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Cognitive Styles and Life Events Interact to Predict Bipolar and Unipolar Symptomatology

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This study examined the interaction of cognitive style (as assessed self-report and information-processing battery) and stressful life events in predicting the clinician-rated depressive and manic symptomatology of participants with Research Diagnostic Criteria lifetime diagnoses of bipolar disorder (n = 49), unipolar depression (n = 97), or no lifetime diagnosis (n = 23). Bipolar and unipolar participants' attributional styles, dysfunctional attitudes, and negative self-referent information processing as assessed at Time 1 interacted significantly with the number of negative life events that occurred between Times 1 and 2 to predict increases in depressive symptoms from Time 1 to Time 2. Within the bipolar group, participants' Time 1 attributional styles and dysfunctional attitudes interacted significantly, and their self-referent information processing interacted marginally, with intervening life events to predict increases in manic symptoms from Time 1 to Time 2. These findings provide support for the applicability of cognitive vulnerability–stress theories of depression to bipolar spectrum disorders.

The role of cognitive processes in the phenomenology, onset, course, and treatment of unipolar depression has been the subject of fruitful scientific investigation over the past two decades. Both Beck's (1967) theory and the hopelessness theory (Abramson, Metalsky, & Alloy, 1989; Alloy, Abramson, Metalsky, & Hartlage, 1988) are cognitive vulnerability–stress models of depression that view maladaptive cognitive patterns as vulnerabilities that heighten the risk both for becoming depressed and for experiencing increased severity and duration of depression when confronted with stressful life events. Whereas these theories have expanded our understanding of unipolar depression (e.g., Abramson, Alloy, & Metalsky, 1995; Alloy, Abramson, et al., 1999; Haaga, Dyck, & Ernst, 1991), little research has been done on the role of cognitive processes in the bipolar mood disorders. Presumably, the dearth of work on cognitive processes in bipolar disorders is due primarily to the long-standing conception that bipolar disorder is a biologically based illness. Although genetic and biological processes are undeniably salient in the etiology, course, and treatment of bipolar disorder (e.g., Goodwin & Jamison, 1990), biological factors cannot fully account for differences in the expression of the disorder, the timing and frequency of symptoms, or the effectiveness of prophylactic lithium usage (e.g., O'Connell, 1986; Prien & Potter, 1990).

Although psychosocial processes have largely been ignored in the study of bipolar mood disorders, suggestions that environmental factors play a part in the precipitation of manic and depressive episodes can be traced back to the pioneering work of Kraepelin (1921). A growing body of evidence suggests that stressful life events and environmental factors influence the onset and course of bipolar disorder (see Johnson & Roberts, 1995, for a review). Indeed, several studies using prospective designs have documented an association between life events and recurrence of manic and depressive episodes in bipolar patients (e.g., Ellicott, Hammen, Gitlin, Brown, & Jamison, 1990; Hunt, Bruce-Jones, & Silverstone, 1992). It is interesting that the Johnson and Roberts review indicated that negative life events preceded manic as well as depressive episodes among bipolar samples. In addition, investigators have found that negative family interactions and attitudes predict relapse rates in bipolar patients (Miklowitz, Goldstein, & Nuechterlein, 1995; Miklowitz, Goldstein, Nuechterlein, Snyder,

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& Mintz, 1988). Researchers have also examined the interaction of circadian rhythms and life stress in bipolar mood episodes (Ehlers, Frank, & Kupfer, 1988; Healy & Williams, 1988). Such models explore the impact of life stress on mood disorders through the destabilizing effects of life events on critical biological rhythms. Along these lines, Malkoff-Schwartz et al. (1998) recently reported that manic bipolars had significantly more preonset life events characterized by social rhythm disruption (e.g., change in sleep–wake cycle) than did depressed bipolars.

The cognitive theories of depression have sought to answer the question of why certain individuals are vulnerable to depression when faced with life stress. According to the attributional vulnerability component of the hopelessness and reformulated helplessness theories of depression (Abramson et al., 1989; Abramson, Seligman, & Teasdale, 1978; Alloy et al., 1988), individuals who tend to attribute negative events to internal (something about the self), stable (enduring), and global (general) causes are hypothesized to be more likely to experience onset of depression or a worsening of current depression when confronted with stressors than are individuals who do not exhibit this depressogenic attributional style. In Beck's (1967) cognitive model of depression, negative self-schemata organized around themes of failure, inadequacy, loss, and worthlessness serve as risk factors for the onset and exacerbation of depression that are activated by the occurrence of stressful life events. Such negative self-schemata are often represented as a set of dysfunctional attitudes or self-worth contingencies in which the person believes that his or her happiness and self-worth depend on being perfect or on others' approval. Consistent with cognitive science and social–cognition perspectives on the operation of self-schemata (e.g., Alba & Hasher, 1983; Taylor & Crocker, 1981), Beck (1967) hypothesized that depressive self-schemata influence the perception, interpretation, and memory of personally relevant experiences, with the result being a negatively biased construal of one's personal world. When activated by the occurrence of stressful life events, depressive self-schemata lead to the onset or exacerbation of depressive symptoms through their effect on preferential encoding and retrieval of negative self-referent information.

Although the cognitive theories have been tested almost exclusively with respect to unipolar depression, recent findings suggest that they may be applicable to bipolar conditions as well. For example, consistent with the hopelessness and reformulated helplessness theories, Alloy, Reilly-Harrington, Fresco, Whitehouse, and Zechmeister (1999) found that attributional styles interacted with the occurrence of intervening life events to predict prospectively increases in depressive and hypomanic symptoms in participants with subsyndromal bipolar and unipolar conditions. In accord with Beck's theory, Hammen, Ellicott, and Gitlin (1992) found that self-schemata in the interpersonal domain (i.e., sociotropy) interacted with interpersonal life events to predict subsequent symptom severity among bipolar patients. In contrast, in a small sample study, Hammen, Ellicott, Gitlin, and Jamison (1989) found support for the cognitive diathesis–stress model only for unipolar, but not bipolar, patients. More generally, Swendsen, Hammen, Heller, and Gitlin (1995) reported that the personality traits of introversion and obsessionality, which themselves may be associated with negative cognitive styles (Abramson et al., 1998), interacted with stressful life events to predict relapse in bipolar patients.

One methodological feature of the few prior studies that tested the cognitive vulnerability–stress hypothesis for bipolar disorders is that they relied solely on self-report measures of cognitive styles, self-schemata, or personality. An alternative approach for assessing cognitive vulnerability involves information-processing paradigms from cognitive psychology modified for use with emotion-relevant stimuli (e.g., Derry & Kuiper, 1981; Gotlib & McCann, 1984; Greenberg & Alloy, 1989; Segal & Vella, 1990). Segal (1988) argued that self-report questionnaires may not be optimal for assessing cognitive vulnerability as represented by self-schemata, in part, because they may reflect fluctuations in negative verbalizations rather than underlying cognitive structure. However, Alloy, Abramson, Murray, Whitehouse, and Hogan (1997) supported the construct validity of both self-report and information-processing measures of cognitive vulnerability. Alloy et al. (1997) found that cognitive styles assessed by self-report questionnaires (Dysfunctional Attitudes Scale [DAS] and a revised Attributional Style Questionnaire [ASQ]) among nondepressed individuals at high versus low cognitive risk for depression were associated with actual differences in information processing as measured by a Self-Referent Information Processing (SRIP) Task Battery adapted from cognitive psychology. Moreover, both the self-report measures of cognitive styles and the SRIP Battery predicted future onsets of diagnosed depressive episodes over a 2.5-year follow-up period (Alloy, Abramson, et al., 1999). Thus, as an extension of prior studies, the major goal of the present study was to examine whether the interaction of stressful life events and the cognitive styles featured as vulnerabilities in hopelessness and Beck's theories, as assessed by both self-report and self-referent information-processing tasks, would predict increases in depressive and manic symptoms among individuals with unipolar and bipolar mood disorders.

A second characteristic feature of prior studies (with the exception of Alloy, Reilly-Harrington, et al., 1999) that test the cognitive vulnerability–stress hypothesis for bipolar disorders is that they used treated patient samples drawn from clinic or hospital settings. In contrast, in the current study, participants were undergraduates who, despite meeting diagnostic criteria for bipolar or unipolar disorders based on structured diagnostic interview, for the most part had received no prior or current treatment for their mood disorders. Treatment with medication or psychotherapy may not only remediate symptoms in unipolar or bipolar individuals, but it may ameliorate, deactivate, or otherwise reduce the likelihood of reporting cognitive vulnerability as well (Alloy, Reilly-Harrington, et al., 1999). The fact that our young student sample was largely untreated is consistent with the results of several studies that have reported long delays between onset of symptoms and treatment seeking in individuals with bipolar and unipolar mood disorders (e.g., Goodwin & Jamison, 1990). For example, Lish, Dime-Meenan, Whybrow, Price, and Hirschfeld (1994) found that 50% of their bipolar sample received no treatment for 5 years after the onset of symptoms. Frequently, such untreated individuals do not receive research attention. Thus, the current study permitted a rare, naturalistic examination of young, largely untreated individuals early in the course of their bipolar and unipolar mood disorders, whose characteristic cognitive patterns and information processing had not been altered by the effects of medication or psychotherapy.

A secondary goal of this study was to examine the cognitive patterns associated with bipolar mood disorders. Little work has

directly compared the cognitive styles or information processing of individuals with bipolar and unipolar disorders. On the basis of the grandiosity that is typically part of the clinical picture of mania and hypomania, one might expect bipolar individuals (who experience manic and hypomanic episodes) to exhibit cognitive styles more positive than those of unipolar depressive individuals. On the other hand, three prior studies reported similarities between the cognitive patterns of bipolar and unipolar depressive persons, suggesting that bipolar depressive individuals may exhibit negative cognitive styles like those of unipolar depressive persons. For example, Winters and Neale (1985) found that although remitted bipolar patients reported higher self-esteem than remitted unipolar patients and normal control participants on self-report measures, they generated causal inferences as negative as those of remitted unipolar patients on a pragmatic inference task. Hollon, Kendall, and Lumry (1986) reported that both depressed unipolar and bipolar patients exhibited similar dysfunctional attitudes and negative automatic thoughts that were more negative than those of normal control participants. Finally, Alloy, Reilly-Harrington, et al. (1999) found that cyclothymic participants' attributional styles and dysfunctional attitudes were as negative as those of dysthymic participants and more negative than those of hypomanic and normal control participants.

In sum, the overarching purpose of the present study was to further explore the applicability of the cognitive theories of unipolar depression to the bipolar spectrum. To this end, we compared the attributional styles, dysfunctional attitudes, and self-referent information processing of individuals with Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) lifetime diagnoses of bipolar disorder (bipolar I, bipolar II, or cyclothymia), unipolar depression (major, minor, or intermittent depression) and no lifetime diagnosis. In addition, using a longitudinal design, we examined the interaction of Time 1 cognitive style (as assessed by means of self-report and information-processing battery) and intervening life events in predicting increases in bipolar and unipolar participants' clinician-rated depressive and manic symptomatology at Time 2. Consistent with hopelessness (Abramson et al., 1989) and Beck's (1967) theories, it was hypothesized that individuals with negative cognitive styles and self-referent processing who experienced intervening negative events would show an increase in depressive symptoms at Time 2. We explored whether the same Cognitive Style × Stress interactions would also predict increases in manic symptoms over time.

Method

Participants

Participants were selected on the basis of a two-stage screening process. In Stage 1, 3,000 Northwestern University undergraduates were screened with the General Behavior Inventory (GBI; Depue, Krauss, Spoont, & Arbisi, 1989) and the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979). Participants who met the established cutoffs on either of these instruments (see the measures sections) and a small, random subset of those who scored in the normal range on both of them were invited for Stage 2. In Stage 2, which occurred from 2 weeks to 4 months after Stage 1, participants were administered a Schedule for Affective Disorders and Schizophrenia–Lifetime Version (SADS-L; Endicott & Spitzer, 1978) diagnostic interview. Those who met RDC for a lifetime diagnosis of a bipolar mood disorder (bipolar I, bipolar II, or cyclothymia) or a unipolar mood disorder (major, minor, or intermittent depression), or who did not meet criteria for any disorder in their lifetime (normal control participants), were asked to participate in a short-term longitudinal study.

The final sample consisted of 169 participants (64 men, 105 women; 113 Caucasian, 10 African American, 4 Hispanic, 13 Asian, 29 not reported; *M* age = 20.04, *SD* = 3.95). Of the 169 participants, 97 had a lifetime diagnosis of unipolar mood disorder (88 major, 6 minor, and 3 intermittent depression), 49 had a lifetime diagnosis of bipolar mood disorder (13 bipolar I, 35 bipolar II, and 1 cyclothymia), and 23 were normal control participants. At Time 1 of the study, on the basis of the SADS-L, 7 of the 49 bipolar participants were in a depressed episode, 5 were in a manic or hypomanic episode, and 37 were not currently in episode. Of the 97 unipolar participants, 31 were in a depressed episode and 66 were not in episode at Time 1. The sample was largely untreated, with 4 bipolar and 4 unipolar participants receiving medication or psychotherapy. Table 1 presents the demographic characteristics of the three lifetime diagnostic groups. As can be seen in Table 1, the groups did not differ on age, $F(2, 157) = 2.18$, *ns*;[1] sex, $\chi^2(2, N = 169) = 1.54$, *ns*; or ethnic distribution, $\chi^2(8, N = 169) = 6.13$, *ns*.

Screening Measures

Stage I: Self-report inventories. The GBI (Depue et al., 1981, 1989) is a self-report questionnaire developed to identify bipolar conditions on a lifetime or trait basis. For bipolar conditions, the GBI has high positive (.94) and negative (.99) predictive power, adequate sensitivity (.78), and high specificity (.99; Depue et al., 1989). Depue et al. (1981) reported a 98% positive concordance rate between the GBI cyclothymia score and blind, structured diagnostic interview. Both internal reliability (αs = .90–.96) and test–retest reliability (*r*s = .71–.74) are excellent. Furthermore, the GBI has been extensively validated in a range of populations, including college, psychiatric outpatient, and offspring of bipolar I samples (Depue et al., 1989). Four groups can be identified by the two subscales of the GBI (Depression [D] items and Hypomania plus Biphasic [HB] items): (a) dysthymia (high scores on the D scale and low scores on the HB scale); (b) cyclothymia (high D and high HB scores); (c) hypomania (low D and high HB scores); and (d) noncases or normal participants (low D and low HB scores). Depue et al. (1989) recommended using a case scoring method, in which only items rated a 3 (*often*) or 4 (*very often or almost constantly*) on the GBI 4-point frequency scale contribute toward total D or HB scores. In this way, only symptoms meeting the criteria of duration, intensity, and frequency are counted toward total scores. In the present study, as recommended by Depue et al. (1989), individuals who scored high (in the top 8% of the Stage 1 screening sample) on either the HB scale (≥13) or D scale (≥11) and a small, random subset of those who scored below these cutpoints on both the HB and D scales were invited to participate in Stage 2 of the screening process.

The BDI (Beck et al., 1979) is a 21-item self-report inventory that assesses the presence and severity of cognitive, motivational, affective, and somatic symptoms of depression. Internal reliability for the BDI in a nonclinical population is good (α = .81) and the test–retest reliability in several studies has also been high (Beck, Steer, & Garbin, 1988). The BDI has been found to be valid for mildly depressed student samples (e.g., Beck et al., 1988; Bumberry, Oliver, & McClure, 1978; Hammen, 1980). Participants who scored in the mildly depressed range or higher (BDI ≥ 10) according to established BDI cutpoints (Kendall, Hollon, Beck, Hammen, & Ingram, 1987) were invited to participate in Stage 2 of screening.

Stage 2: Diagnostic interview. The SADS-L (Endicott & Spitzer, 1978) is a semi-structured diagnostic interview that probes for the occurrence, duration, and severity of symptoms related to mood, psychotic, anxiety, substance abuse, and other disorders over the lifetime.

[1] Degrees of freedom vary across different analyses throughout the article because of missing data.

Table 1
Sample Demographic Characteristics as a Function of Lifetime Diagnosis

Demographic variable	Bipolar (n = 49)	Unipolar (n = 97)	Normal control (n = 23)
Age			
M	19.24	20.59	19.38
SD	1.98	4.88	1.53
Sex (%)			
Men	33	38	48
Women	67	62	52
Ethnicity (%)			
Caucasian	70	68	57
African American	6	4	13
Hispanic	4	1	4
Asian	8	8	4
Not reported	12	19	22

The SADS-L was used at Stage 2 of the screening procedure to identify those participants who met either current or lifetime RDC for a bipolar or unipolar mood disorder or who did not meet criteria for any lifetime disorder (normal control participants). The SADS-L also yielded depression and mania symptom severity scores (described below) at Time 1.

Interrater reliabilities for RDC diagnoses based on the SADS-L were calculated by means of the kappa (κ) statistic based on joint ratings of 60 interviews. For major and minor depressive disorders, there was 100% agreement (κ = 1.00) between interviewers for current episodes and 96% agreement (κ = .89) for past episodes. There was 100% agreement (κ = 1.00) for current and 90% agreement (κ = .81) for past periods of intermittent depressive disorder. For manic and hypomanic episodes, interrater agreements were 97% for current episodes (κ = .90) and 82% for past episodes (κ = .75). Kappas for other disorders ranged from .67 to 1.00. Finally, interrater agreement was 93% for *not currently mentally ill* (κ = .87) and 91% for *not previously mentally ill* (κ = .84).

Measures Administered at Times 1 and 2

Diagnostic interview. The SADS–Change Version (SADS-C; Spitzer & Endicott, 1978) is a semistructured diagnostic interview that probes for the presence and severity of symptoms related to mood, psychotic, anxiety, substance abuse, and other disorders since the last interview. The SADS-C was given at Time 2 to assess any occurrences of depressive and manic symptoms since the time of the SADS-L interview (Time 1). The average time between the SADS-L and SADS-C interviews was approximately 1 month. Depression and mania symptom composite scores were created from the SADS-L and SADS-C by summing the severity ratings (0–5) of individual symptom items for depression and mania, respectively. Across the SADS-L and SADS-C, corresponding items were used to create the depression and mania symptom composite scores, respectively.[2]

High levels of interrater reliability were achieved using the SADS-C in this study. Average kappas based on 60 jointly rated interviews were $\geq$ .80. We also examined the interrater reliability (Pearson r) of the severity ratings on individual symptoms that were included in the depression and mania symptom composite scores. The interrater correlations ranged from .75 (somatic concern) to .95 (sadness, suicidal ideation, loss of interest, insomnia, brooding) for depressive symptoms and from .62 (grandiosity) to .98 (decreased need for sleep) for manic symptoms. Internal consistencies for the symptom composites derived from the SADS-L (αs = .97 and .89 for depression and mania, respectively) and SADS-C (αs = .81 and .73 for depression and mania, respectively) also were quite good.

The SADS-L and SADS-C interviews were conducted by graduate research assistants who received approximately 100 hr of instruction on diagnostic interviewing, RDC, and decision rules before administering any interviews. The training program consisted of didactic instruction, role-plays, videotapes of simulated interviews, and practice interviews.

Cognitive style measures. The original ASQ (Peterson et al., 1982; Seligman, Abramson, Semmel, & von Baeyer, 1979) is a self-report inventory that assesses attributions for 6 positive and 6 negative hypothetical events. Respondents report the major cause for each of the 12 events and then rate this cause on the internality, stability, and globality dimensions on 7-point Likert scales. Positive and negative event composite scores can be derived by summing across the internality, stability, and globality dimensions for the 6 positive and 6 negative events separately. The composite scores provide greater reliability than do the individual subscales (Peterson et al., 1982). Moreover, we obtained a fairly high intercorrelation (r = .50) of the Stability and Globality subscales of the ASQ. Thus, the ASQ composite scores served as the measures of cognitive vulnerability used to test the hopelessness theory in the present study. Peterson et al. reported acceptable internal consistency (αs = .75 and .72) and test–retest reliability (rs = .70 and .64) for the ASQ positive and negative composite scores, respectively. The ASQ has also demonstrated substantial validity as a predictor of depressive symptoms and episodes alone and in interaction with stress (Abramson et al., 1995). The ASQ was administered at Times 1 and 2.

The DAS (Weissman & Beck, 1978) is a 40-item self-report inventory that serves as a measure of the cognitive vulnerability component in Beck's theory. This scale assesses an individual's endorsement of maladaptive attitudes such as "I am nothing if a person I love doesn't love me" (Weissman & Beck, 1978). Participants rate their endorsement of these statements on a 7-point Degree of Belief scale, ranging from 1 (*totally disagree*) to 7 (*totally agree*). The scores for positive items are reversed, and the total severity score is the sum of all items. Weissman and Beck reported high internal consistency (α = .93) and also a strong positive correlation between DAS and BDI scores (r = .65). The DAS has also demonstrated good retest reliability and validity in student and patient samples (Hammen & Krantz, 1985). The DAS was administered at Times 1 and 2.

Self-referent information processing. The SRIP (Alloy et al., 1997; Greenberg & Alloy, 1989) measures the processing component of the self-schemata featured as vulnerabilities for depression in Beck's theory. The SRIP Task Battery consists of four tasks that assess five information-processing effects adapted from basic research in cognitive and social psychology on the measurement of schemata: (a) judgments of self-descriptiveness of trait words, (b) response times (RTs) for these judgments, (c) past behavior descriptions, (d) future behavior predictions, and (e) free recall of the trait words. Each of the tasks uses four types of stimulus content, matched on word length and frequency and on social desirability: negative depression-relevant (NDR; e.g., incompetent, worthless); positive depression-relevant (PDR; e.g., efficient, successful); negative depression-irrelevant (NDI; e.g., rude, unstable); and positive depression-irrelevant (PDI; e.g., polite, predictable) content. Alloy et al. (1997) developed two equivalent forms (A and B) of the SRIP Task Battery to guard against practice effects from repeated administrations. In the present study, αs for the tasks of the SRIP

[2] The SADS-L and SADS-C interviews were chosen for this study because they allowed assessment of change in depressive and manic–hypomanic symptoms over time with corresponding interviews (Spitzer & Endicott, 1978), whereas other diagnostic interviews (e.g., the SCID) did not have appropriate change versions.

Battery range from .64 to .93 and retest stabilities for the 4-week interval between Times 1 and 2 range from r = .68 to .79. Also, the tasks do tend to converge and form a battery, with intertask correlations ranging from r = .35 to .83 (most are > .50). We used standardized (z) scores based on all five dependent measures from the four tasks for the NDR and PDR stimulus content as the self-referent processing predictors in the vulnerability–stress analyses. Prior findings with the SRIP Battery indicate that the various tasks distinguish depressed from anxious and normal participants (e.g., Derry & Kuiper, 1981; Greenberg & Alloy, 1989; Greenberg & Beck, 1989) and nondepressed cognitively vulnerable from nondepressed cognitively invulnerable participants (Alloy et al., 1997). The following four tasks were completed at both Times 1 and 2 (see Alloy et al., 1997, for more details).

The first task assessed participants' judgments of self-descriptiveness of trait adjectives and RTs for these judgments. A total of 40 trait adjectives were presented to participants on a Macintosh computer set up to record RTs. The 40 trait adjectives consisted of 12 NDR, 12 PDR, 8 NDI, and 8 PDI words. Words were presented with an interstimulus interval of 4 s, and each remained on the computer monitor until the participant responded. Participants responded by pressing a "Me" button if the adjective was self-descriptive or a "Not Me" button if the adjective was not self-descriptive, counterbalanced so that one half of the participants had the "Me" button on the right side (/ key) and one half had it on the left side (Z key). RTs were recorded without participants' awareness. The trait adjectives were presented in a different random order for each participant. Internal (α) and test–retest (r) reliabilities were .93 and .79, respectively, for PDR adjectives, and .90 and .68, respectively, for NDR items. For RTs, internal (α) and test–retest (r) reliabilities were .76 and .64, respectively, for PDR adjectives and .66 and .59, respectively, for NDR adjectives. The proportion of words judged "Me" and average RT for "Me" words of each stimulus type served as dependent measures.

In Task 2, participants received three adjectives, chosen at random, from each of the four stimulus types (NDR, PDR, NDI, PDI). They were instructed to circle the adjectives that were self-descriptive and give specific examples from their own past behavior to indicate why a particular adjective was self-descriptive. For example, if a participant believed he or she was incompetent, he or she had to provide specific examples of past incompetent behaviors in which he or she had engaged. Alphas were .76 and .66 for PDR and NDR adjectives, respectively. The mean number of examples provided per word of each stimulus type was the dependent measure.

In Task 3, participants read 24 statements describing six hypothetical behaviors of each of the four stimulus types (NDR, PDR, NDI, PDI), for example, "You give an in-class presentation and communicate your ideas clearly" (PDR). Participants judged the probability (0–100%) that they would behave in the way described if they were in that situation in the future. Alphas were .60 and .68 for the PDR and NDR items, respectively. The dependent measure was the mean probability judgment for each content type.

Finally, Task 4 involved an incidental free-recall test for the adjectives judged as "Me" or "Not Me" in Task 1, administered 24 hr after Task 1. Participants were given 5 min to recall in any order as many of the words they had seen on the computer in Task 1 as they could. Alphas were .85 and .82 for NDR and PDR adjectives, respectively. The dependent measure was the proportion of words of each stimulus type correctly recalled.

Stress/life events measures. The Life Experiences Survey (LES; Sarason, Johnson, & Siegel, 1978) consists of 57 mostly negative events, with three blank spaces provided for write-in events. LES events that represented obvious symptoms of depression or mania (e.g., sexual difficulties, major change in sleeping habits) were not included in final life events scores. Participants were instructed to circle or write in events that happened to them during the past month, to provide the date on which the event occurred, and to indicate the type and extent of the impact the event had on their life. Impact was rated on a scale ranging from −3 (*extremely negative*) to 3 (*extremely positive*). The LES possesses good test–retest reliability (rs = .53–.88), is not contaminated by social desirability biases, and predicts a number of stress-related dependent measures, including maladjustment (Sarason, Johnson, & Siegel, 1978). Also, in interaction with attributional style, the LES prospectively predicted both depressive and hypomanic symptoms in students with subsyndromal bipolar and unipolar mood disorders (Alloy, Reilly-Harrington, et al., 1999). To decrease confounding of the stress variable by participants' cognitive styles or moods, we used the total number of negative events from the LES as our life events score, irrespective of participants' ratings of the impact of the events. The LES was completed at both Times 1 and 2.

The events on the LES were classified as major (e.g., death of close family member, marriage) or minor (e.g., failing an important exam, minor law violation) and independent versus dependent on participants' behavior based on consensus by a group of four raters unaware of participants' LES scores and diagnostic group. Events were classified for dependence along a 7-point scale, with a rating of 1 assigned to events totally independent of one's control (e.g., death of close family member) and higher ratings assigned to events to which an individual's own actions might contribute (e.g., being fired from job). The dependence score for each LES event was then calculated by averaging the scores given by the four raters. This dependence rating resembles that used in interview-based measures such as the Bedford College Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978).

Procedure

Participants in the final sample were told that the study would consist of two sessions on 2 consecutive days at each of two time points 1 month apart (Times 1 and 2). The sessions were described as consisting of several mood and personality questionnaires, an interview, and some cognitive tasks requiring self-judgments, predictions, and recall of past experiences. Participants were assured that all feelings, thoughts, and information provided would remain strictly confidential. Experimenters for the Time 1 and Time 2 sessions were unaware of participants' lifetime diagnoses and current mood states.

Participants were then given three self-report inventories with instructions (DAS, ASQ, LES) to complete before returning the next day. When participants returned on the next day, experimenters administered the Judgment and Latency task (Task 1) of the SRIP Battery Form A. Participants were given the following instructions:

> During this task, words will be presented one at a time at the center of the screen. The words are adjectives that people use to describe themselves. What I'd like you to do is decide whether the word describes you or not. If the word does describe you, then you should press the "Me" key over here [pointed to it]. If the word doesn't describe you, then you should press the "Not Me" key over here [pointed to it].

Next, participants were given the Behavioral Descriptions and then the Behavioral Predictions tasks of the SRIP Battery. Participants were then reminded to return the following day for the final portion of Time 1, the Surprise Recall task of the SRIP Battery. For this task, participants were instructed to try to remember and write down in any order the words they had seen on the computer screen the previous day.

Participants were called in 1 month and asked to return for the Time 2 assessment. Essentially, the Time 1 and 2 assessments were the same, except that participants were given the SADS-C interview at Time 2 to assess the presence and severity of symptoms since Time 1. In addition to the SADS-C, the Time 2 assessment consisted of all of the self-report inventories (DAS, ASQ, LES). Also, Form B of the SRIP Battery was given at Time 2 to control for practice effects. Participants were given $5

Table 2
Study Variables as a Function of Lifetime Diagnosis

Measure	Bipolar (n = 49)	Unipolar (n = 97)	Normal control (n = 23)	F	dfs
SADS-L depression				3.78*	2, 141
M	11.05_a	13.52_a	1.60_b		
SD	18.43	18.85	0.82		
SADS-C depression				8.44**	2, 136
M	17.43_a	13.35_b	7.70_c		
SD	10.23	8.60	5.14		
SADS-L mania				7.18**	2, 142
M	2.85_a	1.01_b	0.19_b		
SD	4.98	1.86	0.60		
SADS-C mania				0.82	2, 136
M	4.58	3.86	3.10		
SD	5.30	3.89	3.77		
ASQ-NC (mean item score)				1.88	2, 165
M	4.30	4.13	4.02		
SD	0.55	0.65	0.66		
ASQ-PC (mean item score)				0.59	2, 151
M	5.16	5.23	5.36		
SD	0.65	0.70	0.70		
DAS (total score)				1.81	2, 153
M	138.23	132.69	120.36		
SD	37.76	33.94	40.41		
SRIP-NC (z score)				0.32	2, 155
M	0.09	0.01	0.07		
SD	0.65	0.57	0.61		
SRIP-PC (z score)				0.60	2, 158
M	0.03	0.06	0.20		
SD	0.63	0.65	0.51		
NEGEV				3.77*	2, 166
M	5.16_a	4.34	2.44_b		
SD	3.45	4.48	1.81		

Note. SADS-L = Schedule for Affective Disorders and Schizophrenia–Lifetime diagnostic interview; SADS-C = Schedule for Affective Disorders and Schizophrenia–Change diagnostic interview; depression = symptom severity score for depression items; mania = symptom severity score for mania items; ASQ-NC = Time 1 Attributional Style Questionnaire composite for negative events; ASQ-PC = Time 1 Attributional Style Questionnaire composite for positive events; DAS = Time 1 Dysfunctional Attitudes Scale; SRIP-NC = Time 1 Self-Referent Information Processing Task Battery composite for negative depression-relevant stimuli; SRIP-PC = Time 1 Self-Referent Information Processing Task Battery composite for positive depression-relevant stimuli; NEGEV = Time 2 Life Experiences Survey total number of negative events. Means in the same row with differing subscripts differ significantly ($p < .05$) from each other.
* $p < .05$. ** $p < .001$.

and five experimental credits for their participation at Time 1 and were paid $20 at the end of the Time 2 session.

Results

Group Comparisons on Study Variables

Table 2 presents the means, standard deviations, and analysis of variance (ANOVA) statistics (Fs and p values) for comparisons among the three diagnostic groups on all study variables. As can be seen in Table 2, ANOVAs indicated that the groups differed on both the SADS-L depression and mania symptom composites at Time 1 and the SADS-C depression symptom composite at Time 2. Post hoc pairwise comparisons of means were conducted with Tukey's honestly significant difference tests. At Time 1, the unipolar depressed group had higher SADS-L depression scores than did the normal control group ($p < .02$). The bipolar group did not differ from the unipolar depressive group and showed a trend ($p < .11$) for higher depression scores than the normal control group. Also at Time 1, the bipolar group had higher SADS-L mania scores than the unipolar and normal groups (ps $< .01$), which did not differ from one another. At Time 2, the bipolar group had higher SADS-C depression scores than the unipolar group ($p < .05$) which, in turn, had higher scores than the normal control group ($p < .03$). There were no group differences on SADS-C mania symptom scores at Time 2.

There were no group differences at Time 1 in attributional style for negative or positive events, dysfunctional attitudes, or self-referent information processing for negative or positive DR stim-

uli[3] (see Table 2). To explore the adequacy of the cognitive vulnerability measures in this sample, we also conducted Sex × Time 1 Depression Status (depressed, nondepressed) ANOVAs to determine whether currently depressed versus nondepressed participants at Time 1 differed on the cognitive style measures.[4] Participants who were in a depressed episode at Time 1 had more internal, stable, global attributional styles for negative events, $F(1, 159) = 14.61$, $p < .001$; less internal, stable, global attributional styles for positive events, $F(1, 145) = 6.95$, $p < .01$; and more negative SRIP composite scores for negative DR stimuli, $F(1, 150) = 2.66$, $p < .05$, one-tailed, than did participants who were not depressed at Time 1. DAS scores and SRIP composite scores for positive DR stimuli did not differ as a function of depression status at Time 1. There were also no sex differences on any of the cognitive style measures.

Table 2 shows that there were lifetime diagnostic group differences on the number of negative life events reported at Time 2. The bipolar group reported more stressful events than did the normal control group ($p < .02$); the unipolar group did not differ from either the bipolar or normal group. In order to explore the possible basis of the bipolar group's report of more life events, we conducted several secondary analyses. Inasmuch as major events (e.g., death of close family member, marriage) are likely to be reported by most participants and are less subject to reporting biases and recall errors than minor events (e.g., minor law violation), we reasoned that a reporting bias explanation would be most plausible only if the bipolar group reported more minor events than the normal control group. In fact, the groups differed significantly on the number of minor events, $F(2, 166) = 3.31$, $p < .05$, with the bipolar group reporting more minor events than the normal group ($p < .01$). It is important, however, that the groups also differed on the number of major events, $F(2, 166) = 5.73$, $p < .001$, with the bipolar group again reporting more major events than the normal group ($p < .01$). Overall, the most frequently endorsed LES events at Time 2 were failing a course (28 participants), breakup of a romantic relationship (20), trouble with employer (20), major change in living conditions (18), major change in closeness with family (17), financial problems with school (15), and fired from job (15).

A stress-generation explanation, in which individuals with mood disorders actually contribute to the occurrence of negative events in their lives through their own behavior (e.g., Hammen, 1991; Monroe & Simons, 1991), would suggest that bipolar participants would have higher event dependence scores than the normal control participants. However, they did not; there were no group differences on event dependence, $F(2, 166) = 0.90$, *ns*. Thus, although not definitive, these secondary analyses suggest that bipolar participants' higher number of negative events may not be primarily due to reporting biases or stress generation; rather, they may have actually experienced more, relatively independent negative events.

Cognitive Vulnerability–Stress Prediction of Depressive and Manic Symptoms

The main study analyses focused on the prediction of changes in depressive and manic symptoms from Time 1 to Time 2 as a function of cognitive styles (ASQ, DAS, SRIP) at Time 1 and intervening life events between Time 1 and Time 2 (assessed at Time 2). To investigate the cognitive vulnerability–stress predictions, we conducted a series of hierarchical regression analyses, analyses of partial variance (Cohen & Cohen, 1983) on residualized change in SADS depression and mania scores from Time 1 to Time 2. A sample full model regression equation follows, with each successive predictor entered at separate steps: Residualized change in SADS depression = Constant + Time 1 Cognitive Style score (ASQ—negative composite, DAS, or SRIP—negative composite) + Intervening Negative Events score (Time 2) + Time 1 Cognitive Style × Time 2 Negative Events. According to the cognitive theories, the interaction term should be a significant predictor of depressive and manic symptom increases, even after controlling for the separate effects of cognitive style and negative events. The normal control participants were excluded from these hierarchical regression analyses. To guard against any treatment effects, we also excluded the 8 participants (4 bipolar, 4 unipolar) who were receiving medication or psychotherapy from the regression analyses.[5] Tables 3 and 4 summarize the results of the three hierarchical regression analyses (one each with the ASQ, DAS, and SRIP scores as the cognitive style measure) for predicting residualized change in SADS depression scores and SADS mania scores, respectively. These tables display the total multiple correlation squared (R^2) accounted for by the complete model as well as the partial correlation, significance (t and p values), and change in R^2 accounted for by each predictor entered at successive steps of the model.

The ASQ—negative composite at Time 1 in interaction with Time 2 negative events significantly predicted change in SADS depression scores (Cohen's $f^2 = .04$), accounting for a unique 4.2% of the variance in depression change after controlling for the main effects of attributional style and life events (Table 3, top). The form of the interaction conformed to prediction. Consistent with the hopelessness theory, only bipolar and unipolar participants with a depressive attributional style for negative events (internal, stable, global) who also experienced high stress (many negative events) showed an increase in SADS depression scores

[3] Although no significant group differences were obtained on the SRIP negative and positive composites used in the cognitive vulnerability–stress analyses (see Table 2), there was some evidence of group differences in self-referent predictions about the future and self-descriptiveness judgments. A Group (bipolar, unipolar, normal) × Sex (male, female) × Content (depression-relevant, depression-irrelevant) × Valence (negative, positive) ANOVA on participants' behavioral predictions (Task 3 of the SRIP Battery) yielded a significant Group × Valence interaction, $F(2, 130) = 3.33$, $p < .05$. Bipolar participants predicted that they would be more likely to behave in negative DR ways and less likely to behave in positive DR ways in the future than did unipolar and normal participants. The Group × Content × Valence interaction was marginally significant, $F(2, 130) = 2.32$, $p < .07$, for participants' self-descriptiveness judgments (Task 1). Bipolar participants were more likely to say "Me" to negative DR adjectives and less likely to say "Me" to positive DR adjectives than were the unipolar and normal participants.

[4] We excluded the 5 bipolar participants who were in a manic or hypomanic episode at Time 1 from these analyses because there were too few of them to include as a separate current episode group.

[5] The results of all of the regression analyses testing the cognitive vulnerability–stress prediction of depressive and manic symptom changes shown in Tables 3 and 4 remained the same when these 8 treated participants were included in the analyses.

Table 3
Hierarchical Multiple Regression Analyses to Predict Change in Clinician-Rated (SADS) Depression in Bipolar and Unipolar Participants

Step/predictor	Beta	*pr*	*t*	*df*	Total R^2	R^2 change
1 ASQ-NC	2.60	.22	2.37*	109	.05	.05
2 NEGEV	0.15	.10	1.05	108	.06	.01
3 ASQ-NC × NEGEV	0.60	.21	2.24*	107	.10	.04
1 DAS	0.05	.24	2.48*	101	.06	.06
2 NEGEV	0.25	.16	1.62	100	.08	.02
3 DAS × NEGEV	0.02	.33	3.46***	99	.18	.10
1 SRIP-NC	2.89	.25	2.63**	100	.06	.06
2 NEGEV	0.16	.11	1.13	99	.08	.02
3 SRIP-NC × NEGEV	0.95	.36	3.77***	98	.20	.12

Note. SADS = Schedule for Affective Disorders and Schizophrenia; ASQ-NC = Time 1 Attributional Style Questionnaire composite for negative events; NEGEV = Time 2 Life Experiences Survey total number of negative events; DAS = Time 1 Dysfunctional Attitudes Scale; SRIP-NC = Time 1 Self-Referent Information Processing Task Battery composite for negative depression-relevant stimuli.
* $p < .05$. ** $p < .01$. *** $p < .001$.

from Time 1 to Time 2. Beck's model was also supported for change in depressive symptoms. Time 1 DAS scores interacted with Time 2 negative events to significantly predict change in SADS depression scores (Cohen's $f^2 = .12$), accounting for a unique 9.9% of the variance in depression change after controlling for the separate effects of dysfunctional attitudes and negative events (Table 3, middle). As predicted, only bipolar and unipolar participants with more dysfunctional attitudes and a high number of negative life events experienced increases in depressive symptoms from Time 1 to Time 2. Similarly, the Time 1 self-referent processing (SRIP) measure of self-schemata also predicted change in SADS depression significantly in interaction with Time 2 negative events (Cohen's $f^2 = .15$), accounting for a unique 11.7% of the variance in depression symptom change, after controlling for the separate effects of self-referent processing and negative events (Table 3, bottom). Again, only bipolar and unipolar individuals who exhibited preferential processing of negative, depression-relevant information about themselves and experienced many negative events had an increase in depressive symptoms from Time 1 to Time 2.[6] Attributional style for positive events and self-referent information processing of positive stimuli did not predict depressive symptom change in interaction with negative or positive events.

We tested the cognitive vulnerability–stress predictions of change in manic symptoms in the bipolar group only, excluding the unipolar depressed subsample from the hierarchical regression analyses. By definition, unipolar depressive individuals do not experience episodes of mania or hypomania. Within the bipolar group, the interaction of attributional style for negative events at Time 1 and the number of negative events at Time 2 significantly predicted a unique 10% of the variance in change in SADS mania scores from Time 1 to Time 2 (Cohen's $f^2 = .12$), after controlling for the main effects of attributional style and stressful events (Table 4, top). Consistent with an extension of the hopelessness theory, bipolar participants with a negative attributional style for negative events (internal, stable, global) and a high number of negative events were the only individuals to experience an increase in manic symptoms over time. Similarly, the measures of cognitive vulnerability featured in Beck's theory predicted manic symptom change in interaction with intervening negative life events. The Time 1 DAS × Time 2 Negative Events interaction accounted for a unique 16.4% of the variance in SADS mania symptom change (Cohen's $f^2 = .23$), after controlling for the separate effects of dysfunctional attitudes and negative events (Table 4, middle). Bipolar participants who had dysfunctional attitudes and experienced many negative events showed greater increases in manic symptoms than those with dysfunctional attitudes and few negative events or those with adaptive attitudes and either high or low stress. Likewise, the Time 1 SRIP—Negative Composite × Time 2 Negative Events interaction marginally ($p < .085$) predicted change in manic symptoms, accounting for a unique 8.2% of the variance in manic symptom change (Cohen's $f^2 = .09$), after the separate effects of self-referent processing and negative events were controlled (Table 4, bottom). Bipolar participants who processed information about themselves negatively and who experienced many negative life events were the only individuals to show increases in manic symptoms over time. Attributional style for positive events and self-referent processing of positive stimuli did not predict manic symptom change either alone or in interaction with negative or positive LES events.

Discussion

The main findings of the present study were twofold. First, as directly predicted by the cognitive theories of depression (Abramson et al., 1989; Alloy et al., 1988; Beck, 1967), unipolar and bipolar participants' cognitive styles at Time 1, as assessed by both self-report and information-processing tasks, interacted significantly with subsequently occurring negative life events to prospectively predict increases in clinician-rated depressive symptoms

[6] All three of the Cognitive Vulnerability (ASQ—negative composite, DAS, SRIP—negative composite) × Negative Events interactions continued to predict change in SADS depressive symptoms significantly when diagnostic group (bipolar, unipolar) was included as a covariate in the regression equation.

Table 4
Hierarchical Multiple Regression Analyses to Predict Change in Clinician-Rated (SADS) Mania in Bipolar Participants

Step/predictor	Beta	*pr*	*t*	*df*	Total R^2	R^2 change
1 ASQ-NC	0.23	.03	0.19	36	.00	.00
2 NEGEV	0.27	.24	1.45	35	.06	.06
3 ASQ-NC × NEGEV	0.62	.33	2.01*	34	.16	.10
1 DAS	0.03	.28	1.68†	33	.08	.08
2 NEGEV	0.24	.22	1.28	32	.13	.05
3 DAS × NEGEV	0.01	.43	2.67**	31	.29	.16
1 SRIP-NC	1.27	.22	1.30	34	.05	.05
2 NEGEV	0.21	.20	1.17	33	.09	.04
3 SRIP-NC × NEGEV	0.38	.30	1.78†	32	.17	.08

Note. SADS = Schedule for Affective Disorders and Schizophrenia; ASQ-NC = Time 1 Attributional Style Questionnaire composite for negative events; NEGEV = Time 2 Life Experiences Survey total number of negative events; DAS = Time 1 Dysfunctional Attitudes Scale; SRIP-NC = Time 1 Self-Referent Information Processing Task Battery composite for negative depression-relevant stimuli.
† $p < .10$. * $p < .05$. ** $p < .01$.

over time. As expected, only bipolar and unipolar individuals with negative cognitive styles who reported a high number of negative life events experienced increases in depressive symptoms. Second, within the bipolar group, Time 1 self-report and information-processing measures of cognitive vulnerability also interacted with subsequently occurring negative life events to prospectively predict increases in clinician-rated manic symptoms over time. Again, it was only bipolar participants exhibiting negative cognitive styles and reporting a high number of intervening negative events who experienced increases in manic symptoms. Although the degree of change in depressive and manic symptoms over the 1-month interval may not have been of a clinical magnitude, these findings, nevertheless, support the applicability of the cognitive theories of unipolar depression to the bipolar spectrum and suggest that further research on cognitive vulnerability–stress approaches to bipolar disorders is warranted.

Cognitive Vulnerability–Stress Prediction of Depressive and Manic Symptoms

Our findings are consistent with an extension of both the hopelessness (and reformulated helplessness; Abramson et al., 1978, 1989; Alloy et al., 1988) and Beck's (1967) theories to bipolar spectrum disorders. Moreover, the consistency of the pattern of the cognitive vulnerability–stress interaction across the three measures of cognitive vulnerability (ASQ, DAS, SRIP) is impressive and gives added confidence to the reliability of the findings. These results are also congruent with those of prior studies by Alloy, Reilly-Harrington, et al. (1999), Hammen et al. (1992), and Swendsen et al. (1995). It is interesting that although the present findings were consistent with both hopelessness and Beck's theories, the self-report measure derived from Beck's theory (DAS) in interaction with negative events accounted for more variance in depressive and manic symptom change than did the self-report measure derived from hopelessness theory (ASQ) in combination with negative events. In contrast, Alloy, Reilly-Harrington, et al. (1999) found that whereas attributional style (ASQ) interacted with life events to significantly predict increases in both depressive and manic symptoms (6%–16% of the variance) among individuals with subsyndromal bipolar and unipolar disorders (cyclothymia, dysthymia, hypomania, and normal controls), dysfunctional attitudes (DAS) did not predict changes in the depressive and manic symptoms of these individuals either alone or in interaction with events. Thus, further work is needed to examine which of a number of potential measures of cognitive vulnerability derived from both hopelessness theory and Beck's theory, as well as other cognitive models, may be most predictive of depressive and manic symptom increases in combination with stressful events in bipolar and unipolar samples.

Several aspects of our cognitive vulnerability–stress findings are noteworthy. First, the fact that self-report (ASQ, DAS) and information-processing (SRIP Battery) measures (including RT and recall indices) of cognitive vulnerability both interacted with stressful events to predict depressive and manic symptom increases suggests that both assessment approaches may tap important aspects of cognitive vulnerability and have predictive validity for mood disorders. Indeed, Alloy et al. (1997) found that cognitive risk for depression as assessed by self-report questionnaires (modified ASQ and DAS) and information processing (SRIP Battery) converged with each other, and Alloy, Abramson, et al. (1999) reported that both of these assessment approaches predicted onsets of major and minor depressive episodes prospectively. Thus, the use of a multimethod approach to the measurement of cognitive vulnerability in the present study strengthens the findings from our diathesis–stress analyses.

A second striking feature of our findings is that among the bipolar participants, negative cognitive styles combined with stressful life events to predict increases in manic as well as depressive symptoms. This finding is consistent with the Johnson and Roberts (1995) review, in which negative life events were found to precipitate both mania and depression in bipolar patients, and with the more recent study by Malkoff-Schwartz et al. (1998), in which severe threatening life events were found to be more likely to occur in pre-onset than control periods of both manic and depressive index episodes of bipolar patients. Not only did nega-

tive life events contribute to the prediction of manic symptom increases in the present study, but they did so in combination with cognitive styles traditionally thought and often found to be depressogenic (Alloy, Abramson, et al. 1999), that is, internal, stable, global attributional styles for negative events, dysfunctional attitudes, and negative self-referent processing. These findings suggest that similar psychological processes may increase vulnerability to both depressive and manic symptoms and are compatible with traditional psychodynamic formulations suggesting that bipolar individuals' manic–hypomanic periods may be a defense or counterraction to underlying depressive tendencies (e.g., Freeman, 1971). They also raise the possibility that there may be some fundamental similarities between unipolar and bipolar depression (Depue & Monroe, 1978; Goodwin & Jamison, 1990).

On the other hand, if negative life events interact with cognitive vulnerability to predict both depressive and manic symptom increases, what determines which type of episode a bipolar individual will experience at a particular point in time? Although our data do not address this issue, it is possible that particular types of stressful events are more likely to trigger manic–hypomanic episodes than depressive episodes. In particular, manic–hypomanic episodes may be more likely to follow stressors that disrupt the sleep–wake cycle (i.e., social rhythm disruptors; Malkoff-Schwartz et al., 1998), whereas depressive episodes may be more likely to follow loss events (e.g., Brown & Harris, 1978).

From a methodological perspective, two additional aspects of our study are worth noting. First, the depressive and manic symptom increases we observed as a function of participants' cognitive styles and life events were assessed by clinician-rating based on SADS diagnostic interviews, rather than by participants' self-reports. Thus, symptom scores were filtered through clinicians' more objective judgments and were less likely to be directly biased by participants' cognitive styles, diagnostic history, or current mood. Second, our study involved a largely untreated student sample who, despite meeting RDC for a diagnosis of unipolar or bipolar disorder, were mostly not in episode at Time 1 and were presumably experiencing less current impairment from their mood disorder than would be true of most clinical samples. On the one hand, the fact that our sample was largely untreated and in a remitted state at the start of the study mitigates against the possibility that their cognitive style and life event assessments were contaminated by treatment effects or current mood disturbance. On the other hand, given the relatively high current functioning of our participants, the clinical significance (Jacobson & Truax, 1991) of our observed depressive and manic symptom increases over time is unclear. It should be noted, however, that the ability to predict symptom exacerbations in people with bipolar and unipolar disorders is clinically important in and of itself, because such symptom exacerbations can impact the course of the disorder and worsen prognosis. Further studies are needed to examine whether the present findings generalize to treated samples and predict depressive and manic symptom exacerbations and full-blown episode onsets in unipolar and bipolar patients.

Cognitive Styles of Unipolar and Bipolar Groups

A secondary goal of our study was to explore the cognitive styles of bipolar participants. Three prior studies (Alloy, Reilly-Harrington, et al., 1999; Hollon et al., 1986; Winters & Neale, 1985) found that individuals with bipolar spectrum disorders exhibited cognitive patterns as negative as those of unipolar depressed persons and more negative than those of normal control persons. Consistent with these studies, we also found that bipolar participants' attributional styles, dysfunctional attitudes, and self-referent information processing generally did not differ from those of unipolar participants; however, their cognitive styles also did not differ from those of the normal control participants. There was some evidence that bipolar participants' behavioral predictions and self-descriptiveness judgments (two components of the SRIP Battery) were more negative than those of unipolar and normal participants (see footnote 2); however, our cognitive measures are more striking for the absence than the presence of diagnostic group differences. Given that participants who were in a depressed episode at Time 1 did differ from those who were not depressed on attributional style and self-referent processing, the absence of lifetime diagnostic group differences in cognitive styles in our study is probably due to the fact that most of our participants were not in episode at Time 1. On the other hand, both Alloy, Reilly-Harrington, et al. (1999) and Winters and Neale found that, even when remitted, bipolar individuals' cognitive styles (attributions and dysfunctional attitudes) were more negative than those of normal control participants. Thus, further work comparing the cognitive patterns of bipolar, unipolar, and normal groups is needed before any definitive conclusions about bipolar individuals' cognitive styles can be drawn.

Limitations of the Present Study

Perhaps the main limitation of our study is the use of a self-report checklist of life events, albeit a reliable and validated one, rather than an interview-based assessment. Although recall and reporting biases may affect reporting of events in structured interviews, there is greater risk of an event representing experiences of different objective magnitudes across individuals on self-report measures. We attempted to minimize such biases by using the number of negative events participants reported in our vulnerability–stress analyses and ignoring their subjective impact ratings for the events, and by using a priori ratings of the dependence of events on an individual's behavior. However, we cannot be sure that this approach eliminated event-reporting biases. Indeed, the bipolar group reported significantly more stressful events at Time 2 than did the normal control group. That the bipolar group reported more major as well as minor events than the normal control group and did not differ from the normal group on the rated dependence of their reported events suggests that the bipolar group's event scores were not likely to be due primarily to reporting biases or generation of stressors. Nonetheless, future tests of the applicability of cognitive vulnerability–stress models to bipolar disorders would benefit from the use of interview assessments of life events.

Implications for Cognitive Theory and Treatment

If replicated and extended by additional studies, our findings suggest that cognitive perspectives may contribute to an understanding of the timing, frequency, and severity of depressive and manic–hypomanic symptoms among individuals with bipolar spectrum disorders. In addition, these findings contribute to an

empirical base for suggesting that cognitive–behavioral interventions aimed at modifying maladaptive cognitive styles and decreasing the impact of environmental stressors that have proved effective for unipolar depression (Beck et al., 1979) may also be useful as adjunctive treatments for bipolar disorders (Newman & Beck, 1992). Indeed, a recent controlled trial of cognitive–behavioral group treatment as an adjunct to standard pharmacotherapy for bipolar disorder (Hirshfeld et al., 1998) reported that patients who completed the adjunctive cognitive–behavioral group treatment had longer periods of euthymia and fewer new episodes than control participants treated with standard pharmacotherapy alone. In conclusion, the present findings suggest that the current biological understanding of the course and treatment of bipolar disorders may have much to gain from the incorporation of a cognitive vulnerability–stress perspective within an interdisciplinary approach.

References

Abramson, L. Y., Alloy, L. B., Hogan, M. E., Whitehouse, W. G., Rose, D. T., & Panzarella, C. (1998). *The Temple–Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Axis II personality disorders and dimensions in individuals at high and low cognitive risk for depression.* Manuscript in preparation, University of Wisconsin—Madison.

Abramson, L. Y., Alloy, L. B., & Metalsky, G. I. (1995). Hopelessness depression. In G. Buchanan & M. E. P. Seligman (Eds.), *Explanatory style* (pp. 113–134). Hillsdale, NJ: Erlbaum.

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96,* 358–372.

Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87,* 49–74.

Alba, J. W., & Hasher, L. (1983). Is memory schematic? *Psychological Bulletin, 93,* 207–231.

Alloy, L. B., Abramson, L. Y., Metalsky, G. I., & Hartlage, S. (1988). The hopelessness theory of depression: Attributional aspects. *British Journal of Clinical Psychology, 27,* 5–21.

Alloy, L. B., Abramson, L. Y., Murray, L. A., Whitehouse, W. G., & Hogan, M. E. (1997). Self-referent information processing in individuals at high and low cognitive risk for depression. *Cognition and Emotion, 11,* 539–568.

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Tashman, N. A., Steinberg, D. L., Rose, D. T., & Donovan, P. (1999). Depressogenic cognitive styles: Predictive validity, information processing and personality characteristics, and developmental origins. *Behaviour Research and Therapy, 37,* 503–531.

Alloy, L. B., Reilly-Harrington, N. A., Fresco, D., Whitehouse, W. G., & Zechmeister, J. S. (1999). Cognitive styles and life events in subsyndromal unipolar and bipolar disorders: Stability and prospective prediction of depressive and hypomanic mood swings. *Journal of Cognitive Psychotherapy: An International Quarterly, 13,* 21–40.

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects.* New York: Harper & Row.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford Press.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8,* 77–100.

Brown, G. W., & Harris, T. O. (1978). *Social origins of depression: A study of psychiatric disorder in women.* New York: Free Press.

Bumberry, W., Oliver, J. M., & McClure, J. N. (1978). Validation of the Beck Depression Inventory in a university population using psychiatric estimate as the criterion. *Journal of Consulting and Clinical Psychology, 46,* 150–155.

Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis of the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Depue, R. A., Krauss, S., Spoont, M. R., & Arbisi, P. (1989). General Behavior Inventory identification of unipolar and bipolar affective conditions in a nonclinical university population. *Journal of Abnormal Psychology, 98,* 117–126.

Depue, R. A., & Monroe, S. M. (1978). The unipolar–bipolar distinction in the depressive disorders. *Psychological Bulletin, 85,* 1001–1030.

Depue, R. A., Slater, J. F., Wolfstetter-Kausch, H., Klein, D., Goplerud, E., & Farr, D. (1981). A behavioral paradigm for identifying persons at risk for bipolar depressive disorder: A conceptual framework and five validation studies. *Journal of Abnormal Psychology, 90,* 381–438.

Derry, P. A., & Kuiper, N. A. (1981). Schematic processing and self-reference in clinical depression. *Journal of Abnormal Psychology, 90,* 286–297.

Ehlers, C. L., Frank, E., & Kupfer, D. J. (1988). Social zeitebers and biological rhythms: A unified approach to understanding the etiology of depression. *Archives of General Psychiatry, 45,* 948–952.

Ellicott, A., Hammen, C., Gitlin, M., Brown, G., & Jamison, K. (1990). Life events and the course of bipolar disorder. *American Journal of Psychiatry, 147,* 1194–1198.

Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry, 35,* 837–844.

Freeman, T. (1971). Observations on mania. *International Journal of Psychology, 52,* 479–486.

Goodwin, F. K., & Jamison, K. R. (1990). *Manic–depressive illness.* New York: Oxford University Press.

Gotlib, I. H., & McCann, C. D. (1984). Construct accessibility and depression: An examination of cognitive and affective factors. *Journal of Personality and Social Psychology, 47,* 427–439.

Greenberg, M. S., & Alloy, L. B. (1989). Depression versus anxiety: Processing of self- and other-referent information. *Cognition and Emotion, 3,* 207–223.

Greenberg, M. S., & Beck, A. T. (1989). Depression versus anxiety: A test of the content specificity hypothesis. *Journal of Abnormal Psychology, 98,* 9–13.

Haaga, D. A. F., Dyck, M. J., & Ernst, D. (1991). Empirical status of cognitive theory of depression. *Psychological Bulletin, 110,* 215–236.

Hammen, C. (1980). Depression in college students: Beyond the Beck Depression Inventory. *Journal of Consulting and Clinical Psychology, 48,* 126–128.

Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology, 100,* 555–561.

Hammen, C., Ellicott, A., & Gitlin, M. (1992). Stressors and sociotropy/autonomy: A longitudinal study of their relationship to the course of bipolar disorder. *Cognitive Therapy and Research, 16,* 409–418.

Hammen, C., Ellicott, A., Gitlin, M., & Jamison, K. (1989). Sociotropy/autonomy and vulnerability to specific life events in patients with unipolar depression and bipolar disorders. *Journal of Abnormal Psychology, 98,* 154–160.

Hammen, C., & Krantz, S. E. (1985). Measures of psychological processes in depression. In E. E. Beckham & W. R. Leber (Eds.), *Handbook of depression: Treatment, assessment, and research* (pp. 408–444). Homewood, IL: Dorsey Press.

Healy, D., & Williams, J. M. G. (1988). Dysrhythmia, dysphoria, and depression: The interaction of learned helplessness and circadian dysrhythmia in the pathogenesis of depression. *Psychological Bulletin, 103,* 163–178.

Hirshfeld, D. R., Gould, R. A., Reilly-Harrington, N. A., Morabito, C., Cosgrove, V., Guille, C., Fredman, S., & Sachs, G. S. (1998, November). *Short-term adjunctive cognitive–behavioral group therapy for bi-*

polar disorder: Preliminary results from a controlled trial. Paper presented at the Association for the Advancement of Behavior Therapy Meeting, Washington, DC.

Hollon, S. D., Kendall, P. C., & Lumry, A. (1986). Specificity of depressogenic cognitions in clinical depression. *Journal of Abnormal Psychology, 95,* 52–59.

Hunt, N., Bruce-Jones, W., & Silverstone, T. (1992). Life events and relapse in bipolar affective disorder. *Journal of Affective Disorders, 25,* 13–20.

Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59,* 12–19.

Johnson, S. L., & Roberts, J. E. (1995). Life events and bipolar disorder: Implications from biological theories. *Psychological Bulletin, 117,* 434–449.

Kendall, P. C., Hollon, S. D., Beck, A. T., Hammen, C. L., & Ingram, R. E. (1987). Issues and recommendations regarding use of the Beck Depression Inventory. *Cognitive Therapy and Research, 11,* 289–299.

Kraepelin, E. (1921). Manic–depressive insanity and paranoia (R. M. Barclay, Trans.). In R. M. Barclay (Ed.), *Textbook of psychiatry.* Edinburgh, Scotland: E & S Livingstone.

Lish, J. D., Dime-Meenan, S., Whybrow, P. C., Price, R. A., & Hirschfeld, R. M. A. (1994). The national depressive and manic–depressive association survey of bipolar members. *Journal of Affective Disorders, 31,* 281–294.

Malkoff-Schwartz, S. F., Frank, E., Anderson, B., Sherrill, J. T., Siegel, L., Patterson, D., & Kupfer, D. J. (1998). Stressful life events and social rhythm disruption in the onset of manic and depressive bipolar episodes: A preliminary investigation. *Archives of General Psychiatry, 55,* 702–707.

Miklowitz, D. J., Goldstein, M. J., & Nuechterlein, K. H. (1995). Verbal interactions in the families of schizophrenic and bipolar affective patients. *Journal of Abnormal Psychology, 104,* 268–276.

Miklowitz, D. J., Goldstein, M. J., Nuechterlein, K. H., Snyder, K. S., & Mintz, J. (1988). Family factors and the course of bipolar affective disorder. *Archives of General Psychiatry, 45,* 225–231.

Monroe, S. M., & Simons, A. D. (1991). Diathesis–stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin, 110,* 406–425.

Newman, C., & Beck, A. T. (1992). *Cognitive therapy of rapid cycling bipolar affective disorder: A treatment manual.* Unpublished manuscript, University of Pennsylvania.

O'Connell, R. A. (1986). Psychosocial factors in a model of manic depressive disease. *Integrative Psychiatry, 4,* 150–161.

Peterson, C., Semmel, A., von Baeyer, C., Abramson, L. Y., Metalsky, G. I., & Seligman, M. E. P. (1982). The Attributional Style Questionnaire. *Cognitive Therapy and Research, 6,* 287–299.

Prien, R. F., & Potter, W. Z. (1990). NIMH Workshop Report on Treatment of Bipolar Disorder. *Psychopharmacology Bulletin, 26,* 409–427.

Sarason, I. G., Johnson, J. H., & Siegel, J. M. (1978). Assessing the impact of life changes: Development of the Life Experiences Survey. *Journal of Consulting and Clinical Psychology, 46,* 932–946.

Segal, Z. V. (1988). Appraisal of the self-schema construct in cognitive models of depression. *Psychological Bulletin, 103,* 147–162.

Segal, Z. V., & Vella, D. D. (1990). Self-schema in major depression: Replication and extension of a priming methodology. *Cognitive Therapy and Research, 14,* 161–176.

Seligman, M. E. P., Abramson, L. Y., Semmel, A., & von Baeyer, C. (1979). Depressive attributional style. *Journal of Abnormal Psychology, 88,* 242–247.

Spitzer, R. L., & Endicott, J. (1978). *Schedule for Affective Disorders and Schizophrenia—Change Version.* New York: New York State Psychiatric Institute, Biometrics Research.

Spitzer, R. L., Endicott, J., & Robins, E. (1978). Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry, 35,* 773–782.

Swendsen, J., Hammen, C., Heller, T., & Gitlin, M. (1995). Correlates of stress reactivity in patients with bipolar disorder. *American Journal of Psychiatry, 152,* 795–797.

Taylor, S. E., & Crocker, J. (1981). Schematic bases of social information processing. In E. T. Higgins, P. Herman, & M. P. Zanna (Eds.), *The Ontario Symposium in Personality and Social Psychology* (Vol. 1, pp. 81–134). Hillsdale, NJ: Erlbaum.

Weissman, A., & Beck, A. T. (1978, November). *Development and validation of the Dysfunctional Attitudes Scale.* Paper presented at the meeting of the Association for the Advancement of Behavior Therapy, Chicago, IL.

Winters, K. C., & Neale, J. M. (1985). Mania and low self-esteem. *Journal of Abnormal Psychology, 94,* 282–290.

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