The psychosocial context of bipolar disorder: Environmental, cognitive, and developmental risk factors

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Abstract

In this article, we review empirical research on the role of individuals' current environmental contexts, cognitive styles, and developmental histories as risk factors for the onset, course, and expression of bipolar spectrum disorders. Our review is focused on the following overarching question: Do psychosocial factors truly contribute risk to the onset, course, or expression of bipolar disorders? As a secondary issue, we also address whether the psychosocial risks for bipolar disorders are similar to those for unipolar depression. We begin by discussing the methodological requirements for demonstrating a psychosocial risk factor and the challenges posed by bipolar spectrum disorders for psychosocial risk research. Next, we review the extant studies on the role of recent life events and supportive and nonsupportive social interactions (current environment) in bipolar disorders, as well as psychosocial treatments designed to remediate these current environmental factors. We then review the role of cognitive styles featured as vulnerabilities in theories of unipolar depression as risk factors for bipolar disorder alone and in combination with life events, including studies of cognitive-behavioral therapies for bipolar disorder. Finally, we review studies of parenting and maltreatment histories in bipolar disorders. We conclude with an assessment of the state of the psychosocial risk factors literature in bipolar disorder with regard to our guiding questions.
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"My temperament, moods, and illness clearly, and deeply, affected the relationships I had with others and the fabric of my work. But my moods were themselves powerfully shaped by the same relationships and work. The challenge was in learning to understand the complexity of this mutual beholdenness..." (Jamison, 1995, p. 88)

Like the disorder itself, empirical and theoretical work on bipolar disorder has "seesawed" back and forth between psychological and biological conceptualizations. Despite the early pioneering work of Kraepelin (1921) emphasizing the psychosocial context of the disorder, conceptions of bipolar disorder as a genetically based, biological illness dominated over the past century. Family, twin, and adoption studies suggesting that bipolar disorder has a strong genetic predisposition (Goodwin & Jamison, 1990; Nurnberger & Gershon, 1992) and pharmacotherapy trials indicating the effectiveness of lithium and anticonvulsive drugs in controlling the cycling of bipolar disorder (e.g., Keck & McElroy, 1996) shifted the focus to the disorder's biological underpinnings. However, in the past decade and a half, there has been resurgence of interest in the role of psychosocial processes in the onset, course, expression, and treatment of bipolar spectrum disorders. This swing of the seesaw back again toward the inclusion of psychosocial factors in bipolarity research is largely attributable to researchers' dual recognition that genetic and biological processes are unable to fully account for differences in the expression, timing, and polarity of symptoms (O'Connell, 1986) and that lithium's and other drugs' prophylactic effects are limited. In fact, a 1990 NIMH workshop report (Prien & Potter, 1990) called for further exploration of the impact that psychosocial factors have on the course of bipolar disorder as well as the development of psychosocial treatments as an adjunct to pharmacotherapy. Given that bipolar disorder is often a severe, recurrent, or unremitting illness with significant impairment including alcohol abuse, suicide, divorce, and erratic work history (Goodwin & Jamison, 1990) that affects about 1.5% of the U.S. population (Hyman, 2000) and between 0.5% and 3.5% of the world population (Kleinman et al., 2003), an understanding of the role of psychosocial factors in the onset, course, expression, and treatment of this disorder has great importance.

Consequently, in this article, we review empirical research on individuals' current environmental contexts, cognitive/personality styles, and developmental histories as psychosocial risk factors for the onset, course, and expression of bipolar disorder, as well as psychosocial treatments based on these putative risk factors. The overarching question that provides the foundation for our review is: Do psychosocial factors truly contribute to the onset, course, or expression of bipolar disorders? Do they do so above and beyond the role of genetic predisposition? Given that much of the research on the role of psychosocial factors in the psychopathology and treatment of bipolar disorders has evolved out of work on unipolar depression, we also address a secondary question in our review: Are the psychosocial factors that contribute risk to bipolar disorder similar or dissimilar to those found to be important in unipolar depression? Research has provided support for a continuum or spectrum of severity within the bipolar category ranging from the milder, subsyndromal Cyclothymia, to Bipolar II disorder, to full-blown Bipolar I disorder (Akiskal, Djenderedjian, Rosenthal, & Khani, 1977; Akiskal, Khani, & Scott-Strauss, 1979; Cassano et al., 1999; Depue et al., 1981; Goodwin & Jamison, 1990; Klein, Depue, & Slater, 1985; Waters, 1979). Consequently, our review covers studies on the full range of bipolar spectrum disorders.

We begin our article by discussing the methodological requirements for demonstrating a psychosocial risk factor and the challenges posed by bipolar spectrum disorders for such endeavors. Next, we review the extant studies on the role of recent life events and supportive and non-supportive social interactions (current environment) in bipolar disorders, as well as psychosocial treatments

designed to remediate these current environmental factors. We then review the role of cognitive styles featured as vulnerabilities in theories of unipolar depression as risk factors for bipolar disorder alone and in combination with life events, including studies of cognitive-behavioral therapies for bipolar disorder. Finally, we review studies of parenting and maltreatment histories (early developmental environment) in bipolar disorders. In order to provide an estimate of the magnitude of the effects of psychosocial risk factors on the onset, course, or expression of bipolar disorders, we also present effect sizes (ES) for the findings reviewed, whenever it was possible to do so.[1] We conclude with an assessment of the state of the psychosocial risk factors literature in bipolar disorder with regard to our guiding questions.

1. Methodological requirements of psychosocial risk research and challenges posed by bipolar spectrum disorders

How does one decide whether a particular psychosocial variable is a risk factor for the onset, course, or expression of bipolar disorder? Researchers (Alloy, Abramson, Raniere, & Dyller, 1999; Ingram, Miranda, & Segal, 1998) have suggested that a risk factor for a disorder (e.g., bipolar disorder) must meet two criteria: (1) It must temporally precede mood episodes or symptom exacerbations of bipolar disorder; and (2) it must exhibit some degree of stability independent of bipolar symptomatology. Given these criteria, cross-sectional or retrospective studies that compare bipolar individuals to a normal control group or to a group with another disorder (e.g., unipolar depression) on concurrent or past psychosocial variables can suggest potential risk factors but are inadequate for establishing temporal precedence or stability of the psychosocial variables independent of bipolar symptoms. An improvement over cross-sectional or retrospective studies are studies that compare remitted or euthymic bipolar individuals to normal controls on potential psychosocial risk factors or that longitudinally compare bipolar individuals in depressed, manic/hypomanic, and euthymic states because they can demonstrate independence of the potential risk factor from the symptoms of the disorder. However, such "remitted designs" cannot distinguish between the alternatives that the psychosocial characteristics are risk factors for versus consequences of bipolar disorder (see Just, Abramson, & Alloy, 2001; Lewinsohn, Steinmetz, Larson, & Franklin, 1981). Thus, prospective, longitudinal designs are needed in which the putative psychosocial risk factor is assessed prior to the occurrence of bipolar mood episodes. Such prospective designs can establish both the risk factor's temporal precedence and independence from symptoms (Alloy, Abramson et al., 1999). Consequently, in our review of current environmental factors, cognitive styles, and developmental histories in the course and expression of bipolar disorders, we discuss cross-sectional and retrospective studies more briefly and focus on the methodologically stronger longitudinal and prospective studies. Unfortunately, in some areas of the psychosocial literature (e.g., developmental histories), there are few, if any, longitudinal or prospective studies, and thus, we can only review studies with the methdologically weaker designs.

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[1] We provided effect sizes (ES) either when they were provided in the original articles or when it was possible to calculate them from the information provided in the articles. Following typical conventions, ES based on Cohen's d may be interpreted as follows: $d = .20$ is a small ES, $d = .50$ is a medium ES, and $d = .80$ is a large ES. ES based on Pearson r may be interpreted as follows: $r = .10$ is a small ES, $r = .30$ is a medium ES, and $r = .50$ is a large ES. ES based on ω^2 may be interpreted as follows: $\omega^2 = .01$ is a small ES, $\omega^2 = .06$ is a medium ES, and $\omega^2 = .15$ is a large ES.

However, even prospective longitudinal designs cannot by themselves establish that a psychosocial variable is a causal risk factor for bipolar disorder (Kraemer et al., 1997). To demonstrate a causal risk factor, one must rule out plausible third-variable explanations as well as show that manipulations of the psychosocial factor leads to changes in the expression of the disorder or likelihood of mood episodes. One plausible third variable important to rule out in the case of bipolar disorder is a genetic explanation for findings suggestive of psychosocial risk. Genetic vulnerability as expressed in temperament or other behavioral substrates may be associated with particular cognitive styles or a greater likelihood of exposure to negative, mood-destabilizing environments, as seen in the phenomenon of "genotype–environment correlation" (Plomin & Crabbe, 2000). Almost no studies to date attempt to rule out genetic predisposition. Those few that do (which we note in our review) control for family history of bipolar disorder and thus, control for family environment associated with having a bipolar relative in addition to any genetic predisposition to bipolarity.

Psychosocial treatment studies provide findings relevant to demonstrating that a psychosocial risk factor causally affects the course or expression of bipolar disorder. Given that ethical concerns would prohibit the manipulation of psychosocial variables to induce bipolar symptomatology, treatment studies that manipulate psychosocial variables by attempting to reduce their effects provide the most powerful means of testing the causal significance of psychosocial risk factors. Consequently, we review the findings of three types of psychosocial treatments for bipolar disorder that were developed specifically to remediate the impact of putative environmental and cognitive risk factors. However, the majority of psychosocial intervention studies for bipolar disorder share several common limitations that make it difficult to conclude with any certainty that the manipulation of the psychosocial risk factor is the mechanism underlying any therapeutic change. Typically, these psychosocial interventions differ from the comparison or control therapy not only in their focus on the psychosocial variable of interest, but also on nonspecific therapeutic factors such as therapist allegiance, expectancy of success by both therapist and bipolar patient, and amount of clinical attention received by the patient. Thus, therapeutic improvement may be attributable to these nonspecific factors rather than to amelioration of the psychosocial risk factor of interest. Moreover, to demonstrate the potential causal significance of a psychosocial risk factor, it would also be necessary for treatment studies to demonstrate that this risk factor has actually changed as a result of the intervention. Few of the psychosocial treatment studies on bipolar disorder conducted to date examine the mechanisms of change.

From a methodological perspective, bipolar spectrum disorders present especially challenging problems for demonstrating psychosocial risk. First, these disorders are highly recurrent with significant interepisode symptomatology and functional impairment. Thus, it is difficult to assess environmental or cognitive factors at a time when the individual is asymptomatic in order to establish independence of these potential risk factors from bipolar symptoms. The possibility exists that residual symptoms may bias the assessment of psychosocial variables and there is a need to control for current mood and symptoms in studies of psychosocial risk. Second, many bipolar individuals have chaotic lives as a consequence of many mood swings and interepisodic symptoms. This, in turn, increases the likelihood that they actually contribute to negative features of their environment such as stressful events, poor social support, or negative parenting through poor judgment, poor coping skills, and other symptoms (Alloy, Abramson et al., 1999; Hammen, 1991; Johnson & Roberts, 1995). To deal with this "stress generation" problem, some studies in the life events literature have included only those events that are independent of the participants' behavior, and we note these studies in our review. Given the methodological challenges posed by bipolar disorders, it is not surprising that our conclusions regarding

the role of current environments, cognitive styles, and developmental histories as risk factors for the course and expression of bipolar disorders must remain somewhat tentative.

2. Current environment and bipolar disorder: role of life events and social support

A growing body of evidence suggests that the current environmental context has an important impact on the onset, course, and expression of bipolar spectrum disorders (Alloy, Abramson, Neeren et al., in press; Alloy, Reilly-Harrington, Fresco, & Flannery-Schroeder, 2005; Johnson & Kizer, 2002; Johnson & Roberts, 1995). The role of two kinds of environmental factors has been studied in bipolar disorder: recent life events and social support (including negative support such as expressed emotion). The life events literature has been fairly consistent in suggesting that bipolar individuals experience increased stressful events prior to onset or subsequent episodes of their disorder (Alloy, Abramson, Neeren et al., in press; Alloy, Reilly-Harrington et al., 2005; Johnson & Kizer, 2002; Johnson & Roberts, 1995). In addition, there is reasonable evidence that social support from significant others leads to a more positive course of bipolar disorder, whereas negative support (e.g., high expressed emotion) from family and friends predicts a worse course of bipolar disorder (e.g., Johnson, Meyer, Winett, & Small, 2000; Johnson, Winett, Meyer, Greenhouse, & Miller, 1999; Miklowitz, Goldstein, Nuechterlein, Snyder, & Mintz, 1988; Priebe, Wildgrube, & Muller-Oerlinghausen, 1989; Rosenfarb et al., 2001). Moreover, there is preliminary evidence that two psychosocial interventions developed from the life events and social support/expressed emotion literatures, respectively, may be effective adjunctive treatments for bipolar disorder (e.g., Clarkin et al., 1998; Colom et al., 2003; Frank et al., 1997, 1999; Miklowitz et al., 2000; Miklowitz, George, Richards, Simoneau, & Suddath, 2003; Rea et al., 2003).

However, both of these bodies of literature are characterized by important methodological limitations. First, many studies of life events and social support use retrospective designs, which might lead to "effort after meaning" bias (Brown & Harris, 1978) in recall by the bipolar individuals in providing information on the pre-episode environment. In addition, retrospective designs make it impossible to determine whether the environmental factors are causes or consequences of bipolar symptoms. Related to this, in the life events literature, many studies have failed to differentiate between events that are independent of or dependent on people's behavior, a distinction of considerable importance given the chaotic lifestyles of individuals with bipolar disorders. Second, most studies do not control for any reporting biases associated with bipolar individuals' mood state at the time they are reporting life events or social support. Third, the manner in which environmental risk factors are operationalized and measured is problematic in some studies. For example, some studies rely on self-report measures of life events or social support, which can lead to different subjective interpretations of what experiences count as an instance of a particular life event category or of a non-supportive social interaction. Further, use of self-reports compounds the potential problem of mood-based report biases. Thus, greater weight should be given to studies that employ interviewer assessments of life events or social support. Fourth, some studies do not include an appropriate control group to allow for a determination of whether bipolar individuals' current environment differs from that of normal controls. Fifth, many of the studies do not distinguish between the depressive and manic/hypomanic episodes of bipolar individuals; thus, in these studies, it is unclear whether stressful events or social support affect risk of mania as well as depression. Sixth, the majority of studies use a method of first identifying mood episodes and then examining life events or social support in an interval prior to episode onset, which can lead to type I errors by exclusion

of instances when events or poor support do not lead to onset of episodes. Seventh, some studies use admission to the hospital or the start of a treatment regimen as the time of episode onset, which does not necessarily correspond well with the actual time of episode onset. Finally, many studies use small samples with insufficient power to examine environment–disorder relationships and many rely on severe, patient samples (Bipolar I), which leaves open the question of whether current environmental factors play the same role across the entire spectrum of bipolar disorders (e.g., Bipolar II, Cyclothymia). With these methodological shortcomings in mind, we review what is known about recent life events and social support as risk factors for the onset and course of bipolar disorders.

2.1. Recent life events and bipolar disorder

Overall, studies of life events have found that bipolar individuals experience increased stressful events prior to first onset and recurrences of mood episodes. Moreover, most studies have found that negative life events precede the manic/hypomanic as well as the depressive episodes of bipolar individuals. We briefly review the more methodologically limited retrospective studies first, followed by the stronger prospective studies. We then consider whether specific types of life events or events at earlier points in the bipolar disorder's course are particularly likely to precipitate bipolar mood episodes. In addition, we review studies of a psychosocial treatment designed to reduce the effects of life events that are likely to trigger bipolar mood episodes.

Four studies relied on retrospective review of medical charts to assess life events in patients with bipolar disorder (Ambelas, 1979, 1987; Clancy, Crowe, Winokur, & Morrison, 1973; Leff, Fischer, & Bertelson, 1976) and, thus, did not use optimal measures of life events. These studies found that from 20% to 66% of bipolar patients experienced at least one stressful event rated as independent of their behavior in the 1–3-month period prior to onset of a mood episode and this rate was higher than controls (d's = .62–1.81 in Ambelas, 1979, 1987 and Clancy et al., 1973). Another three retrospective studies (Bidzinska, 1984; Dunner, Patrick, & Fieve, 1979; Kulhara, Basu, Mattoo, Sharan, & Chopra, 1999) administered questionnaires to bipolar individuals regarding their past life events and found that both the first episode (d = .51; Bidzinska, 1984) and episode relapses (d's = .44 and .22 from Bidzinska, 1984 and Kulhara et al., 1999, respectively) were preceded by the occurrence of stressful events. Dunner et al. (1979) reported that an increase in work and interpersonal difficulties was specifically associated with onset of a manic vs. a depressed episode (d = .75).

Two retrospective studies incorporated further methodological improvement by including interview assessments of life events, but they did not assess the independence of the events from bipolar individuals' behavior or include control groups. These studies (Glassner & Haldipur, 1983; Glassner, Haldipur, & Dessauersmith, 1979) found that more first episode (75%) than later episode (56%; d = −1.00) and more late onset (64%) than early onset (23%; d = −1.02) bipolar patients reported a stressful event prior to onset. Another three retrospective studies (Aronson & Shukla, 1987; Davenport & Adland, 1982; Perris, 1984) that examined stressful events independent of individuals' behavior also found that bipolar individuals experienced increased stress prior to episode onsets (d = −.75; Aronson & Shukla, 1987). In the Perris (1984) study, bipolar patients reported nonsignificantly fewer independent events in the year prior to episode onset than neurotic patients (d = −.29), but more than unipolar depressed patients (d = .45). In an experience sampling study (Myin-Germeys et al., 2003) in which participants were signalled at unpredictable times and asked to report on stressors, thoughts, and moods, the bipolar group exhibited significantly more activity-related, but not social, stress than psychotic,

major depressed, and control groups (d's = 1.71, 1.54, and 2.00) and had the largest decrease in positive affect in response to stress.

Five retrospective studies employing life event interviews specifically examined the role of independent stressors in onsets of manic episodes. Four of these studies (Bebbington et al., 1993; Joffe, MacDonald, & Kutcher, 1989; Kennedy, Thompson, Stancer, Roy, & Persad, 1983; Sclare & Creed, 1990) found that manic patients experienced more independent negative events during the period prior to onset than either controls (d's = 1.26, Bebbington et al., 1993; .79, Kennedy et al., 1983) or the period after onset (d's = .70–.75; Joffe et al., 1989; Kennedy et al., 1983; Sclare & Creed, 1990). In contrast, Chung, Langeluddecke, and Tennant (1986) found that manic patients' rate of independent threatening events in the 26 weeks prior to onset did not differ significantly from that of controls (d = .40; although the rate was twice as high in the manic patients).

The methodologically sounder prospective studies provide stronger, albeit not completely consistent, evidence for the role of stressful events as triggers of mood episodes in bipolar individuals. In an early study with questionnaire assessment of life events monthly for 10 months, Hall, Dunner, Zeller, and Fieve (1977) found that although overall numbers of events did not differ significantly for bipolar patients who relapsed vs. those who did not (d = .77), hypomanic relapsers had greater numbers of work-related events than did non-relapsers. In another study using questionnaire assessment of life events every 3 months for up to 3 years, Christensen et al. (2003) found that bipolar women, but not the men, experienced a greater number of events in the 3 months prior to a depressive phase compared to a control period. A major limitation of this study was the failure to track relapses between the 3-month assessments. Finally, in a third study using daily questionnaires for 21 days to track stressors, Lovejoy and Steuerwald (1997) studied 12 cyclothymic, 16 intermittent depressive, and 19 control under-graduates. The cyclothymic group had significantly greater stress than the intermittent depressive group (d = 1.56), which had more stress than the controls.

Two prospective studies examined combined samples of patients and did not examine the bipolar patients separately in their analyses. Marks, Wieck, Checkley, and Kumar (1992) assessed whether life events predicted relapses in 47 pregnant women with a history of bipolar, schizoaffective or major depressive disorder compared with 45 control pregnant women. Women with problematic marital relationships were at higher risk for psychotic relapses (d = .94), whereas women with at least one life event in the 12 months pre-onset were at higher risk for non-psychotic relapses (d = 1.08). Perry, Lavori, Pagano, Hoke, and O'Connell (1992) assessed life events and symptoms with interviews every 3–6 months in a sample of bipolar, schizotypal, and antisocial personality disorder patients. Depressive symptoms were more likely in the 8 weeks following a life event and following events dependent on patients' behavior.

Another six prospective studies used interview assessments of life events in samples of bipolar patients, but most did not include a control group. In a study of 62 bipolar patients followed for 2 years, Hunt, Bruce-Jones, and Silverstone (1992) reported that 19% of 52 relapses were preceded by a severe event in the previous month, compared to a background rate of 5% of patients with a severe event each month at other times (d = .76). Manic and depressive relapses did not differ in the rate of prior events. In contrast, with similar methods, McPherson, Herbison, and Romans (1993) found no significant difference in the number of moderately severe, independent events in the month preceding relapse as compared with control periods (d's = 1.00 and 1.08). However, this study was limited by a high dropout rate and the absence of a required well period prior to study entry. Pardoen et al. (1996) followed 27 recovered bipolar patients, 24 unipolar depressed patients, and 26 normal controls for 1

year with interview assessments of life events and symptoms every 2 months. Bipolar and unipolar patients who relapsed did not report more life events in the 2 months before the relapse compared to those who did not relapse, but among the bipolar patients, those with a manic/hypomanic relapse had more marital stressors prior to the relapse than other bipolar patients. In a study of 61 bipolar outpatients followed over a 2-year period with interviews to assess life events and symptoms, Ellicott, Hammen, Gitlin, Brown, and Jamison (1990) found that bipolar outpatients with high stress showed a 4.5-fold greater relapse rate than those with lower stress and these findings were not accounted for by differences in levels of medication or treatment adherence. Using similar methods in a subsample of 52 bipolar outpatients, Hammen and Gitlin (1997) again found that patients with relapses during the 2-year follow-up period had more severe events and more total stress during the preceding 6 months than those with no episodes (d=.56). Finally, Johnson and Miller (1997) examined negative events via monthly interviews as a predictor of time to recovery from an episode of bipolar disorder. Bipolar inpatients who experienced a severe, independent event during the index episode took three times longer to recover than those who did not experience a severe, independent event ($d=-.92$) and this effect was not mediated by medication compliance.

Are there particular types of life events that are associated with relapses/recurrences of mood episodes among bipolar individuals? Based on consideration of biological mechanisms through which stressful events may influence the onset and course of bipolar disorders, some theorists (e.g., Ehlers, Frank, & Kupfer, 1988; Healy & Williams, 1988) have suggested that life events precipitate mood episodes through their destabilizing effects on circadian rhythms. Specifically, life events that disrupt daily social rhythms (meal times, sleep–wake times, etc.) are hypothesized to trigger mood episodes among bipolar individuals through the effects of the disrupted social rhythms on destabilizing circadian rhythms. Four of five studies conducted to date have provided considerable support for this hypothesis. In two retrospective studies using structured interview assessments and ratings of life events, Malkoff-Schwartz et al. (1998, 2000) reported that manic bipolar patients were significantly more likely to experience pre-onset events characterized by social rhythm disruptions than depressed bipolar (d's=1.36 and 1.19) or unipolar (d=.98) patients. Kadri, Mouchtaq, Hakkou, and Moussaoui (2000) did not actually assess social rhythms, but found that 45% of 20 bipolar patients relapsed during Ramadan (Muslim fasting month with significant changes in social rhythms, i.e., no meals), with 71.4% of these relapses of manic polarity. In a prospective study of 206 bipolar spectrum participants (bipolar II, cyclothymic) and 206 demographically matched normal controls, Shen, Alloy, and Abramson (submitted for publication) found that bipolar individuals had significantly less regular social rhythms than normal controls (d=.20). Moreover, in survival analyses, less regular social rhythms at Time 1 predicted time to onset of major depressive, minor depressive, and hypomanic/manic episodes during an average of 33 months of follow-up. Finally, Ashman et al. (1999) studied social rhythms and mood in nine rapid-cycling bipolar I outpatients and six normal controls and found that the bipolar patients had lower rhythmicity scores than the controls ($d=-1.68$). However, there was no significant relationship between daily social rhythms and mood (d's=.29 and $-.22$), but this was likely due to insufficient statistical power.

Two prospective studies have supported the hypothesis that life events involving goal attainment or goal striving may be especially likely to trigger manic/hypomanic episodes among bipolar individuals. This hypothesis is based on the theory (Depue & Iacono, 1989; Fowles, 1987; Gray, 1991; Johnson, Sandrow et al., 2000; Urosevic et al., 2005) that bipolar individuals are characterized by a hyper-sensitive Behavioral Approach System (BAS) that responds with too extreme positive effect, high

energy, and motivation (i.e., mania/hypomania) to events involving high incentive motivation and goal striving or attainment and with too extreme negative effect, low energy, and anhedonia (i.e., depression) to events involving uncontrollable loss and failure. Consistent with this hypothesis, Johnson, Sandrow et al. (2000) found that goal attainment events, rated based on structured life events interviews, predicted increases in manic symptoms ($d=.36$), but not depressive symptoms ($d=-.02$), among 43 bipolar I patients over the prospective follow-up, whereas general positive events did not predict increases in manic symptoms. Nusslock, Abramson, Harmon-Jones, Hogan, and Alloy (2005) reasoned that among university students, the final exam period should be a pre-goal attainment event involving goal striving given that most students are invested in doing well on exams and, thus, should be particularly likely to elicit hypomania/mania among individuals prone to bipolar disorder. Consistent with prediction and Johnson, Sandrow et al.'s (2000) findings, Nusslock et al. found that individuals in the bipolar spectrum (Bipolar II, Cyclothymia) were especially likely to develop new onsets of hypomanic ($d=1.08$), but not depressive ($d=-.46$), episodes during the final exam period compared to a prior control period.

Are life events more likely to trigger early rather than later mood episodes among bipolar individuals? According to Post's (1992) "kindling" model of the neurobiological changes that may occur with recurrent mood episodes, episodes become increasingly autonomous with each recurrence such that psychosocial stressors are hypothesized to be less likely to precipitate episodes that occur later in the course of disorder than early episodes. Four retrospective and one prospective study have tested the kindling hypothesis in bipolar samples. In a retrospective study of 16 bipolar, 58 unipolar, 81 reactive-neurotic, and 51 unspecified mood disorder patients, Perris (1984) found that patients with recurrent depression had fewer negative events during the 3-month pre-onset period than patients with a first depressive episode ($d=.31$), consistent with the kindling hypothesis. Johnson, Andersson-Lundman, Aberg-Wistedt, and Mathe (2000) conducted a retrospective medical chart review of episode onsets and life events in 190 bipolar and 92 unipolar depressed patients. Also consistent with kindling, the proportion of patients with at least one pre-episode event decreased across episodes and for bipolar patients, 63% experienced an event prior to the first episode vs. only 30% prior to the fifth episode ($d=.85$). Ehnvall and Agren (2002) administered a retrospective interview of life events and episodes to 10 bipolar and 20 unipolar patients. They found a decreased rate of events prior to onsets over the first 9 episodes, with the greatest difference over the first 3 episodes. In contrast, in another retrospective study of 64 bipolar I patients assessed with interviews of life events and episodes, Hlastala et al. (2000) found that number of previous episodes did not predict stress level either in pre-onset or control periods, whereas age did predict stress level in pre-onset, but not control, periods. The probability of experiencing low stress increased as age increased. Hlastala et al. suggested that the aging process rather than illness progression might account for prior studies showing support for the kindling model, given that prior studies ignored the effects of age. Finally, the one prospective study also failed to support kindling. In a sample of 52 bipolar outpatients assessed via interview for independent events every 3 months, Hammen and Gitlin (1997) reported that a significantly greater proportion of patients with many past episodes experienced a severe negative event prior to relapse than of patients with few past episodes ($d=-.96$). Consequently, although there is some evidence from retrospective studies that life events play a smaller role in triggering mood episodes later than earlier in the course of bipolar disorder, the one prospective study conducted to date fails to support the kindling hypothesis. Further prospective studies are needed before any definitive conclusions about the kindling hypothesis may be drawn.

2.2. Psychosocial treatment based on life events: interpersonal and social rhythm therapy

Interpersonal and social rhythm therapy (IPSRT) (Frank, Swartz, & Kupfer, 2000) was designed to be an adjunctive treatment with pharmacotherapy for bipolar disorder and is based on the social rhythms/circadian rhythms theory of bipolar disorder. This intervention is an interpersonally focused individual therapy that incorporates behavioral and environmental interventions to help stabilize irregularities of social rhythms and the sleep–wake cycle that are presumed to be involved in triggering bipolar episodes. IPSRT involves encouraging bipolar patients to focus on the link between life events and mood, the importance of maintaining regular daily rhythms, and the identification and management of interpersonal stressors that may precipitate rhythm dysregulation and, thus, mood episodes.

Four studies have evaluated IPSRT as an adjunctive treatment to pharmacotherapy. In Frank et al. (1997), 38 bipolar I patients were enrolled during an acute mood episode and randomly assigned to either IPSRT or clinical management (CM). Once patients were stabilized, they were randomly reassigned to either IPSRT or CM for a preventive phase, continuing on lithium throughout both acute and preventive phases. Frank et al. (1997) found that IPSRT was more effective than CM in regularizing patients' daily lifestyles, but they never examined the effect of the two treatment conditions on onset of new mood episodes. Using the same design, Frank et al. (1999) found that both IPSRT and CM, in conjunction with medication, were associated with good outcomes. However, bipolar patients whose treatment was switched from the acute to the preventive phase (from IPSRT to CM or from CM to IPSRT) had higher rates of mood episode recurrence than patients whose treatment remained the same across phases ($d = .69$). Frank et al. (1999) suggested that this finding is consistent with the importance of maintaining a stable routine for bipolar individuals. Frank (1999) reported that bipolar patients who completed a full year of preventive treatment with IPSRT were significantly more likely to maintain a euthymic state over the year than those in CM. Finally, Rucci et al. (2002) reported that both IPSRT and CM, in combination with pharmacotherapy, were associated with a reduction in suicide attempts during the preventive phase compared to the acute phase ($d = -.44$ for both IPSRT and CM), but there was not enough statistical power to compare the efficacy of the two treatments in preventing suicide attempts. However, 4 of the 5 suicide attempts observed occurred among the patients in CM rather than IPSRT. Thus, consistent with the possible importance of life events that disrupt social and circadian rhythms as a risk factor for bipolar mood episodes, there is some evidence (Frank et al., 1997) that IPSRT may help bipolar patients to regularize their daily social rhythms and some, but not consistent, evidence (Frank, 1999; Frank et al., 1999; Rucci et al., 2002) that IPSRT may have some promise as an adjunct to pharmacotherapy in improving the course of bipolar disorder.

2.3. Social support and bipolar disorder

Another important aspect of an individual's current environment that affects the course of bipolar disorder is supportive or non-supportive interpersonal relationships. Social support from family and friends can buffer against the deleterious effects of stress or directly enhance functioning among bipolar individuals, whereas high criticism and emotional over-involvement (high "expressed emotion or EE") from family members can provide additional stress and worsen the course of bipolar disorder. We review the social support and EE literatures in bipolar disorder as well as the effectiveness of psychosocial treatment designed to improve family support and communication. We note that although the assessment

of EE across studies has been relatively consistent, the methods used to operationalize social support vary widely across studies.

Four cross-sectional and one retrospective study found that bipolar individuals experience less social support than various control groups and that low social support is associated with mood episode relapses. Using an interview assessment of social support, Romans and McPherson (1992) found that euthymic bipolar I participants reported less social support than community controls (d's $= -.44$ and $-.51$), but they were not significantly different from the controls with past psychopathology. Predominantly manic bipolar participants had less adequate attachments ($d = -.46$) and less available social integration ($d = -.50$) than predominantly depressive bipolar individuals. Although they assessed social adjustment rather than social support per se, Bauwens, Tracy, Pardoen, Vander Elst, and Mendlewicz (1991) reported that both remitted bipolar and unipolar patients scored lower on a measure of social adjustment derived from a semi-structured interview than normal controls ($d = 1.38$ for bipolar patients vs. controls). Within the bipolar group, social maladjustment was related to current symptoms and the number of lifetime mood episodes. Similarly, Kulhara et al. (1999) found that lower social support was associated with a higher frequency of lifetime relapses in bipolar inpatients ($d = .42$). Beyer et al. (2003) used a self-report questionnaire and found that both older and younger bipolar participants perceived lower social support compared to their age-matched peer controls and currently manic bipolar individuals perceived less support than currently depressed or euthymic bipolar participants. In the retrospective study, Stefos, Bauwens, Staner, Pardoen, and Mendlewicz (1996) found that relapses over the past 3 years estimated from medical charts in 21 remitted bipolar patients were significantly associated with low social support ($d = 1.17$) on a self-report questionnaire, maladjustment in social activities on an interview, and poor relationships with extended family.

Four prospective studies also found that poor social support predicts greater relapses and longer time to recovery. In their sample of 27 remitted bipolar, 24 remitted unipolar, and 26 control participants followed for 1 year with self-report measures of social adjustment and self-esteem, Staner et al. (1997) found that among the patients, social maladjustment and low self-esteem predicted relapses. In two prospective studies of bipolar I patients using social support questionnaires, Johnson et al. (Johnson, Meyer et al., 2000; Johnson et al., 1999) found that poorer social support predicted longer time to recovery and prospective depressive (d's $= -.26$ and $-.45$), but not manic ($d = 0$), symptoms. The association of low social support with depressive symptoms was mediated by low self-esteem. Finally, in a sample of 94 bipolar I and II patients in full or partial remission followed for 1 year, Johnson, Lundstroem, Aberg-Wistedt, and Mathe (2003) reported that those who relapsed had lower social support than those who did not relapse on one of two questionnaires ($d = -4.30$) and that levels of social support were equivalent for manic vs. depressive relapses.

Five cross-sectional studies of "expressed emotion" (EE) and bipolar disorder have focused on the characteristics of bipolar individuals' family interactions. Bromet, Ed, and May (1984) found that in a combined sample of bipolar and unipolar patients, those with more symptoms perceived their families more negatively on cohesion, expressiveness, conflict, moral–religious emphasis, and organization subscales (d's $= -.31$ to $-.52$) of a family environment scale. Miklowitz, Goldstein, and Nuechterlein (1995) found that based on observers' ratings of family interactions, schizophrenic patients' relatives made more intrusive statements than bipolar patients' relatives ($d = -.85$), bipolar patients made more supportive statements about their relatives than did schizophrenic patients ($d = .44$), and among the bipolar patients, those with higher hostility/suspicion had relatives who made more intrusive statements.

Koenig, Sachs-Ericsson, and Miklowitz (1997) examined bipolar I patients' ratings of a family interaction with their relatives. Bipolar patients with greater critical or intrusive ratings of their relatives' statements reported more distress (d's = .50–.74), but ratings of relatives' statements were not associated with patients' symptom levels. Simoneau, Miklowitz, and Saleem (1998) rated the EE levels of the relatives of bipolar I patients during a family interaction with the Camberwell Family Interview (CFI). Bipolar patients from high EE families had more manic symptoms (d = .64) and a trend toward more depressive symptoms (d = .57) than those from low EE families. In addition, high EE families were more likely than low EE families to show complex negative interaction sequences. Finally, Wendel, Miklowitz, Richards, and George (2000) assessed both EE levels and attributions of relatives during family interactions with the CFI in bipolar I patients. High EE relatives' causal attributions for the patient's role in negative events were more personal (d = .74) and controllable (d = 1.35) than those of low EE relatives.

Three prospective studies found that high EE among relatives is predictive of a worse course of bipolar disorder. Miklowitz et al. (1988) assessed EE with the CFI and affective style (guilt-inducing, critical, and intrusive statements from relatives) with a family interaction involving the patient in the relatives of 23 bipolar and schizoaffective manic inpatients. High EE approached significance in predicting patients' relapse over 9 months, controlling for affective style, and affective style significantly predicted relapse, controlling for EE. Neither EE nor affective style predicted depressive vs. manic relapses. Priebe et al. (1989) examined relatives' EE with the CFI and followed 21 mostly bipolar patients for 9 months. Patients with high EE relatives had 8 times the prospective morbidity rate (hospital admissions, symptoms, additional medications) as patients with low EE relatives (d = 2.31). Rosenfarb et al. (2001) examined relatives' affective style during a family interaction in 27 bipolar I patients followed for 9 months. Relatives of patients who relapsed had more critical (d = .96) and supportive (d = 1.39) statements during the interactions than did relatives of patients who did not relapse, and among the relapsing group, relatives' criticism was positively related to patients' unusual thoughts during the interaction (d = .53).

2.4. Psychosocial treatment based on social support/EE: family-focused psychoeducation

The objective of family-focused psychoeducation (FFT) is to educate patients and their families/spouses about bipolar disorder, enhance communication skills and family social support, and teach patients problem-solving skills in order to reduce the chance of relapse. Typically, these studies involved a 21-session program administered over 9 months, with patients beginning treatment in a euthymic state. Most studies used a randomized controlled trial in which patients were assigned to either FFT or a Crisis Management (CM) intervention. All patients received concomitant pharmacotherapy. Two-year follow-up assessments were typically conducted.

To date, five studies (Clarkin et al., 1998; Colom et al., 2003; Miklowitz, George et al., 2003; Miklowitz et al., 2000; Rea et al., 2003) have directly compared the efficacy of FFT to CM as a prophylactic treatment for bipolar disorder. All of these studies found FFT to be superior to CM, or a comparable comparison group, either in reducing relapse rates (d's = − .49, − .84, and .64), increasing the time to relapse (d = .59), decreasing hospitalization rates (d's = − 1.07 and − 1.29), or reducing inter-morbid symptoms (d's = − .32 to − .37). FFT was also found to be effective in increasing medication compliance (Clarkin et al., 1998; d = .99). However, these studies indicated that FFT is more effective in managing depression than mania. In Colom et al. (2003), at the end of follow-up, FFT patients had a

lower number of all types of recurrences than CM patients ($d=-.97$), except for mania. Three studies (Fristad, Arnett, & Gavazzi, 1998; Fristad, Gavazzi, & Soldano, 1998; Fristad, Goldberg-Arnold, & Gavazzi, 2003) are also suggestive that FFT is efficacious for managing mood disorders in children and adolescents as well. However, these studies did not conduct separate analyses for children with unipolar depression and bipolar disorder, and bipolar children only comprised about 25–30% of the participants in these studies. Moreover, only one of these studies (Fristad et al., 2003) of FFT in children was a controlled trial. Fristad et al. (2003) found that compared to the control condition, FFT was associated with greater social support among the children ($d=1.10$) and a decrease in EE among the parents of the children ($d=-.75$). Interestingly, Miklowitz, Richards et al. (2003) integrated FFT with IPSRT and found that patients given this combined therapy along with medication showed longer time to relapses than those given CM ($d=.48$). Consistent with other FFT studies, the combined treatment had a greater impact on depressive symptoms than manic symptoms.

Two studies have examined possible mechanisms by which FFT works. Simoneau, Miklowitz, Richards, Saleem, and George (1999) examined the effect of FFT on verbal and non-verbal interaction patterns of bipolar patients and their relatives, as assessed during a problem-solving task conducted before and after FFT intervention. Following treatment, patients and relatives who received FFT showed a greater amount of positive interactional behavior as compared to those in the CM condition ($d=.72$). Additionally, Honig, Hofman, Rozendaal, and Dingemans (1997) found that FFT lowered EE ($d=-.87$) and that patients with key relatives who had low EE had a better course (lower hospital admissions) than patients with high EE key relatives. These findings suggest that changes in social support/EE constructs central to FFT may be mediating the positive effects of FFT, although further work is needed on the mechanisms of FFT.

2.5. Summary of current environment findings

In summary, the evidence relating current environmental factors (stressful life events, social support, EE) to the course of bipolar disorders has been fairly consistent. Although relatively few in number, the methodologically sound prospective studies suggest that similar to the case for unipolar depression, the occurrence of stressful events may contribute proximal risk to onsets and recurrences of mood episodes in individuals with bipolar disorders. Given the extensive literature on the role of stress as a precipitant of episodes of unipolar depression, it is not surprising that negative events may trigger bipolar depressive episodes. However, our review, as well as other reviews (Alloy, Abramson, Neeren et al., in press; Alloy, Reilly-Harrington et al., 2005; Johnson & Roberts, 1995), indicate that negative events may also contribute risk for manic/hypomanic episodes. Further research is needed to determine whether it is negative events that specifically disrupt social and circadian rhythms that are most likely to precipitate bipolar individuals' mood episodes. Preliminary evidence suggests that IPSRT, designed to decrease interpersonal stressors that may trigger social and circadian rhythm dysregulation, could have some effectiveness in lessening the likelihood of mood episodes in bipolar individuals. However, studies that examine the mediating mechanisms by which IPSRT exerts its effects are needed to more clearly support the role of social rhythm disrupting events as a risk factor for bipolar disorder. Given that almost no studies have investigated positive events, future research should examine whether positive events also play a role in the course of bipolar spectrum disorders. Such positive events as achievements and upcoming goals could activate bipolar individuals' BAS and engagement in goal striving, which, in turn, might lead to hypomanic/manic symptoms such as high

activity and energy levels, racing thoughts, increased self-confidence, and risky behaviors (Johnson, Sandrow et al., 2000; Urosevic et al., 2005).

The social support/EE literature is also reasonably consistent in indicating that positive vs. negative interactions with family and friends have an important impact on the course of bipolar disorder, as they do for unipolar depression. Although few in number, the methodologically stronger prospective studies indicate that bipolar individuals with poor social support or relatives with high EE or negative affective style have a longer time to recovery, greater likelihood of relapse, and more impairment than those with high social support or relatives with low EE/positive affective style. Moreover, there is consistent evidence that FFT, designed to reduce relatives' EE and enhance family communication skills and support, decreases risk for mood episode relapse, and beginning evidence that FFT may indeed work via its intended mechanisms. However, it is important to remember that much of the literature on the role of the current environment in bipolar disorder is characterized by important methodological limitations including failure to control for current mood state and family history, small samples, absence of control groups, varying operationalizations of life events and social support, and failure to attend to predictors of episode polarity. Thus, a more definitive understanding of the current environment's impact on the course of bipolar disorder awaits methodological refinements in future studies.

3. Cognitive styles and bipolar disorders

Over the past two decades, there has been growing interest in the role of cognition in bipolar disorders. This line of research has generally addressed two issues: whether bipolar individuals exhibit dysfunctional cognitive styles similar to those observed among unipolar depressed individuals; and whether these cognitive patterns, alone or in combination with life events, serve as risk factors that predict the expression or course of bipolar disorder (Alloy, Abramson, Neeren et al., in press; Alloy, Abramson, Walshaw, & Neeren, in press; Alloy, Reilly-Harrington et al., 2005). Studies of cognitive styles and processes in bipolar disorder have been guided by two main theoretical perspectives: logical extensions of the cognitive models of unipolar depression (Abramson, Metalsky, & Alloy, 1989; Beck, 1967, 1987) and the "manic defense" hypothesis, a psychodynamic model (Abraham, 1911/1927; Dooley, 1921; Freeman, 1971; Klein, 1994; Rado, 1928) updated in cognitive-behavioral terms by Neale (1988).

Given the success of cognitive models in contributing to the understanding of the etiology, course, and treatment of unipolar depression, the logic of these theories has been extended to bipolar disorder (Alloy, Abramson, Neeren et al., in press; Alloy, Abramson, Walshaw et al., in press; Alloy, Reilly-Harrington et al., 2005; Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999; Hammen, Ellicott, & Gitlin, 1992; Newman, Leahy, Beck, Reilly-Harrington, & Gyulai, 2002; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999). Cognitive models of unipolar depression hypothesize that maladaptive cognitive patterns [negative styles for inferring causes, consequences and self-worth implications in Hopelessness theory (Abramson et al., 1989) and negative self-schemata, dysfunctional attitudes, and sociotropic and autonomous personality modes in Beck's (1967, 1987) theory] act as vulnerabilities for depression when individuals experience stressful life events. Such maladaptive cognitive styles increase the likelihood of negative appraisals of negative life events that are encountered, thereby leading to hopelessness and negative views of one's self and personal world, and ultimately, depressive symptoms. The same cognitive processes that contribute vulnerability to

unipolar depressive episodes may also confer risk to the depressive episodes experienced by bipolar individuals following negative events. With respect to risk for manic/hypomanic episodes, two types of predictions may follow from an extension of cognitive theories of unipolar depression (Alloy, Abramson, Walshaw et al., in press). On the one hand, bipolar individuals may also possess positive cognitive styles and self-schemata that increase risk for mania/hypomania when they are activated by the occurrence of positive life events. Alternatively, given that negative events have been found to trigger manic as well as depressive episodes among bipolar individuals (see Life Events section above), bipolar individuals' cognitive styles for appraising negative events, rather than their styles for construing positive events, may be more important in affecting their vulnerability to manic/hypomanic episodes.

Consistent with the potential relevance of negative, depressive cognitive styles to mania/hypomania, psychodynamic formulations suggest that the grandiosity of manic states is a "defense" or counter-reaction to underlying depressive tendencies (e.g., Abraham, 1911/1927; Dooley, 1921; Freeman, 1971; Klein, 1994; Rado, 1928). In a cognitive reconceptualization of this "manic defense" hypothesis, Neale (1988) suggested that life events perceived as a threat to underlying fragile self-esteem lead to grandiose thoughts which function to prevent the underlying depressive cognitions from entering conscious awareness. Thus, mania is seen not as the polar opposite of depression, but rather akin to it cognitively. Inasmuch as depression and mania involve similar negative cognitive styles from this perspective, Neale (1988) postulates that the determining factors of which type of mood episode occurs are relevant life events and one's response to feelings of helplessness and threatened self-esteem. When the individual cannot handle the threat to self-esteem and ensuing helplessness with a cognitive defense mechanism, depression results. On the other hand, mania results from a reactance to threatened self-esteem and helplessness with a last extreme effort to regain control and mastery. Tests of the manic defense hypothesis depend on a comparison of both explicit (i.e., direct) and implicit (i.e., indirect) assessments of cognitive styles or self-esteem. If this hypothesis is correct, bipolar individuals, particularly when in a manic state, should exhibit positive cognitions on explicit measures, but negative, depressive cognitions on implicit measures.

In this section, we review the extant literature on the cognitive styles associated with and predictive of the course of bipolar disorders, as well as studies on the effectiveness of cognitive behavioral therapy (CBT) for bipolar disorders. Most studies in this area use cross-sectional designs and are only relevant to examining the cognitive styles characteristic of bipolar individuals and their similarity to those of unipolar depressed persons. Only a small number of longitudinal studies have tested the cognitive vulnerability and vulnerability–stress hypotheses for the course of bipolar disorder. A central methodological issue in this literature is the need to establish the nature of cognitive styles in bipolar individuals independent of mood states and symptoms of the disorder (Alloy, Abramson, Neeren et al., in press; Alloy, Abramson, Walshaw et al., in press; Alloy, Reilly-Harrington et al., 2005). Studies of cognition and bipolar disorder have addressed this issue in several different ways: by controlling statistically for concurrent mood and symptoms; by examining cognitions among remitted or euthymic bipolar individuals; by comparing bipolar individuals in depressive vs. manic episodes; and by conducting within-subject longitudinal studies of the same bipolar individuals in different mood states. However, many studies in this area suffer from one or more limitations that need to be addressed systematically in future studies, including small sample sizes, undiagnosed samples, failure to take medication status into account, absence of control groups, and unvalidated cognitive measures. Overall, the evidence to date suggests that the observed cognitive patterns of bipolar individuals depends to some degree on their mood state and on whether the cognitive style assessment is based on explicit or implicit

measures (Alloy, Abramson, Neeren et al., in press; Alloy, Abramson, Walshaw et al., in press; Alloy, Reilly-Harrington et al., 2005). Most studies indicate that bipolar individuals exhibit cognitive patterns as negative as those of unipolar depressed individuals (but with certain unique features), but sometimes present themselves in a positive fashion on more explicit cognitive style measures. Moreover, there is some evidence that cognitive styles do predict prospectively the expression and course of bipolar disorder, particularly in combination with relevant life events, and that CBT is an effective adjunctive treatment for bipolar disorder.

3.1. Cross-sectional studies: cognitive styles in a depressed state

Five studies have compared diagnosed bipolar depressed and unipolar depressed groups on cognitive styles and all but one found no differences between the two groups' cognitive styles. For example, both depressed bipolar and unipolar participants showed equally negative dysfunctional attitudes, automatic thoughts, and attributional styles on self-report questionnaires, as well as self-referent information processing characteristic of depression and more negative than normal comparison groups (Hill, Oei, & Hill, 1989; Hollon, Kendall, & Lumry, 1986; Reilly-Harrington et al., 1999; ω^2's = .25–.53 from Hollon et al., 1986). Rosenfarb, Becker, Khan, and Mintz (1998) observed that, on the Depressive Experiences Questionnaire (DEQ; Blatt, D'Afflitti, & Quinlan, 1976), both depressed unipolar and bipolar women were more self-critical than non-psychiatric controls (ω^2 = .57), but only the unipolar depressed women were more dependent than controls (ω^2 = .16). The one exception is an older study by Donnelly and Murphy (1973), with no normal control group that found that the bipolar depressed group had higher ego strength and lower social introversion than the unipolar depressed group. Thus, the cognitive patterns of bipolar individuals in a current depressive episode generally have been found to be as negative as those of unipolar individuals.

3.2. Cross-sectional studies: cognitive styles in a manic/hypomanic state

Three studies, all involving samples of undiagnosed undergraduates who scored high on the Hypomanic Personality Scale (Eckblad & Chapman, 1986), tested the manic defense hypothesis. Failing to support the hypothesis, on an explicit measure of attributional style (Attributional Style Questionnaire [ASQ]; Seligman, Abramson, Semmel, & von Baeyer, 1979), Thompson and Bentall (1990) found that hypomania was associated with global attributions for both positive (r = .24) and negative (r = .19) events. However, consistent with the manic defense hypothesis, using an implicit emotional Stroop test in which participants named the ink colors of depression-related and euphoria-related words, Bentall and Thompson (1990) found that controlling for depressive symptoms, high hypomanic students took longer than low hypomanic students to name the color of depression, but not euphoria, words (ω^2 = .72). These findings were similar to prior findings for unipolar depressed individuals (Ingram et al., 1998). Moreover, they were replicated by French, Richards, and Scholfield (1996) controlling for the effects of anxiety on emotional Stroop performance (ω^2's = .45 and .50).

3.3. Cross-sectional studies: cognitive styles in a remitted/euthymic state

Eleven studies have assessed the cognitive styles of remitted or euthymic bipolar individuals. Across these studies, observations of negative cognitive styles among remitted bipolar individuals did

not appear to depend on whether the cognitive measures are explicit responses on self-report questionnaires or implicit assessments on information processing tasks. Five studies using primarily explicit measures obtained little evidence of negative cognitions in the remitted state. In two studies of the same sample (Pardoen, Bauwens, Tracy, Martin, & Mendlewicz, 1993; Tracy, Bauwens, Martin, Pardoen, & Mendlewicz, 1992), remitted bipolar patients' self-esteem and attributional styles did not differ from normal controls' and were less negative than those of remitted unipolar depressed individuals (ω^2's = .08 and .26). MacVane, Lange, Brown, and Zayat (1978) obtained no differences between euthymic bipolar and normal control participants on locus of control orientation. Hollon et al. (1986) observed that the dysfunctional attitudes and automatic thoughts of remitted bipolar patients did not differ from those of normal controls and remitted unipolar patients and were less negative than those of currently depressed bipolar or unipolar patients. Similarly, Reilly-Harrington et al. (1999) did not obtain differences on attributional style, dysfunctional attitudes, or most measures of self-referent information processing among remitted bipolar, remitted unipolar, and normal control participants.

In contrast, six other studies, also employing mostly explicit, questionnaire-based measures of cognition, obtained more support for negative cognitive styles among remitted bipolar individuals. In the only remitted study directly supportive of the manic defense hypothesis, Winters and Neale (1985) found that remitted bipolar patients exhibited higher self-esteem than remitted unipolar patients and normal controls on explicit questionnaire measures (d's = 3.03 and 1.35) but generated attributions as negative as the remitted unipolar patients on an implicit pragmatic inference task. Alloy, Reilly-Harrington et al. (1999) reported that cyclothymic and dysthymic participants in a euthymic state did not differ from each other and exhibited more negative attributional styles and dysfunctional attitudes on self-report questionnaires than hypomanic and normal participants (ω^2's = .23 and .42). Scott, Stanton, Garland, and Ferrier (2000) found that remitted bipolar patients and normal controls did not differ on explicit self-esteem, but the remitted bipolar patients exhibited more explicit dysfunctional attitudes (ω^2 = .49), greater sociotropy (ω^2 = .07) and perfectionism (ω^2 = .31), fewer solutions on a social problem-solving task (ω^2 = .07), and greater implicit over-general recall on an autobiographical memory task (ω^2 = .06). The perfectionism subscale of dysfunctional attitudes and over-general autobiographical memory best distinguished remitted bipolar patients from the normal controls. Rosenfarb et al. (1998) observed that both remitted bipolar and unipolar women were more self-critical than controls (w^2 = .57), but only the remitted unipolar women were also more dependent than the controls (w^2 = .16) on the DEQ. Lam, Wright, and Smith (2004) found that euthymic bipolar patients only scored higher than euthymic unipolar patients on goal attainment dysfunctional attitudes (ω^2 = .12), but not achievement- or dependency-related dysfunctional attitudes. Finally, controlling for concurrent depressive and hypomanic symptoms, Abramson, Alloy, Walshaw, Whitehouse, and Hogan (2005) found that on explicit self-report questionnaires, euthymic bipolar spectrum participants exhibited more negative inferential styles (d = .35), dysfunctional attitudes (only the perfectionism subscale; d = .39), autonomy (d's = .32–.44), self-criticism (d = .74), private self-consciousness (d = .61), and rumination (d = .80) than did normal controls, but the two groups did not differ on sociotropy, dependency or approval by others. These last four studies converge on the idea that euthymic bipolar individuals exhibit a unique profile of negative cognitive styles consistent with the high drive/incentive motivation associated with high BAS (Depue & Iacono, 1989; Fowles, 1987; Gray, 1991) sensitivity, but not by dependency and attachment attitudes typically observed among unipolar depressed individuals.

3.4. Cross-sectional studies: comparisons of cognitive styles across mood states

Five studies compared depressed bipolar to manic/hypomanic bipolar (and sometimes euthymic bipolar) participants and thereby examined the invariance of cognitive patterns across mood states. Three of these studies utilized only explicit questionnaire cognitive measures. Ashworth, Blackburn, and McPherson (1982) and Hayward, Wong, Bright, and Lam (2002) both found that bipolar manic individuals had higher explicit self-esteem than bipolar depressed individuals ($d = .36$ from Hayward et al., 2002), although in Ashworth et al. (1982), neither group's self-esteem differed significantly from non-psychiatric controls. Scott and Pope (2003) reported that hypomanic bipolar participants had higher levels of both negative and positive self-esteem ($w^2 = .05$) than depressed or remitted bipolar participants, whereas their dysfunctional attitudes were lower than depressed bipolar participants, but higher than remitted bipolar individuals ($w^2 = .05$). Lyon, Startup, and Bentall (1999) employed both explicit and implicit measures of cognitive style. On the explicit questionnaire measures, manic bipolar patients exhibited a positive attributional bias and endorsed more positive than negative words as self-descriptive, similar to normal controls and unlike the depressed bipolar patients (who showed the opposite effects; $\omega^2 = .61$). However, consistent with the manic defense hypothesis, on the implicit tests, manic bipolar patients, like depressed bipolar patients, attributed negative events internally on the Winters and Neale (1985) pragmatic inference task ($\omega^2 = .54$), showed slower color-naming for depression-related rather than euphoria-related words on the emotional Stroop task ($\omega^2 = .20$), and recalled more negative than positive words on the self-referent incidental recall task ($\omega^2 = .59$). Finally, Murphy et al. (1999) administered an emotional attentional shifting task and found that bipolar manic patients exhibited positive mood congruent attentional bias ($\omega^2 = .06$), whereas bipolar depressed patients showed negative mood congruent attentional bias ($\omega^2 = .04$).

3.5. Longitudinal studies: stability of cognitive styles within individuals across moods

Only three studies to date used a longitudinal design to investigate the actual stability of cognitive patterns across different mood states of the same bipolar individuals. Ashworth, Blackburn, and McPherson (1985) retested their manic and depressed groups after they remitted on a measure of explicit self-esteem and observed that previously depressed patients showed an increase in self-esteem to normal levels and previously manic patients showed a decrease in self-esteem to normal levels after they recovered. Eich, Macaulay, and Lam (1997) studied autobiographical event generation and recall in a small sample of rapid-cycling bipolar patients. Although their design was longitudinal, their analyses were between rather than within subjects. They found that mood-dependent recall occurred in both depressed and manic states, but patients generated more positive than negative autobiographical events when manic, but more negative than positive events when depressed ($d = .72$). Alloy, Reilly-Harrington et al. (1999) assessed attributional styles, dysfunctional attitudes, and more state-like self-perceptions with explicit questionnaires in diagnosed cyclothymic, dysthymic, hypomanic, and normal control participants on three separate occasions when the different mood states characteristic of their disorder naturally occurred (euthymic, depressed, and hypomanic moods). They observed that both attributional styles and dysfunctional attitudes were stable across participants' mood swings, with dysthymic and cyclothymic individuals exhibiting more negative styles than hypomanic and normal individuals across all mood states (ω^2's = .23 and .42). In contrast, self-perceptions varied across moods and were more positive in hypomanic than depressed moods ($\omega^2 = .28$).

3.6. Longitudinal studies: cognitive styles as predictors of bipolar course

Do cognitive styles alone or in combination with life events act as vulnerabilities that predict the course of bipolar disorder? Three longitudinal studies have examined various self-report questionnaire measures of cognitive styles as predictors of bipolar course without considering the role of life events. In a small sample of bipolar I patients, Johnson, Meyer et al. (2000) reported that low self-esteem (assessed 6 months into the follow-up period) predicted average depression ($r = -.68$), but not average mania, symptom severity, over an 8-month follow-up. Similarly, with a larger bipolar I sample, Johnson and Fingerhut (2004) found that more negative and fewer positive automatic thoughts predicted increases in depressive ($r = .46$), but not manic, symptoms over a 2-year follow-up, controlling for baseline symptoms. Dysfunctional attitudes did not predict either depression or mania symptom change. In contrast to Johnson, Meyer et al. (2000), Scott and Pope (2003) found that negative self-esteem was the most robust predictor of relapse at 12-month follow-up among a small sample of hypomanic bipolar patients.

3.7. Longitudinal studies: cognitive vulnerability × stress predictors of bipolar course

Given that the cognitive theories of unipolar depression are vulnerability-stress models, extensions of these theories to bipolar disorder have led to several efforts to examine the interaction between cognitive styles and life events as predictors of the course of bipolar disorder. To date, six studies have examined the cognitive vulnerability–stress hypothesis for bipolar disorder. All of these studies used explicit self-report questionnaires to assess cognitive styles and one (Reilly-Harrington et al., 1999) also used an implicit measure of self-referent information processing as a measure of cognitive vulnerability as well.

Four of the cognitive vulnerability–stress studies tested Beck's (1987) event congruence, vulnerability–stress hypothesis for sociotropic and autonomous cognitive styles in which the experience of stressful events congruent with one's style (interpersonal events for sociotropic individuals and achievement events for autonomous individuals) should lead to an onset or exacerbation of symptoms. In a small sample of unipolar and bipolar patients followed for 6 months, Hammen, Ellicott, Gitlin, and Jamison (1989) obtained support for the event congruence hypothesis only in the unipolar patients; although there were trends consistent with the hypothesis for the bipolar patients as well. Indeed, in a later study of a larger sample of remitted bipolar patients followed for 18 months, Hammen et al. (1992) found that subsequent symptom severity, but not symptom onset, was predicted by the interaction of sociotropy and negative interpersonal events ($w^2 = .06$). The autonomy × negative achievement events interaction did not predict symptom onset or severity. Based on 4 months of follow-up, Francis-Raniere, Alloy, and Abramson (submitted for publication) found that among bipolar spectrum individuals, controlling for initial depressive symptoms and the total negative events experienced, the self-criticism/performance evaluation × self-criticism-relevant negative events interaction predicted increases in depressive symptoms over the 4 months ($\omega^2 = .05$). Similarly, after controlling for initial hypomanic symptoms and the total positive events experienced, the self-criticism/performance evaluation × congruent positive events interaction predicted increases in hypomanic symptoms over the follow-up ($\omega^2 = .08$). In contrast, sociotropy/dependency buffered against depressive symptoms following both congruent and non-congruent negative events. In a sample of remitted bipolar patients followed for 1 year, Swendsen, Hammen, Heller, and Gitlin (1995) found that those who relapsed were distinguished from those who did not by interactions of stressful events with both obsessionality and extraversion.

Two studies tested the cognitive vulnerability–stress hypotheses of hopelessness (Abramson et al., 1989) as well as Beck's (1967) theories for attributional style and dysfunctional attitudes. In a sample of individuals with both unipolar and bipolar conditions, consistent with hopelessness theory, Alloy, Reilly-Harrington et al. (1999) reported that a negative attributional style (internal, stable, global) for negative events at Time 1 (euthymic state) interacted with subsequent negative events to predict increases in depressive symptoms at Times 2 and 3 (r's = .24). In addition, a positive attributional style (internal, stable, global) for positive events combined with subsequent positive events to predict increases in hypomanic symptoms at Time 2 (r = .40). Dysfunctional attitudes combined with life events were not predictive of subsequent depressive or hypomanic symptoms. Consistent with both hopelessness and Beck's theories, in a large sample of unipolar and bipolar individuals, Reilly-Harrington et al. (1999) found that controlling for initial symptom levels, Time 1 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 (r's = .20–.35) and, within the bipolar group, manic symptoms (r's = .28–.40).

3.8. Psychosocial treatment based on cognitions: cognitive behavioral therapy

As an adjunctive treatment for bipolar disorder, CBT involves teaching bipolar individuals to recognize prodromes to manic and depressive episodes and to modify cognitions and behavior to prevent prodromes from developing into full-blown episodes (Lam et al., 2003; Newman et al., 2002). For example, once bipolar clients have learned to recognize manic prodromes, they are taught strategies to subvert the onset of the episode such as regulating schedules and daily rhythms, reality testing excessive positive beliefs and feelings, using daily thought records, and delaying risky behavior for 48 h in order to recognize the potential negative implications of the behavior. Overall, there is reasonably consistent evidence from the small number of studies conducted to date that CBT has a positive prophylactic effect on bipolar disorder and preliminary evidence from one study that it is also effective in managing acute episodes of bipolar depression. However, several of these studies were not controlled trials and had small sample sizes. In addition, there is considerable variation in how the studies operationalized CBT and several studies incorporated therapeutic techniques that are more consistent with FFT and IPSRT; thus, whether the mechanisms contributing to CBT's success as an adjunctive treatment for bipolar disorder are cognitive, psychoeducational, or based on stabilizing social rhythms is still unclear.

The majority of CBT studies had bipolar individuals on maintenance mood-stabilizing medication begin treatment while in a euthymic state and then examined whether CBT was effective in preventing relapse, managing inter-morbid symptoms, and reducing hospitalization. Two uncontrolled studies (Fava, Bartolucci, Rafanelli, & Mangelli, 2001; Patelis-Siotis et al., 2001) found that CBT reduced depressive and manic episode relapse rates over a 30-month follow-up compared to the 30 months prior to beginning CBT (d = − .92) and improved psychosocial functioning (d = .56), respectively. Five randomized controlled trials of CBT for bipolar patients receiving maintenance medication provided more convincing evidence of the prophylactic effects of CBT. In a psycho-educational study designed to promote the recognition of prodromes and prevent relapse (that contained some characteristics of CBT), Perry, Tarrier, Morriss, McCarthy, and Limb (1999) compared their psycho-educational treatment with a "treatment as usual" control group and found that the psycho-educational treatment increased time to the next manic relapse and reduced the number of manic relapses relative to the

control condition ($d = -.79$). Interestingly, the psycho-educational treatment did not affect depressive relapses. Lam et al. (2000) found that compared to a "treatment as usual" control group, a CBT group had significantly fewer bipolar episodes ($d = -.87$) and hospitalizations ($d = -.56$) throughout a 12-month follow-up and lower depressive ($d = -.39$) and manic symptoms ($d = -.85$) at 6 and 12 months post-treatment. Scott, Garland, and Moorhead (2001) reported that compared to a wait list control, their CBT group exhibited fewer relapses ($d = -1.19$) and hospitalizations ($d = -1.17$) during an 18-month follow-up, significant improvements in global functioning ($d = .62$) and in some symptoms (particularly, depressive symptoms; $d = -.47$), and greater medication adherence ($d = .85$). Cochran (1984) also found that their CBT plus lithium group showed greater medication compliance ($d = 1.12$) than a lithium-only group, as well as fewer mood episodes ($d = -.35$) and hospitalizations ($d = -1.68$) precipitated by medication non-adherence at 6-month follow-up. Lam et al. (2003) found that compared to a "treatment as usual" control group, their CBT group had significantly fewer bipolar episodes ($d = -.85$) and hospitalizations ($d = -.21$), fewer days in mood episodes ($d = -.80$), less residual depressive symptoms ($d = -.43$), and less manic symptom fluctuation ($d = -.39$) during the 12-month follow-up. Moreover, Lam et al. (2003) specifically targeted highly driven and extreme goal attainment beliefs in their CBT condition and found that the CBT group scored significantly lower than the control group on these beliefs at 6-month follow-up ($d = -.35$). Finally, one study (Zaretsky, Segal, & Gemar, 1999) compared the efficacy of CBT for managing an acute episode of bipolar depression to standard CBT for unipolar depression and found the two forms of CBT to be equally effective (d's $= -1.47$ and -1.30).

3.9. Summary of cognitive style findings

In sum, there is considerable evidence that individuals with bipolar disorders exhibit cognitive styles as negative as those with unipolar depression, consistent with an extension of cognitive theories of unipolar depression to bipolar disorder. Compared to unipolar depressed individuals, the cognitive styles of bipolar persons may be more uniquely characterized by goal striving, perfectionism, self-criticism, and autonomy, features characteristic of high BAS sensitivity, rather than dependency, attachment, and sociotropy. However, the strength of the association between negative cognitive styles and bipolar disorder may depend on the current mood state of bipolar individuals (depressed, manic, remitted) and whether the measures of cognition are explicit or implicit, although the evidence for the manic defense hypothesis is quite mixed. Clearly, further research is needed to more clearly establish the effects of current mood and type of cognition assessment on bipolar individuals' observed cognitive styles and whether bipolar individuals' cognitions are specifically BAS-relevant. In addition, consistent with an extension of cognitive theories of unipolar depression to bipolar disorder, there is considerable evidence that cognitive styles alone, and particularly in combination with relevant life events, prospectively predict the course of bipolar depression and more mixed evidence that they predict the course of bipolar mania/hypomania. Further longitudinal studies are needed to test the cognitive vulnerability–stress hypothesis for bipolar disorder and whether it applies equally well to mania as it does to depression. Perhaps greater focus on BAS-relevant cognitive styles will increase predictive power for mania/hypomania. Finally, there is a small body of consistent evidence that CBT has beneficial prophylactic effects on the course of bipolar disorder. However, the mechanism by which CBT works is as yet unknown. Further randomized controlled trials of CBT are needed that specifically test the active mechanisms of this promising adjunctive therapy for bipolar disorder.

4. Developmental factors and bipolar disorder

To date, the role that the early familial and non-familial environment plays in the development, expression, and course of bipolar disorder has been understudied. However, a small, but growing, body of literature has begun to address potential developmental factors in bipolar disorder and their similarity to those observed to be important in unipolar depression. Two lines of research have been conducted in this area: one on the parenting practices of bipolar individuals' parents and the other on the maltreatment histories of bipolar individuals. Both of these lines of research are characterized by important methodological limitations that make it difficult to draw firm conclusions regarding the role of these developmental factors in influencing the onset, expression, or course of bipolar disorder. First, all but one of the developmentally relevant studies have used retrospective designs, asking adult bipolar individuals to recall their childhood histories. Thus, even studies that obtain associations between parenting or abuse histories and bipolar disorder cannot determine whether these developmental factors were a causal contributor to or a consequence of the bipolarity. Second, and related, even with their retrospective designs, only three studies in this literature have attempted to examine whether these developmental factors preceded the onset of the bipolar disorder (and thus, whether they could have contributed to the bipolarity). Third, and also relevant to this issue, most studies do not control for bipolar participants' mood states at the time their childhood histories are assessed; consequently, reporting biases associated with current mood and symptoms cannot be ruled out in most cases. Fourth, some studies do not include an appropriate control group and, thus, cannot determine whether bipolar individuals' familial and non-familial histories differ from those of normal controls. Fifth, the operationalizations of parenting and maltreatment histories differ widely across studies, with some studies using measures of questionable reliability and validity (e.g., only one or two item indicators of childhood history). Finally, with only one exception, the studies in this area do not attempt to rule out third-variable explanations, such as shared genes, for the association between reported familial environment and bipolar disorder. Thus, with these caveats in mind, we review what is known about the developmental histories of bipolar individuals, making note of those methodologically stronger studies.

4.1. Parenting/attachment histories

Eight studies have examined the parenting and attachment histories of individuals with bipolar disorder. As a group, most of these studies have examined whether bipolar individuals' parents were characterized by low care and high overprotection or psychological control, a pattern of parenting dubbed "affectionless control" by Parker (1983) and reported in the families of unipolar depressed individuals (e.g., Alloy et al., 2001 for a review). In an early qualitative study, Davenport, Adland, Gold, and Goodwin (1979) interviewed six families including both a parent and adult child with bipolar I disorder. They noted that these families were characterized by avoidance of affect, the absence of intimate relationships apart from the family, domineering mothers, and emotionally or physically absent fathers. Four quantitative studies obtained no differences between the reported parenting of bipolar and comparison groups, although two of these found that parenting practices were associated with the course of bipolar disorder. Parker (1979) found that, on a self-report questionnaire, only a unipolar depressed group perceived both parents as less caring and their mothers as more overprotective than general practice controls; the bipolar and control groups did not differ in perceived parenting. Similarly, Perris, Arrindell, Van der Ende, and Knorr (1986), also using a self-report questionnaire, found that only

unipolar depressed patients reported less emotional warmth and greater rejection for both parents and more maternal overprotection than normal controls; the bipolar group did not differ from the controls. Although both Joyce (1984) and Cooke, Young, Mohri, Blake, and Joffe (1999) also did not obtain differences on parenting and family environment between bipolar and normal control groups, both studies found that within the bipolar group, familial environment was associated with the severity and course of the disorder. In Joyce (1984), bipolar individuals who reported low parental care and high overprotection on a self-report questionnaire had more hospitalizations for both depression and mania than those who did not. Cooke et al. (1999), using a family environment questionnaire, found that within the bipolar group, lower ratings of family expressiveness were associated with a history of comorbid dysthymia and lower ratings of family cohesiveness were associated with a history of past suicide attempts.

Three other methodologically stronger studies did find that bipolar individuals' parenting and attachments differed from those of normal controls. Using a self-report measure of parenting, Rosenfarb, Becker, and Khan (1994) reported that both bipolar and unipolar patients reported less maternal affection than normal controls, but bipolar patients did not differ from controls on paternal affection or over-control from either parent. On an explicit, self-report measure of attachment, both the bipolar and unipolar groups reported less attachment to their mothers than controls; bipolar patients did not differ from controls on either paternal or peer attachment. In contrast, on an implicit, projective attachment measure (family circle drawings), the bipolar group perceived less attachment to their fathers throughout all developmental stages than did the controls. In the only non-retrospective study, Geller et al. (2000) compared 7–16-year-old youth with bipolar disorder to both ADHD and community control groups on contemporaneous family and peer characteristics as assessed by semi-structured interview of both youth and their mothers. Bipolar youth evidenced greater impairment on parent–child interaction items indicating less maternal warmth and greater maternal and paternal tension/hostility, compared to the ADHD (r's = .25–.38) and community controls (r's = .38–.48). Also, bipolar youth had fewer friends and poorer social skills than the ADHD youth. Finally, in the only study to control for current depressive and manic symptoms as well as family history of mood disorder, Neeren, Alloy, and Abramson (2005) found that a bipolar spectrum group reported less warmth/acceptance (r's = .20–.27) and greater psychological control (r's = .27) for both parents on a self-report parenting questionnaire than did a demographically matched normal group.

4.2. Maltreatment histories

Eight studies have investigated the maltreatment histories (and other childhood stressors) of bipolar individuals; six of these did not include a normal control group. Mueser et al. (1998) examined the rates of overall trauma exposure (not just abuse) and PTSD among inpatients and outpatients with severe mental illness. They found that 98% of the patients reported exposure to at least one traumatic event, but the rate of PTSD was higher among unipolar depressed (58%) than among bipolar (40%) patients. Three other studies specifically compared the physical abuse (PA) and sexual abuse (SA) histories of bipolar and unipolar individuals and obtained mixed results. In a community survey using self-report questionnaire items to assess abuse, Levitan et al. (1997) found that bipolar individuals reported higher rates of childhood PA than unipolar depressed individuals, but the groups did not differ on childhood SA. In contrast, in an outpatient sample, based on a clinician-administered interview, Hyun, Friedman, and Dunner (2000) observed that bipolar patients reported a higher incidence of childhood SA than

unipolar patients ($r=.10$), but the groups did not differ on PA history ($r=.02$). In a study that is problematic because it used only a single item measure of PA and SA combined, Wexler, Lyons, Lyons, and Mazure (1997) reported that unipolar depressed outpatients (30%) reported higher rates of childhood abuse than bipolar outpatients (5%). Two other studies without normal comparison groups did find an association between childhood maltreatment and the expression or course of bipolar disorder. Hammersley et al. (2003) found no differences between bipolar patients with and without childhood SA histories on age of onset or first hospitalization, but bipolar patients with any type of trauma history were more likely to have auditory hallucinations than those with no trauma history ($r=.40$). In no case did the onset of bipolar disorder predate the reported trauma. However, a serious problem with this study is that trauma histories were not systematically assessed; instead, such histories were noted by clinicians if the patient happened to mention a trauma or abuse during therapy. Based on a self-report questionnaire of abuse, Leverich et al. (2002) studied a large sample of bipolar patients and found high rates of reported childhood PA and SA. A history of childhood PA or SA compared to no abuse was associated with a higher incidence of lifetime Axis I and II disorders, an early (≤ 14) age of onset (r's$=.25$ for PA and SA), and faster cycling frequencies (r's$=.10$ for PA and SA), including ultra-rapid cycling (4 episodes/month) and ultradian cycling (dramatic mood switches within a day). PA and SA were associated with an increased incidence of suicide attempts (r's$=.20$ and $.13$ for PA and SA) and a history of PA was associated with an increased severity of mania ($r=.20$). Finally, in a subset of bipolar patients followed prospectively for at least 1 year, those with childhood abuse compared to those with no abuse exhibited greater severity of course as reflected in a greater percent of time ill.

Three studies did utilize a comparison group of normal controls to examine childhood stressors, and two of these attempted to rule out report biases and genetic third variables by controlling for participants' current depressive and manic symptoms and family history of mood disorder. Coverdale and Turbott (2000) compared the prevalence of PA and SA occurring both during childhood and adulthood ($\geq$ age 16) in a sample of outpatients with schizophrenia and bipolar disorder to a demographically matched sample of medical outpatients with no psychiatric history. Bipolar diagnoses comprised only 15.6% of the patient sample. Combined childhood PA and SA did not differ between patients and controls (r's$=.01$–$.04$), but more patients reported combined adult PA and SA than controls (r's$=.10$–$.12$). Unfortunately, Coverdale and Turbott did not examine the rates of abuse for bipolar patients specifically. Controlling for current depressive and manic symptoms as well as family history of mood disorder and considering age of onset of bipolar disorder, Neeren et al. (2005) found that, on a very extensive maltreatment questionnaire, bipolar spectrum individuals reported more PA from mothers ($r=.10$) and more emotional abuse from both parents (r's$=.18$) prior to their age of onset than did demographically matched normal controls (prior to the same age). Similarly, Grandin, Alloy, and Abramson (2005) used a self-report questionnaire to examine childhood stressful events including maltreatment (both PA and SA), controlling for current depressive and manic symptoms and family history of mood disorder. In addition, Grandin et al. specifically examined separate associations between bipolar disorder and childhood stressors that were independent (fateful, uncontrollable) vs. dependent on the individual's behavior and that occurred prior to vs. after the age of onset of bipolar individuals' first mood episode (using the corresponding age for the matched normal control participant). They found that controlling for current symptoms and family history, only independent events occurring prior to the age of onset were associated with bipolarity (Odds Ratio=1.12), whereas bipolarity was associated with both independent and dependent events occurring after the age of onset. Moreover, a greater number of childhood stressors occurring before the age of onset actually predicted an earlier age of onset. Childhood maltreatment (PA

and SA combined) and achievement failure events were the only specific event categories to be associated with bipolar status after bipolar individuals' age of onset (r's = .35–.36).

4.3. Summary of developmental findings

The evidence relating parenting practices and maltreatment histories to bipolar disorder is decidedly mixed. There is some suggestion of parenting characterized by low care and high overprotection, poor attachment relations, and childhood abuse in the histories of individuals with bipolar disorder, but the studies conducted to date are inconsistent in supporting these associations. There is also some evidence that less than optimum parenting and maltreatment histories may be associated with a worse course of bipolar disorder. A major difficulty in this literature is that many of the studies suffer from serious methodological limitations such as retrospective designs, lack of control groups, absence of controls for current mood state, failure to consider family history, and failure to consider whether the developmental factors of interest preceded the bipolar disorder. The methodologically stronger studies seem to provide greater evidence of associations between parenting and maltreatment histories and bipolarity than do studies with greater limitations. Thus, firm conclusions regarding the role of early familial and non-familial environments in contributing to the emergence or course of bipolar disorder await further research that addresses these methodological issues.

5. Conclusion

Do current environmental context, cognitive styles, or developmental histories provide risk for the onset, course, or expression of bipolar disorders, and are these potential psychosocial risk factors similar to or different from those found to be important in contributing vulnerability to unipolar depression? Our conclusions must be tentative until such time as the methodological limitations characteristic of the psychosocial risk factors literature in bipolar disorder are more fully addressed. More prospective, longitudinal studies are needed with adequately sized samples of bipolar individuals, normal control groups, controls for initial mood state and symptoms, controls for genetic predisposition or use of genetically informative designs (e.g., prospective twin studies), standardized and well-validated measures of the psychosocial risk factors, separate examination of depressive and manic/hypomanic episodes, and direct comparison with unipolar depressed samples. In addition, because they involve experimental manipulation of psychosocial variables, psychosocial treatment studies are in a position to contribute importantly to our knowledge of risk factors for bipolar disorder. However, to do so, future treatment studies will need to begin to focus on research designs and assessment strategies that address the mechanisms of change operating within psychosocial therapy regimens.

With these caveats in mind, there is fairly consistent evidence from prospective studies that recent life events and supportive interpersonal relationships predict the likelihood of onsets and recurrences of bipolar mood episodes, as they do for onsets and recurrences of unipolar depression. In addition, psychosocial treatments designed to decrease interpersonal stressors and destabilization of daily rhythms (IPSRT) or to improve family communication skills and social support (FFT) show great promise as adjunctive treatments for bipolar disorder. Further exploration of specific types of life events, such as social rhythm disrupting and BAS-relevant events, that may provide specific risk for bipolar mood episodes is clearly warranted. The cognitive style literature provides some consistency in suggesting that

bipolar individuals' cognitive styles may be as negative as those of unipolar depressed individuals, although perhaps more uniquely characterized by styles reflective of high BAS sensitivity. Also, prospective studies suggest that cognitive styles alone and in combination with relevant life events predict onsets and recurrences of bipolar depressive episodes and sometimes, of manic episodes, as they do for episodes of unipolar depression. Moreover, CBT studies indicate that treatments based on cognitive principles also show great promise as adjuncts to pharmacotherapy for bipolar disorder. The literature on parenting and maltreatment histories as risk factors is only just developing, contains no prospective studies, and is less consistent overall. However, there are initial findings suggesting that early environmental risk factors for bipolar disorder are worth further exploration. The exploration of psychosocial risk approaches to bipolar disorder is only at its beginning. We hope that future investigators will be inspired by the current review to conduct further, more sophisticated studies of the role of psychosocial risk factors in the onset, course, and expression of bipolar spectrum disorders.

Acknowledgements

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