, for details).

The cognitive vulnerability hypothesis of Hopelessness and Beck's theories was tested both retrospectively and prospectively in the CVD Project. In the retrospective test of the hypothesis, Alloy et al. (2000) reported that, controlling for current levels of depressive symptoms, cognitively HR participants exhibited higher lifetime rates of *DSM-III-R* and RDC major depression (MD) and HD than did LR participants, as well as marginally higher lifetime rates of RDC minor depression (MiD). Indicating possible specificity of cognitive vulnerability to depressive disorders, Alloy et al. (2000) found that there were no risk group differences in lifetime history of anxiety disorders, substance use disorders, or other psychiatric disorders. In a follow-up to the Alloy et al. (2000) study, Haeffel et al. (2003) used a new sample of unselected undergraduates to examine the unique associations between negative inferential styles and dysfunctional attitudes and lifetime history of depressive and other disorders. Haeffel et al. found that negative inferential styles were more strongly and consistently associated with lifetime history of RDC MD and HD than were dysfunctional attitudes, suggesting that negative inferential styles, as assessed by the CSQ, may have been a particularly potent component of the "generic" cognitive vulnerability effect in Alloy et al.'s (2000) study.

Although these retrospective findings are suggestive, alone they do not adequately address whether negative cognitive styles confer vulnerability to depression, because the findings are equally consistent with the "scar hypothesis" (Lewinsohn et al., 1981). Thus, data from the prospective part of the CVD project are needed to decide among these alternatives. Results from the first 2.5 years of follow-up indicated that negative cognitive styles did indeed predict prospectively both first onsets and recurrences of depressive disorders (Alloy, Abramson, Whitehouse et al., 1999; Alloy, Abramson, Whitehouse, Hogan, Panzarella et al., 2006). Among participants with no prior history of depression, HR individuals were more likely than LR individuals to experience a first lifetime onset of MD, MiD, and HD, but did not differ in their likelihood of onset of any anxiety disorder or other Axis I disorder. These findings provide especially strong support for the cognitive vulnerability hypothesis because they are based on a truly prospective test, uncontaminated by prior history of depression.

Given that depression is highly recurrent (Judd, 1997), it is also important to examine whether negative cognitive styles provide risk for recurrences of depression. Among individuals with a past history of depression, HR participants also were more likely than LR participants to have a recurrence of MD, MiD, and HD, in the first 2.5 years of follow-up. Again, there were no significant risk group differences in the rates of anxiety and other psychiatric disorders among the recurrence subsample. All of these findings were maintained even after statistically controlling for individuals' initial depressive symptoms at the study outset. The CVD Project findings suggest that, at least in part, similar cognitive vulnerabilities may play a role in risk for both the first and subsequent episodes of depressive disorders.

Although cognitive styles are not immutable (Just et al., 2001), findings from the CVD Project support the relative stability of negative inferential styles and dysfunctional attitudes. Specifically, Berrebbi, Alloy, and Abramson (2006) found that the cognitive styles of CVD Project participants remained stable from before to during and after intervening episodes of MD. Similarly, Raniere, Alloy, and Abramson (2006) found that participants' inferences for specific life events they experienced remained stable over up to 5 years of follow-up. Thus, negative cognitive styles may be a relatively stable vulnerability factor for depression, independent of individuals' current symptom status.

According to the cognitive theories of depression, people with negative cognitive styles are vulnerable to depression, in part, because they tend to engage in negatively biased information processing about themselves when they encounter stressful events. Specifically, according to Beck's theory (1967, 1987; Clark et al., 1999), depression-prone individuals possess negative self-schemata that negatively bias their appraisals and memory of personally relevant experiences. Data from the CVD Project are consistent with the hypothesis that cognitively HR individuals engage in negative self-referent information processing. CVD Project participants were administered a Self-Referent Information Processing Task Battery (SRIP) at Time 1. Controlling for concurrent depressive symptoms, Alloy et al. (1997) found that, compared to LR participants, HR participants showed preferential processing of negative depression-relevant adjectives (e.g., "failure," "unmotivated," "useless") as evidenced by relatively greater endorsement, faster response times, greater accessibility, better recall, and higher predictive certainty of this material. In addition, HR participants were less likely than LR participants to process positive depression-relevant adjectives (e.g., "worthy," "competent," "energetic"). These findings demonstrate that information-processing biases previously observed in currently depressed individuals (e.g., Ingram et al., 1998) also occur in

nondepressed individuals who are cognitively vulnerable to depression.

Moreover, prospective data from the CVD Project indicate that negative self-referent information processing acts as an additional cognitive vulnerability factor for depression. Steinberg, Oelrich, Alloy, and Abramson (2006) examined whether negative self-referent information processing exacerbated the predictive association between negative cognitive styles and both first onsets and recurrences of depressive disorders during the first 2.5 years of prospective follow-up. They found that negative self-referent information processing (based on a composite of the dependent measures from the SRIP for negative depression-relevant stimuli) interacted with cognitive risk to predict first onsets, but not recurrences, of MD and MiD combined and of HD significantly, even when initial depressive symptom levels were controlled statistically. HR participants who also engaged in negative self-referent processing were more likely to have a first onset of MD or MiD and HD than were HR participants who did not exhibit negative information processing, whereas among LR participants, there was no association between negative self-referent processing and first onsets of depression.

According to the response styles theory (Nolen-Hoeksema, 1991), a ruminative response style also provides vulnerability to full-blown depressive episodes. Several laboratory and field studies have found that depressive rumination is associated with longer and more severe episodes of depression (see Spasojevic et al., 2003, for a review). In addition, prospective studies, including the CVD Project, have found that depressive rumination predicts onsets of clinically significant MD (Nolen-Hoeksema, 2000; Spasojevic & Alloy, 2001). Moreover, using CVD Project data, Spasojevic and Alloy (2001) found that a tendency to ruminate about one's dysphoria mediated the association between several more general depressive risk factors (negative cognitive styles, dependency, self-criticism, neediness, and past history of depression) and onsets of MD episodes. Indeed, Abramson et al. (2002) argued that negative cognitive styles, by their very nature, should increase the likelihood of rumination because cognitively vulnerable individuals have difficulty disengaging their attention from the self-implications of negative life events.

Expanding upon the response styles theory, Robinson and Alloy (2003) hypothesized that individuals who exhibit negative cognitive styles and who also tend to ruminate about these negative cognitions in response to the occurrence of stressful life events ("stress-reactive rumination" instead of "emotion-focused rumination") may be more likely to develop episodes of depression in the first place. They reasoned that negative cognitive styles generate negative content, which should be

more likely to lead to depression when it is “on one’s mind” and repetitively rehearsed than when it is not. Thus, Robinson and Alloy hypothesized that stress-reactive rumination would exacerbate the association between negative cognitive styles and onsets of depressive episodes. Consistent with this hypothesis, they found that cognitive risk status and stress-reactive rumination measured at Time 1 of the CVD Project did indeed interact to predict onsets of MD and HD. Among HR participants, those who were also high in stress-reactive rumination were more likely to have an onset of MD and HD than those who didn’t tend to ruminate in response to stressors. Low risk participants exhibited low rates of future depressive episodes, regardless of their levels of stress-reactive rumination.

To our knowledge, the prospective findings from the CVD Project provide the first and clearest demonstration that negative cognitive styles, negative self-referent information processing, and a ruminative response style indeed confer vulnerability for full-blown, clinically significant depressive disorders. In addition, the risk conferred by negative cognitive styles may have specificity to depressive disorders insofar as we did not obtain risk group differences in onsets of anxiety or other disorders. Given this evidence for cognitive vulnerability to unipolar depression, we turn now to the status of the evidence for cognitive vulnerability to bipolar disorders.

BIPOLAR DISORDER

In the last decade, there has been growing interest in the role of cognitive styles and information processing as vulnerabilities for episodes of bipolar disorder. Here, we review the extant cross-sectional studies on the cognitive patterns associated with bipolar disorders and the independence of such patterns from bipolar individuals’ current symptomatic state, as well as preliminary findings from the Wisconsin-Temple LIBS Project suggesting that certain cognitive styles prospectively predict episodes of bipolar disorder. The studies conducted to date suggest that the observed positivity or negativity of bipolar individuals’ cognitive patterns depends to some degree on their symptomatic state at the time of the assessment and on whether the assessment of cognitive patterns is based on explicit or implicit tasks (Alloy, Abramson et al., 2006; Alloy, Reilly-Harrington et al., 2006). Most studies indicate that bipolar individuals show cognitive styles and self-referent information processing as negative as those of unipolar depressed individuals, but sometimes present themselves in a positive fashion on more explicit cognitive tasks.

Studies of bipolar individuals in a current depressive episode generally have found their cognitive patterns to be as negative as those of uni-

polar depressives. For example, both depressed unipolar and bipolar patients show equally negative automatic thoughts and dysfunctional attitudes characteristic of depression (Hill, Oei, & Hill, 1989; Hollon, Kendall, & Lumry, 1986). On the Depressive Experiences Questionnaire (DEQ; Blatt, D'Afflitti, & Quinlan, 1976), Rosenfarb, Becker, Khan, and Mintz (1998) found that both depressed unipolar and bipolar women were more self-critical than nonpsychiatric controls, but only the unipolar depressed women were more dependent than controls. Finally, Reilly-Harrington et al. (1999) compared the subset of their sample (31 unipolar, 7 bipolar) currently in a depressive episode with 23 normal controls and the currently euthymic mood disordered participants. They found that both currently depressed unipolar and bipolar participants exhibited more internal, stable, global attributional styles for negative events, more external, unstable, specific attributional styles for positive events, and more negative self-referent information processing than did nondepressed participants.

Studies of currently manic or hypomanic bipolar individuals have obtained results consistent with the importance of distinguishing between explicit and implicit measures of cognitions. Bentall and Thompson (1990) compared students who scored high versus low on a hypomania scale on an implicit emotional Stroop test in which the participants named the ink colors of depression-related and euphoria-related words. Consistent with prior findings on the emotional Stroop test with unipolar depressed patients (Ingram et al., 1998), hypomanic students took longer to name the color of depression-related, but not euphoria-related words (see also replication by French, Richards, and Scholfield, 1996, controlling for the effects of anxiety on Stroop performance). Lyon, Startup, and Bentall (1999) assessed 15 bipolar manic patients, 15 bipolar depressed patients, and 15 normal controls on an explicit attribution questionnaire, Winters and Neale's (1985) pragmatic inference task, assessing attributions for hypothetical scenarios in an implicit way, the emotional Stroop test, and a self-referent incidental recall task. Consistent with the hypothesis that bipolar depressed individuals possess negative cognitive patterns like those of unipolar depressives, Lyon et al. found that bipolar depressed patients exhibited a negative attributional style on both the attribution questionnaire and the pragmatic inference task, exhibited slower color-naming for depression-related words on the emotional Stroop task, and endorsed as self-descriptive and recalled more negative trait adjectives on the incidental recall task. Although, like the normal controls, the bipolar manic patients showed a positive attributional bias on the explicit attribution questionnaire and endorsed more positive than negative words as self-descriptive, they exhibited negative cognitive patterns like those of depressed

individuals on the more implicit tasks. Specifically, manic patients attributed negative events internally rather than externally on the pragmatic inference task, showed slower color-naming for depression-related rather than euphoria-related words on the Stroop task, and recalled more negative than positive words on the self-referent incidental recall task.

The few studies examining the stability of the cognitive patterns of bipolar individuals, independent of their symptomatic state, have relied on either cross-sectional studies of euthymic bipolar individuals who have remitted from a depressive or hypomanic/manic episode or longitudinal studies of bipolar individuals across depressed, hypomanic, and euthymic periods. Three studies of remitted bipolar individuals did not obtain much evidence of negative cognitive patterns in the remitted state. In two studies of the same sample, Tracy and colleagues (Pardoen, Bauwens, Tracy, Martin, & Mendlewicz, 1993; Tracy, Bauwens, Martin, Pardoen, & Mendlewicz, 1992) found no evidence of negative attributions or low self-esteem among remitted bipolar patients compared to controls. Similarly, Reilly-Harrington et al. (1999) did not obtain differences in attributional style, in dysfunctional attitudes, or on most measures of self-referent processing among 66 remitted unipolar, 37 remitted bipolar, and 23 normal control undergraduates. However, the remitted bipolar participants endorsed more depression-relevant than nondepression-relevant adjectives as self-descriptive and predicted that they would be more likely to behave in depression-relevant than nondepression-relevant ways in the future compared to remitted unipolar and control participants.

However, four other studies obtained much greater support for negative cognitive styles and information processing among remitted bipolar individuals. Winters and Neale (1985) assessed groups of remitted bipolar and unipolar patients and normal controls on explicit measures of self-esteem and on an implicit pragmatic inference task measuring causal attributions. Although remitted bipolar patients exhibited higher self-esteem than the remitted unipolar patients and normal controls on the explicit measures, they generated attributions as negative as those of the remitted unipolar patients on the implicit pragmatic inference task. Among their subsample of remitted unipolar and bipolar women and nonpsychiatric control women, Rosenfarb et al. (1998) found that both the remitted unipolar and bipolar women were more self-critical than controls, but only the remitted unipolar women were more dependent than the controls. In a sample of 41 euthymic bipolar patients compared with 20 normal controls, Scott, Stanton, Garland, and Ferrier (2000) found that the remitted bipolar patients exhibited more dysfunctional attitudes (particularly perfectionism and need for approval), greater

sociotropy, greater over-general recall on an autobiographical memory task, and fewer solutions on a social problem-solving task. Finally, at Time 1 of the LIBS Project, Abramson, Alloy, and their colleagues (Abramson et al., 2006; Alloy, Abramson, Whitehouse, Hogan, Grandin et al., 2006) compared the cognitive styles and self-referent information processing of 206 euthymic bipolar spectrum (Bipolar II, Cyclothymia) individuals and 206 demographically matched normal controls. Bipolar participants showed depressive cognitive styles and information processing comparable or worse than that shown by participants at high risk for unipolar depression in the CVD Project. Bipolar individuals exhibited more negative inferential styles, dysfunctional attitudes (particularly, perfectionism), autonomy (particularly, mobility/freedom from control), self-criticism, self-consciousness (particularly, private self-consciousness), and ruminative response styles than did normal controls, but did not differ on dependency from the DEQ, sociotropy from the SAS, or the attachment subscale of the DAS. In addition, bipolar participants showed greater processing of negative, depression-relevant stimuli and less processing of positive, depression-relevant stimuli on the SRIP Task Battery compared to the normal controls. Thus, euthymic bipolar individuals' negative cognitive patterns were characterized especially by concerns with performance evaluation, perfectionism, autonomy, self-criticism, and rumination, rather than by dependency, sociotropy, or attachment concerns, as is often true of individuals at risk for unipolar depression.

Only two studies to date used a longitudinal design to investigate the stability of cognitive styles across the mood swings of individuals with bipolar mood disorders. Among 10 rapid-cycling bipolar patients, Eich, Macaulay, and Lam (1997) found that recall of autobiographical memories was more negative in the depressed than in the manic state. Alloy, Reilly-Harrington et al. (1999) assessed attributional styles and dysfunctional attitudes, as well as state cognitions about the self, in a sample of individuals with untreated, subsyndromal unipolar and bipolar conditions (13 cyclothymic, 8 dysthymic, 10 hypomanic, and 12 normal controls) on 3 separate occasions as the different mood states characteristic of their disorder naturally occurred. At Time 1, all groups were assessed in a normal, euthymic state. At Time 2, cyclothymic and dysthymic participants were in a depressed state, hypomanic participants were in a hypomanic state, and normal controls were in a normal mood state. At Time 3, dysthymics were in another depressed state, cyclothymics and hypomanics were in a hypomanic state, and normals were in a normal mood state. The interval between each of the sessions averaged 4.7 weeks, with a range of 1 to 9 weeks. Analyses of participants' depression and mania/hypomania symptom scores across the 3 sessions indicated

that, as intended, participants were successfully assessed in the different mood states appropriate to their diagnoses at each time point.

Alloy, Reilly-Harrington et al. (1999) found that attributional styles and dysfunctional attitudes were stable across participants' mood swings. Across the various mood states, cyclothymics' and dysthymics' dysfunctional attitudes and attributional styles for negative events did not differ from each other and both groups had more negative styles than hypomanics and normal controls, whose scores also did not differ from each other. In contrast, the more state-like self perceptions did differ as a function of current mood state. Whereas the four groups did not differ on self perceptions at Times 1 and 3, at Time 2, when cyclothymics and dysthymics were in a depressed state and hypomanics were in a hypomanic state, dysthymics' and cyclothymics' thoughts about the self were more negative than those of hypomanics and normal controls. Also, cyclothymics' self-referent thoughts were more negative when they were depressed (Time 2) than when they were either hypomanic (Time 3) or euthymic (Time 1).

The Alloy, Reilly-Harrington et al. (1999) findings are intriguing in several respects. First, in contrast to cyclothymics, unipolar hypomanic participants who have no depressive episodes as part of their history, showed much more positive attributional styles and attitudes, similar to those of normal controls. This suggests that the cognitive styles of unipolar mania/hypomania may be quite different and more positive in nature than mania/hypomania in the context of a history of depression. Further studies are needed that examine other cognitive styles and information-processing biases in bipolar versus unipolar manic/hypomanic groups to determine whether unipolar mania/hypomania is associated with more positive cognitive patterns in general than mania/hypomania in the context of a history of depressive episodes. Second, the cognitive vulnerabilities (negative attributional styles and dysfunctional attitudes) featured in the cognitive theories of unipolar depression showed considerable stability across large changes in naturally occurring mood states. This is in contrast to the results of at least some of the studies of remitted bipolar and unipolar individuals described above. Alloy, Reilly-Harrington et al. speculated that participants' cognitive styles showed stability across mood swings in their study because they had a sample of untreated individuals. Most of the prior remitted studies involved treated samples and thus, cognitive styles may have improved as a byproduct of treatment rather than as a naturally occurring result of symptom remission without intervention. Finally, whereas cyclothymic individuals in Alloy, Reilly-Harrington et al.'s (1999) study exhibited stable distal cognitive styles across mood states, their proximal cognitions (self perceptions) varied as a function of

current mood state and were more positive in a hypomanic than in a depressed period. This suggests that whereas bipolar individuals may exhibit negative cognitive styles that are relatively stable, they may also possess more latent positive self-schemata that are only activated in positive mood states. Future studies will need to examine this idea directly.

We are aware of only four studies that examined whether the cognitive styles that provide vulnerability to onsets and recurrences of unipolar depression also increase vulnerability to symptoms or episodes of bipolar disorder. First, in a small sample of Bipolar I patients, Johnson, Meyer, Winett, and Small (2000) examined whether level of self-esteem predicted future depressive and manic symptoms. They found that low self-esteem predicted increases in depressive symptoms, but not manic symptoms, over time. Similarly, with a larger Bipolar I sample, Johnson and Fingerhut (2004) found that more negative and fewer positive automatic thoughts predicted increases in depressive, but not manic, symptoms over a 2-year follow-up. Neither depression nor mania symptom change were predicted by dysfunctional attitudes. In contrast to Johnson et al. (2000), Scott and Pope (2003) reported that negative self-esteem was the most robust predictor of relapse at 12-month follow-up among a small sample of hypomanic bipolar patients. Finally, as part of the LIBS Project, we (Alloy, Abramson, Walshaw, Whitehouse, & Hogan, 2006) examined whether cognitive styles predicted onsets of MD episodes and hypomanic/manic (Hyp/Ma) episodes during the longitudinal follow-up period. Controlling for initial depressive and hypomanic symptoms, preliminary analyses based on an average of 33 months of follow-up found that negative inferential styles for negative events, private self-consciousness, and autonomy all prospectively predicted onsets of MD. In addition, private self-consciousness, autonomy, and self-criticism predicted onsets of Hyp/Ma. These findings provide preliminary evidence that cognitive styles related to increased vulnerability to episodes of unipolar depression may also increase risk for episodes of bipolar disorder.

COGNITIVE VULNERABILITY-STRESS PREDICTION OF UNIPOLAR AND BIPOLAR MOOD EPISODES

The cognitive theories of depression are vulnerability-stress models in which particular cognitive styles and information-processing biases increase vulnerability to depression in response to life events. In this section, we address whether cognitive patterns do, in fact, interact with the occurrence of negative or positive life events to predict onsets of unipolar depression and bipolar episodes.

UNIPOLAR DEPRESSION

Many studies have used longitudinal, prospective designs to test the cognitive vulnerability–stress hypotheses of Hopelessness and Beck’s theories for increases in depressive mood and symptoms. The majority of these studies have found that the interaction between negative cognitive styles or negative information processing and the occurrence of negative life events predict increases in depressive mood and symptoms over time in children, adolescents, and adults (see Abramson et al., 2002; Garber & Flynn, 2001; Ingram et al., 1998, for reviews). Surprisingly little is yet known about whether negative cognitive styles combine with stressful events to predict onsets of diagnosable unipolar depressive disorders.

In a sample of adolescents, Lewinsohn, Joiner, and Rohde (2001) found that stressful events interacted with both attributional styles and dysfunctional attitudes (at the level of a statistical trend) to predict onsets of MD over a 1 year follow–up period. The form of the interaction, however, was different for attributional styles and dysfunctional attitudes. Whereas dysfunctional attitudes were most likely to lead to MD under conditions of high stress, consistent with a “synergistic model” of vulnerability–stress relations and most past research involving prediction of depressive symptoms, attributional style was most predictive of MD under conditions of low stress, consistent with the “titration model” of vulnerability–stress relations in the original statement of the Hopelessness theory (Abramson et al., 1989). According to a synergistic model, a person must be high on both vulnerability and stress to develop depression, whereas the titration model suggests that low levels of one factor may be compensated by high levels of the other factor to lead to depression (see Abramson et al., 2002). However, Lewinsohn et al. (2001) acknowledged important limitations in their assessment of attributional styles and stress that require caution in interpreting their findings.

Preliminary analyses of data from the CVD Project based on the first year of follow–up are also relevant to whether negative cognitive styles in interaction with stressful life events predict the onset of clinically significant depressive episodes and of HD. Life events were assessed in the CVD Project every 6 weeks with a combination self–report and structured interview assessment. The preliminary analyses indicated that after controlling for the separate main effects of cognitive risk status and the number of negative events experienced in the prior 6–week interval, the cognitive vulnerability–stress interaction predicted onsets of MD or MiD and of HD significantly. The form of the interaction fit the synergistic model of vulnerability–stress relations. High risk participants who

experienced high levels of stressful events were much more likely to experience an onset of MD, MiD, or HD than were HR participants who experienced low levels of stressful events or than LR individuals regardless of their stress levels. Consistent with these preliminary findings from the CVD Project, in a sample of high school students, Hankin, Abramson, Miller, and Haefel (2004) also found that both negative inferential styles and dysfunctional attitudes interacted with negative life events occurring over a 2-year interval to predict increases in depressive symptoms and onsets of MD, but not of anxiety symptoms or disorders. Thus, initial evidence with respect to the cognitive vulnerability–stress prediction of clinically significant depressive disorders is promising.

BIPOLAR DISORDER

We are aware of five studies to date that have examined the cognitive vulnerability–stress hypothesis for bipolar disorders. All of these studies have examined prediction of depressive and manic/hypomanic symptom changes, but not episodes. Hammen, Ellicott, Gitlin, and Jamison (1989) tested Beck's (1987) event congruence, vulnerability–stress hypothesis in 22 unipolar and 25 bipolar patients, categorized into sociotropic and autonomous subtypes and then followed for 6 months. Based on Beck's theory, it was predicted that patients who experienced a preponderance of negative events congruent with their cognitive style (interpersonal events for sociotropic patients and achievement events for autonomous patients) would be more likely to experience an onset or exacerbation of symptoms. Hammen et al. obtained support for the event congruence hypothesis only in the unipolar patients; but, there were trends consistent with the hypothesis for bipolar patients as well. Indeed, in a later study, Hammen et al. (1992) followed a larger sample of 49 remitted bipolar patients for an average of 18 months. Although symptom onset was not associated with cognitive style–life event congruence, subsequent symptom severity was significantly related to the interaction of sociotropy and negative interpersonal events, consistent with the vulnerability–stress hypothesis. Using the first 4 months of follow-up data in the LIBS Project, Francis–Raniere, Alloy, and Abramson (2006) found that among bipolar participants, after controlling for initial depressive symptoms and the total number of negative life events experienced, the interaction of autonomous cognitive styles with congruent, autonomy–relevant negative events predicted increases in depressive symptoms over the 4 months. Similarly, among bipolar individuals, after controlling for initial hypomanic symptoms and the total number of positive life events experienced, the autonomous styles ×

autonomy–relevant positive events interaction predicted increases in hypomanic symptoms over 4 months.

Two studies tested the cognitive vulnerability–stress hypotheses of Hopelessness as well as Beck’s theories in samples including bipolar individuals. Alloy, Reilly-Harrington et al. (1999) examined whether attributional styles and dysfunctional attitudes assessed at Time 1 in a normal, euthymic mood state interacted with subsequent positive and negative life events to predict prospective increases in depressive and hypomanic symptoms in their sample with unipolar and bipolar conditions. Consistent with Hopelessness theory, an internal, stable, global attributional style for negative events at Time 1 interacted with subsequent negative events to predict increases in depressive symptoms at Times 2 and 3. In addition, an internal, stable, global attributional style for positive events at Time 1 interacted with subsequent positive events to predict increases in hypomanic symptoms at Time 2. Dysfunctional attitudes did not combine with positive or negative events to predict changes in either depressive or hypomanic symptoms at later times. Reilly-Harrington et al. (1999) also examined whether the interaction of Time 1 attributional styles, dysfunctional attitudes, and negative self-referent information processing (assessed by the SRIP) and intervening negative life events predicted increases in 97 unipolar and 49 bipolar individuals’ clinician-rated depressive and manic symptoms a month later. Consistent with both Hopelessness and Beck’s theories, negative attributional styles, dysfunctional attitudes, and negative self-referent information processing each interacted significantly with subsequent negative life events to predict increases in depressive symptoms and, within the bipolar group, manic symptoms. Only individuals with negative cognitive styles or information processing at Time 1 who reported a high number of negative events experienced increases in depressive and manic symptoms at Time 2.

In summary, the results of the few vulnerability–stress studies to date are promising in supporting the applicability of the cognitive theories of unipolar depression to bipolar spectrum disorders. As such, they suggest that cognitive processes may contribute vulnerability to both unipolar and bipolar forms of mood disorder.

Two issues raised by the vulnerability–stress findings in bipolar disorder to date remain to be resolved in future research. First, although two studies (Hammen et al., 1992; Reilly-Harrington et al., 1999) found that negative events interacted with negative cognitive styles to predict both depressive and manic/hypomanic symptoms among bipolar individuals, two studies (Alloy, Reilly-Harrington et al., 1999; Francis-Raniere et al., 2006) found that it was positive events in interaction with positive

cognitive styles that predicted manic/hypomanic symptoms. Thus, more work is needed to understand the conditions under which positive versus negative events and positive versus negative cognitive styles provide vulnerability to mania/hypomania. Second, given that two studies found that negative events interact with negative cognitive styles and information processing to predict increases in both depressive and manic/hypomanic symptoms, what determines which type of episode a bipolar individual will experience at any given time? Reilly–Harrington et al. (1999) speculated that the particular kind of stressful event may be key, with manic/hypomanic episodes more likely to follow stressors that disrupt the sleep–wake cycle (i.e., social rhythm disruptors; Malkoff–Schwartz et al., 1998) and depressive episodes more likely to follow loss events (e.g., Brown & Harris, 1978). Alternatively, the perceived controllability of stressful life events may be important. In accord with Wortman and Brehm’s (1975) reactance model as well as behavioral approach system (BAS) dysregulation theories of bipolar disorder (see Depue & Iacono, 1989; Johnson et al., 2000; Urosevic et al., 2006), when bipolar individuals experience negative life events they perceive to be completely uncontrollable, depression may ensue; whereas when they experience stressors that they perceive to be surmountable challenges, they may react with increased energy and goal directedness and hypomania may result. Future work involving assessments of both objective characteristics and subjective interpretations of the nature of stressful events that trigger depressive and manic/hypomanic episodes is needed to test both of these intriguing proposals.

DEVELOPMENTAL ORIGINS OF COGNITIVE VULNERABILITY TO UNIPOLAR AND BIPOLAR MOOD DISORDERS

If maladaptive cognitive styles and information processing do confer vulnerability to episodes of both unipolar depression and bipolar disorder, then it becomes important to understand the developmental origins of these cognitive vulnerabilities. In this section, we review what is known about the developmental antecedents of cognitive vulnerability to unipolar and bipolar disorder, with particular emphasis on findings from the CVD and LIBS Projects.

UNIPOLAR DEPRESSION

Recent reviews of the developmental antecedents of depression and cognitive vulnerability to depression (e.g., Garber & Flynn, 1998; Gibb, 2002; Goodman & Gotlib, 1999; Rose & Abramson, 1992) suggest that ge-

netic, neurochemical, social learning, and early traumatic processes all contribute to the development of negative cognitive styles that, in turn, increase risk for depression. In the CVD Project, we focused on exploring in some detail the social learning and early traumatic mechanisms that may contribute to the development of cognitive vulnerabilities and risk for depression (see Alloy, Abramson, Gibb et al., 2004 for a review). Toward this end, as part of the CVD Project, 335 of the parents (217 mothers and 118 fathers) of the HR and LR participants were assessed on their cognitive styles and parenting practices. In addition, the parents reported on their offspring's early childhood life events and the HR and LR participants reported on their own histories of childhood maltreatment from both parents and nonrelatives.

Children's cognitive styles may develop in part through social learning mechanisms, such as indirect modeling of parents' cognitive styles or direct learning of cognitive styles from parental inferential feedback regarding the causes and consequences of negative events in the child's life. If modeling occurs, then children's cognitive styles should correlate with those of their mothers or fathers. In the CVD Project, mothers of HR individuals had more dysfunctional attitudes, but not more negative inferential styles, than mothers of LR individuals, controlling for the mothers' levels of depressive symptoms (Alloy et al., 2001). Fathers' cognitive styles didn't differ for HR and LR participants. Similarly, other studies have obtained mixed support for the modeling hypothesis, with some studies finding a relationship between mothers', but not fathers', and children's negative cognitions, and others showing no association between parents' and their offspring's cognitive styles (see Alloy et al., 2001, for a review).

In contrast, studies have provided more consistent support for the hypothesis that negative inferential feedback from parents may contribute to children's development of negative cognitive styles (see Alloy et al., 2001). For example, in the CVD Project, Alloy et al. (2001) found that according to both participants' and parents' reports, both mothers and fathers of HR individuals provided more negative (stable, global) attributional and consequence feedback for negative events in their child's life than did mothers and fathers of LR individuals, controlling for either respondent's level of depressive symptoms. In addition, mothers' inferential feedback predicted the likelihood of their child developing an episode of MD, MiD, or HD over the 2.5-year prospective follow-up, mediated in part by the child's cognitive risk status (Alloy et al., 2001). Moreover, Crossfield, Alloy, Abramson, and Gibb (2002) found that parental inferential feedback moderated the association between negative childhood life events and cognitive vulnerability. Specifically, a history of high levels of negative childhood events combined with neg-

ative maternal inferential feedback was associated with HR status among participants. Thus, parents may contribute to the development of cognitive vulnerability to depression in their children, not so much by modeling negative appraisals of events in their own lives, but by providing negative attributional and consequence feedback to their children for negative events in the child's life.

In addition to parental inferential feedback, negative parenting practices may also contribute to the development of cognitive vulnerability to depression. In particular, a parenting style involving lack of emotional warmth and negative psychological control (criticism, intrusiveness, and guilt-induction), a pattern referred to as "affectionless control" by Parker (1983), is associated with both depression and negative cognitive styles in offspring (see Alloy et al., 2001; Alloy, Abramson, Gibb et al., 2004; and Garber & Flynn, 1998, for reviews). Consistent with the lack of emotional warmth part of the "affectionless control" pattern, Alloy et al. (2001) found that controlling for respondents' depressive symptom levels, fathers of HR participants exhibited less acceptance than did fathers of LR participants, as reported by both the participants and their fathers. There were no risk group differences, however, for fathers' levels of psychological control or for mothers' parenting. In addition, low acceptance from fathers predicted prospective onsets of MD, MiD, and HD in their children during the 2.5-year follow-up, but only the prediction of HD episodes was mediated by the children's cognitive risk status. Interestingly, negative parenting practices also predicted a ruminative response style among participants, but it was negative psychological control by both parents, rather than low emotional warmth, that was related to depressive rumination (Spasojevic & Alloy, 2002). In addition, rumination mediated the relationship between the overcontrolling parenting and prospective onsets of MD in the offspring. Thus, both low emotional warmth and overcontrolling parenting were related to offspring's cognitive vulnerability to depression, through the alternative mechanisms of negative cognitive styles and rumination, respectively.

Rose and Abramson (1992) proposed a developmental pathway by which childhood negative life events, especially childhood maltreatment, may lead to the development of a negative cognitive style. They suggested that whereas a child may initially explain being beaten or verbally abused by his/her father by saying, "He was just in a bad mood today" (an external, unstable, specific attribution), repeated occurrences of abuse will lead these more benign attributions to be disconfirmed, leading the child to begin making hopelessness-inducing attributions about recurrent maltreatment (e.g., "I'm a terrible person who deserves all the bad things that happen to me"; an internal, stable, global attribu-

tion). Over time, the child's negative attributions may generalize until a negative cognitive style develops. Rose and Abramson (1992) hypothesized that emotional maltreatment may be even more likely than either physical or sexual maltreatment to contribute to the development of negative cognitive styles because with emotional abuse, the abuser directly supplies the negative cognitions to the child (e.g., "You're so stupid; you'll never amount to anything").

A review of the childhood maltreatment and cognitive vulnerability to depression literature indicated that sexual and emotional abuse histories, but not physical abuse, were associated with negative cognitive styles (Gibb, 2002). In the CVD Project, controlling for participants' levels of depressive symptoms, HR participants reported significantly higher levels of childhood emotional, but not physical or sexual, maltreatment than did LR participants (Gibb et al., 2001). In addition, even when maltreatment by parents and parental history of psychopathology were controlled statistically, emotional maltreatment by nonrelatives (peers and romantic partners) still was significantly associated with cognitive vulnerability (Gibb, Abramson, & Alloy, 2004), suggesting that the association between emotional abuse and cognitive vulnerability is not entirely attributable to genetic transmission or a negative family environment in general. Moreover, participants' cognitive risk status mediated the relationship between levels of emotional maltreatment and prospective onsets of MD and HD episodes during the follow-up (Gibb et al., 2001). Childhood history of emotional abuse was also related to a ruminative response style and negative self-referent information processing (Spasojevic & Alloy, 2002; Steinberg, Gibb, Alloy, & Abramson, 2003).

Whereas these findings are based on retrospective reports of maltreatment, Gibb, Alloy et al. (2006) conducted a prospective study examining the role of emotional maltreatment in predicting change in attributional style over a 6 month period in 10-year-olds. Emotional maltreatment occurring during the 6 month follow-up, as well as in the 6 months prior to Time 1, predicted change in children's attributional styles over the follow-up. The more emotional abuse a child experienced, the more negative his or her attributional style became over the follow-up. These findings suggest that emotional maltreatment may, at least, show temporal precedence with respect to the development of some negative cognitive styles.

BIPOLAR DISORDER

Aside from the work on genetic transmission, little research has been conducted to explore the developmental antecedents of bipolar disorder

(but see, Alloy, Abramson et al., 2006, for a review). The few available studies, however, suggest that there is an association between family environment and parenting practices and the severity and course of bipolar disorder. Parker (1981) and Joyce (1984) did not find differences between bipolar patients' and general medical patients' reports of the parenting practices they experienced growing up; however, Joyce (1984) found that within the bipolar group, high levels of "affectionless control" (low care, high overprotection) from parents were associated with increased hospitalizations for both depression and mania. Cooke, Young, Mohri, Blake, and Joffe (1999) also found that low levels of family cohesion were associated with a history of past suicide attempts among bipolar individuals. Relatedly, in a sample of 631 bipolar outpatients, Leverich et al. (2002) found that a history of childhood or adolescent physical or sexual abuse was associated with a higher incidence of early illness, suicide attempts, faster cycling frequencies, greater alcohol and substance abuse, greater lifetime Axis I and II disorders, and a higher incidence of stressful events reported as occurring before the first and most recent affective episode. Moreover, negative family interactions and attitudes have been found to predict relapse of episodes among bipolar patients (e.g., Miklowitz, Goldstein, & Nuechterlein, 1995; Miklowitz, Goldstein, Nuechterlein, Snyder, & Mintz, 1988).

As part of the LIBS Project, Neeren, Alloy, Abramson, Pieracci, and Whitehouse (2006) explored whether the same parenting styles and maltreatment that are characteristic of the parents of individuals at high cognitive risk for unipolar depression are also associated with a bipolar diagnosis. Neeren et al. found that controlling for a family history of mania and/or bipolar disorder, as well as depressive and hypomanic symptoms, participants' reports of low warmth from mothers and high negative psychological control from both parents and physical abuse from mothers and emotional abuse from both parents were associated with bipolar versus normal group status. Given that these parenting characteristics were related to bipolarity, despite controlling for bipolar family history, the results suggest that negative parenting may contribute to the development or expression of bipolar spectrum disorders among offspring over and above purely genetic transmission of vulnerability to bipolar disorder. Also as part of the LIBS Project, Grandin, Alloy, & Abramson (in press) examined the association between bipolarity and childhood stressful events occurring prior to or after bipolar individuals' age of onset (using the same cutoff age for demographically matched normal controls). Controlling for family history of mania and bipolar disorder, as well as depressive and hypomanic symptoms (to control for any reporting biases associated with mood state), Grandin et al. found that only independent stressful events occurring before the age of onset

were associated with bipolarity. The fact that bipolar participants reported more independent, but not dependent, events prior to the age of onset suggests that the association between bipolar disorder and increased childhood stressors is not attributable to bipolar individuals' behaviors and symptoms contributing causally to the occurrence of childhood stressors ("stress-generation"). An implication of these findings is that childhood stress may contribute to the emergence of bipolarity in vulnerable individuals. Thus, these initial findings from the developmentally relevant portion of the LIBS Project suggest that similar developmental precursors may be associated with vulnerability to both unipolar and bipolar mood disorders.

CONCLUSION

A fundamental question in the mood disorders area is the extent to which unipolar and bipolar forms of mood disorder share similar vulnerabilities, pathophysiologies, courses, and treatments. The work reviewed herein suggests that at least with respect to vulnerability factors, unipolar and bipolar mood disorders may indeed share similar cognitive precursors. Negative cognitive styles and information-processing biases are associated with both unipolar depression and bipolar disorder and initial evidence indicates that, alone and in combination with life events, these cognitive patterns predict prospective symptoms and episodes of both unipolar depression and bipolar disorder. In addition, similar parenting styles and childhood adversities may be predictive of vulnerability to both forms of mood disorder. Clearly, much more work is needed to more fully explore the role of cognitive vulnerability in both unipolar and bipolar mood disorders. However, the initial findings suggest that the way individuals appraise and process information about their life experiences, or their cognitive styles, may increase their risk for both unipolar and bipolar mood disorders.

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