

Chapter 2

Psychosocial risk factors for bipolar disorder

Current and early environment and cognitive styles

Lauren B. Alloy, Lyn Y. Abramson, Amy M. Neeren, Patricia D. Walshaw, Snezana Urosevic and Robin Nusslock

What is transmitted are these affectively disregulated temperaments and that the progression to full-blown bipolar illness is due to environment.

(Akiskal, 1986, p. 671)

About 1.5 per cent of the US population will experience bipolar disorder (Hyman, 2000). Often, bipolar illness is a severe, recurrent or unremitting disorder with significant impairment such as divorce, alcohol abuse, suicide, and erratic work history (Goodwin and Jamison, 1990). Indeed, bipolar disorder has been ranked as the sixth leading cause of disability among all disorders worldwide (Murray and Lopez, 1996). Within the bipolar category, there is a continuum or spectrum of severity from the milder, subsyndromal cyclothymia, to Bipolar II disorder, to full-blown Bipolar I disorder (Cassano *et al.*, 1999). Kraepelin (1921) emphasized that the milder bipolar conditions not only are on a continuum with Bipolar I disorder but actually may be precursors to it. Research has provided support for this continuum model (Akiskal, Djenderedjian, Rosenthal, and Khani, 1977; Akiskal, Khani, and Scott-Strauss, 1979; Depue *et al.*, 1981; Goodwin and Jamison, 1990; Klein, Depue, and Slater, 1985; Waters, 1979).

Like the disorder itself, research and theory on bipolar disorder has swung back and forth like a pendulum between the psychological and biological perspectives. Although early clinical reports emphasized the psychosocial

context of the disorder (Kraepelin, 1921), conceptions of bipolar disorder as a genetically based, biological illness dominated over the past century. Indeed, the data from family, twin, and adoption studies suggesting that bipolar disorder has a strong genetic predisposition (Goodwin and Jamison, 1990; Nurnberger and Gershon, 1992) and the successful use of lithium to treat individuals with the condition began to shift the focus to the disorder's biological underpinnings. More recently, as researchers have recognized both the inability of genetic and biological processes to fully account for differences in the expression of the disorder or the timing and polarity of symptoms (O'Connell, 1986) and the limitations of the prophylactic effects of lithium, the pendulum has begun to swing back again toward the inclusion of psychosocial factors in bipolarity research. In fact, a 1990 NIMH workshop report (Prien and Potter, 1990) called for an increased focus on the impact that psychosocial factors have on the course of bipolar disorder. In line with the return of interest in the psychosocial context, recent evidence suggests that environmental and psychological factors may play an important role in the course and expression of bipolar spectrum disorders.

Consequently, in this chapter, we review empirical research on the role of individuals' current and early environment and cognitive/personality styles as risk factors for the onset, course, and expression of bipolar spectrum disorders. Our review is focused on an overarching question: Do psychosocial factors truly contribute to the onset, course, or expression of bipolar disorders? And, do they do so above and beyond the role of genetic predisposition? We begin by discussing the methodological challenges the bipolar spectrum disorders pose for psychosocial risk research. Next, we review the extant studies on the role of recent life events and supportive and nonsupportive social contexts (current environment), followed by studies of parenting and maltreatment histories (early environment) in bipolar disorders. 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. We conclude with an assessment of the state of the psychosocial risk factors literature in bipolar disorder with regard to our guiding question.

Methodological challenges of psychosocial risk research in bipolar spectrum disorders

If a psychosocial factor contributes risk to the onset, course, or expression of bipolar disorders, it must meet two criteria (Alloy, Abramson, Ranieri, and Dyller, 1999a; Ingram, Miranda, and Segal, 1998): (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 studies that compare bipolar individuals with a normal control group or with a group with another disorder (e.g., unipolar depression) can generate hypotheses about potential risk factors, but are inadequate for establishing temporal precedence or stability independent of bipolar symptoms. An improvement over cross-sectional studies are studies that compare euthymic or remitted bipolar individuals to a normal control group 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, and Alloy, 2001; Lewinsohn, Steinmetz, Larson, and 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 independence from symptoms and temporal precedence (Alloy *et al.*, 1999a). Unfortunately, there are relatively few prospective studies of the role of current and early environmental factors and cognitive styles in the course and expression of bipolar disorders. Thus, in our review, we note these more important prospective studies.

Moreover, if one is to demonstrate that a psychosocial risk factor truly contributes risk to bipolar disorder, one must in some way rule out the third variable genetic explanation for any findings suggestive of psychosocial risk. This is important because genetic vulnerability as expressed in temperament or other behavioural 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 and Crabbe, 2000). Almost no studies to date even 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 bipolar disorder.

From a methodological perspective, bipolar spectrum disorders present especially challenging problems for demonstrating psychosocial risk. First, these disorders are highly recurrent with significant inter-episode 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 inter-episodic 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 judgement, poor coping skills, and other symptoms (Alloy *et al.*, 1999a; Hammen, 1991; Johnson and 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' behaviour, 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 and early environment and cognitive styles as risk factors must remain somewhat tentative.

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 *et al.*, 2005a; Alloy, Reilly-Harrington, Fresco, and Flannery-Schroeder, in press, b; Johnson and Roberts, 1995; Johnson and Kizer, 2002). Studies of the role of the current environment in bipolar disorder have focused on two kinds of environmental factors: 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. 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. AQ1

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 and Harris, 1979) 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. Second, most studies do not control for any reporting biases associated with bipolar individuals' mood AQ2

state at the time they are reporting life events or social support. Third, some studies rely on self-report measures of life events, social support, and/or symptoms, which can compound the potential problems of mood-based report biases. 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. Eighth, 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). Finally, in the life events literature, many studies also have failed to differentiate between events that are independent of or dependent on people's behaviour, a distinction of considerable importance given the chaotic lifestyles of individuals with bipolar disorders. 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 (see also Chapter 6).

Recent life events and bipolar disorder

Overall, studies of the role 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 review the more methodologically limited cross-sectional and retrospective studies first, followed by the stronger prospective studies. In addition, 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.

Four studies relied on retrospective review of medical charts to assess life events in patients with bipolar disorder. Leff, Fischer, and Bertelson (1976) found that 35 per cent of bipolar inpatients reported a stressful event rated as

independent of their behaviour in the month prior to onset of episode. Clancy, Crowe, Winokur, and Morrison (1973) found that 39 per cent of unipolar, 27 per cent of bipolar, and 11 per cent of schizophrenic patients had a stressful event in the three months prior to onset of their disorder. No significant differences were found for types of precipitating stressful events for bipolar vs. unipolar patients. Ambelas (1979, 1987) conducted two retrospective chart review studies. In the 1979 study, 28 per cent of 67 hypomanic or manic inpatients vs. 6 per cent of 60 surgical control patients had experienced an independent stressful event during the four weeks prior to hospital admission. In almost all the cases reported, the stressful event precipitating mania or hypomania was a loss or threat event. In the 1987 study of 90 bipolar manic inpatients, compared with 8 per cent of an age-matched surgical control group, 66 per cent of first episode and 20 per cent of repeat admission bipolar patients reported a severe independent event in the four weeks before admission.

An improvement over retrospective chart reviews is represented by three retrospective studies that administered questionnaires to bipolar individuals regarding their past life events. Bidzinska (1984) reported that acute and chronic stress preceded the onset of illness in 90 per cent of bipolar and 89.4 per cent of unipolar patients. However, bipolar patients reported more work-related stressors than did unipolar patients. Kulhara, Basu, Mattoo, Sharan and Chopra (1999) reported that the frequency of episode relapses was predicted by the number of life events and total stress experienced in 118 bipolar inpatients. Poor lithium responders also experienced more life events and total stress than good lithium responders. Dunner, Patrick, and Fieve (1979) distinguished between manic and depressive episodes and found that about half of 79 bipolar patients recalled a stressful event in the three months before their initial episode; an increase in work and interpersonal difficulties was associated with onset of a manic vs. a depressed episode.

Six retrospective studies incorporated further methodological improvement by including interview assessments of life events or online experience sampling of events, but only some assessed the independence of the events from bipolar individuals' behaviour. Glassner, Haldipur, and Dessauersmith (1979) interviewed 25 bipolar patients and their relatives about the patients' life events preceding their first and most recent mood episodes. They found that 75 per cent of first episode and 56 per cent of later episode patients reported a stressful event prior to onset. Using the same methodology with 46 bipolar patients and their relatives, Glassner and Haldipur (1983) reported that 64 per cent of late onset (after age 20) vs. 23 per cent of early onset bipolar patients reported a stressful event preceding their initial episode. Myin-Germeys *et al.* (2003) conducted an experience sampling study of non-affective psychotic,

bipolar, major depressed, and control participants 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 all other groups and had the largest decrease in positive affect in response to stress.

The three retrospective studies that examined stressful events independent of individuals' behaviour also found that bipolar individuals experienced increased stress prior to episode onsets. In a study of bipolar patients that capitalized on the occurrence of a natural disaster, Aronson and Shukla (1987) found a significant increase in relapse two weeks after a major hurricane. However, relapsers also had less symptom stability before the hurricane than did non-relapsers. Similarly, Davenport and Adland (1982) reported a 50 per cent onset rate of mood disorder episodes among 40 bipolar men during or immediately following their wives' pregnancies. In a sample of remitted depressed bipolar, unipolar, and neurotic-reactive patients retrospectively assessed over a one year interval, Perris (1984) found that neurotic patients reported more independent events in the year prior to episode onset than did bipolar patients who, in turn, reported more pre-onset events than unipolar depressed patients.

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Five retrospective studies employing life events interviews specifically examined the role of independent stressors in onsets of manic episodes. Kennedy, Thompson, Stancer, Roy, and Persad (1983) found that, compared to control participants or to the period following hospital admission, manic patients experienced twice as many independent negative events during the four months prior to hospital admission. Joffe, MacDonald, and Kutcher (1989) matched 14 recently relapsed manics to more stable bipolar patients and also found significantly more uncontrollable and unexpected life events among the relapsers prior to onset. Using both a retrospective and prospective design, Sclare and Creed (1990) reported that manic patients experienced more independent events prior to onset than after recovery. Bebbington *et al.* (1993) found that psychotically depressed patients experienced more severe, independent life events in the six months prior to onset of psychosis than did both manic and schizophrenic patients. However, the manic patients also reported more severe, independent events prior to relapse than did nonpsychiatric controls. In contrast, Chung, Langeluddecke, and Tennant (1986) found that the rate of independent threatening events in the 26 weeks prior to onset for 14 manic patients did not differ significantly from that of controls (although the rate was twice as high in the manics).

The eleven methodologically sounder prospective studies provide stronger, although 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 the overall numbers of events did not differ for bipolar patients who relapsed vs. those who did not, hypomanic relapsers had greater numbers of work-related events than did non-relapsers. In another study using a questionnaire assessment of life events every three months for up to three years, Christensen *et al.* (2003) studied 56 bipolar I and II patients, diagnosed based on medical records. The bipolar women, but not the men, experienced a greater number of events in the three 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 three-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 undergraduates. The cyclothymic group had significantly greater stress than the intermittent depressive group, 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, whereas women with at least one life event in the 12 months pre-onset were at higher risk for non-psychotic relapses. 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 eight weeks after a life event and following events dependent on patients' behaviour.

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 two years, with interviews assessing life events and mental state every three months, Hunt, Bruce-Jones, and Silverstone (1992) reported that 19 per cent of 52 relapses were preceded by a severe event in the previous month, compared to a background rate of 5 per cent of patients with a severe event each month at other times. Manic and depressive relapses did not differ in the rate of prior events. In contrast, using similar methods, McPherson, Herbison, and Romans (1993) found no difference in the number of moderately severe, independent events in the month preceding relapse as compared with control periods. The McPherson *et al.* study was limited by a high dropout rate and the absence of a required well

period prior to study entrance. 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 two months. Bipolar and unipolar patients who relapsed didn't report more life events in the two months before the relapse compared to those who didn't relapse, but among the bipolar patients, those with a manic/hypomanic relapse had more marital stressors in the two months prior to the relapse than other bipolar patients. In a study of 61 bipolar outpatients followed over a two-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. 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 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, and Kupfer, 1988; Healy and 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, Malkoff-Schwartz *et al.* (1998, 2000) reported that bipolar patients in a manic episode were significantly more likely to experience pre-onset events characterized by social rhythm disruptions than depressed bipolar or unipolar patients. Kadri, Mouchtaq, Hakkou, and Moussaoui (2000) did not actually assess social rhythms, but found that 45 per cent of 20 Muslim bipolar patients relapsed during Ramadan (the Muslim fasting month with significant changes in social rhythms, i.e., no meals), with 71.4 per cent of these relapses of manic

polarity. In a prospective study of 206 bipolar spectrum participants (bipolar II, cyclothymic), Shen, Alloy, and Abramson (2005) found that less regular social rhythms at Time 1 predicted onsets of major depressive and hypomanic/ manic episodes during 20 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. However, there was no significant relationship between daily social rhythms and mood, 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 and Iacono, 1989; Fowles, 1987; Gray, 1991; Johnson *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 affect, high energy, and motivation (i.e., mania/ hypomania) to events involving high incentive motivation and goal striving or attainment and with too extreme negative affect, low energy, and anhedonia (i.e., depression) to events involving uncontrollable loss and failure. Consistent with this hypothesis, Johnson *et al.* (2000) found that goal attainment events predicted increases in manic symptoms, but not depressive symptoms, among 43 bipolar I patients over the prospective follow-up, whereas general positive events did not predict increases in manic symptoms. Nusslock *et al.* (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 *et al.* (2000), Nusslock *et al.* found that individuals in the bipolar spectrum (bipolar II, cyclothymia) were especially likely to develop new onsets of hypomanic, but not depressive, episodes during the final exam period compared to a prior control period.

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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 three-month pre-onset period than patients with a first depressive episode, consistent with the kindling hypothesis. Johnson *et al.* (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 event pre-episode onset decreased across episodes and for bipolar patients, 63 per cent experienced an event prior to the first episode vs. only 30 per cent prior to the fifth episode. 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 nine episodes, with the greatest difference over the first three 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 ageing process rather than illness progression may 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 three 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. Consequently, 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, but more prospective studies are needed to draw definitive conclusions about the kindling hypothesis.

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Social support and bipolar disorder

In addition to the effect of recent life events, supportive or non-supportive interpersonal relationships are another important aspect of an individual's current environment that affects the course of bipolar disorder. 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.

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. Romans and McPherson (1992) administered a social support interview to 52 euthymic bipolar I participants and 232 community controls. The bipolar group reported less social support than the controls, but they weren't significantly different from the controls with past psychopathology. Predominantly manic bipolars had less adequate attachments and less available social integration than predominantly depressive bipolar individuals. Bauwens, Tracy, Pardoën, Vander Elst, and Mendlewicz (1991) compared 27 bipolar and 24 unipolar depressed individuals, all in remission with 26 normal controls on a social support interview. Both bipolar and unipolar patients scored lower on social adjustment than the controls. Within the bipolar group only, social maladjustment was related to current symptoms and the number of lifetime mood episodes. Similarly, in a sample of 118 bipolar inpatients, Kulhara *et al.* (1999) found that lower social support (assessed via questionnaire) was associated with a higher frequency of lifetime relapses. Beyer *et al.* (2003) compared 29 older bipolar I (ages 50–89) and 56 younger bipolar I (ages 18–49) individuals with two peer control groups (33 young and 23 older) on a self-report social support measure. Both older and younger bipolar participants perceived lower social support compared to their peer controls and currently manic bipolars perceived less support than currently depressed or euthymic bipolars. In the retrospective study, Stefanos, Bauwens, Staner, Pardoën and Mendlewicz (1996) examined the association between interview assessments of social support and relapses over the past three years estimated from medical charts in 21 remitted bipolar patients. Relapses were significantly associated with low social support, maladjustment in social activities, 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 one 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 and colleagues (Johnson, Winett, Meyer, Greenhouse, and Miller, 1999; Johnson *et al.* 2000) found that poorer social support predicted longer time to recovery and prospective depressive, but not manic, 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 and assessed

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with two social support questionnaires, Johnson, Lundstroem, Aberg-Wistedt, and Mathe (2003) reported that relapsers had lower social support than non-relapsers on one measure 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 of a family environment scale. Miklowitz, Goldstein, and Nuechterlein (1995) examined the family interactions of 42 schizophrenic and 22 bipolar I patients and found that schizophrenic patients' relatives made more intrusive statements than bipolar patients' relatives, bipolar patients made more supportive statements about their relatives than did schizophrenic patients, and among the bipolars, those with higher hostility/suspicion had relatives who made more intrusive statements. Koenig, Sachs-Ericsson, and Miklowitz (1997) examined 31 bipolar I patients' ratings of a family interaction with their relatives. Patients' ratings of the interactions were correlated with observers' ratings. Bipolar patients with greater critical or intrusive ratings of their relatives' statements reported more distress, 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 48 bipolar I patients during a family interaction. Bipolar patients from high EE families had more manic symptoms and a trend toward more depressive symptoms than those from low EE families. In addition, high EE families were more likely than low EE families to show sequences in which a relative's negative statement led to the patient's negative statement, leading, in turn, to a second relative's negative statement. Finally, Wendel, Miklowitz, Richards, and George (2000) assessed both EE levels and attributions (causal beliefs) of relatives during family interactions with 52 bipolar I patients. High EE relatives' causal attributions for the patient's role in negative events were more personal and controllable than those of low EE relatives (that is, they tended to believe that the patients were more responsible for causing negative events).

Three prospective studies found that high EE among relatives is predictive of a worse course of bipolar disorder. Miklowitz, Goldstein, Nuechterlein, Snyder, and Mintz (1988) assessed EE with the Camberwell Family Interview (CFI) and affective style (guilt-inducing, critical, and intrusive statements from relatives during a family interaction) in the relatives of 23 bipolar and schizoaffective manic inpatients. High EE approached significance in predicting

patients' relapse over nine months, controlling for affective style and affective style significantly predicted relapse, controlling for EE. Neither predicted depressive vs. manic relapses. Priebe, Wildgrube, and Muller-Oerlinghausen (1989) examined relatives' EE with the CFI and followed 21 mostly bipolar patients for 9 months. Patients with high EE relatives had eight times the prospective morbidity rate (hospital admissions, symptoms, additional medications) as patients with low EE relatives. Rosenfarb *et al.* (2001) examined relatives' affective style during a family interaction with the patient in a sample of 27 bipolar I patients followed for nine months. Relatives of patients who relapsed made more critical and supportive statements during the interactions than did relatives of non-relapsers. In addition, among the relapsing group, relatives' criticism was positively correlated with patients' unusual thoughts during the interaction (see Chapter 5 for a discussion of the clinical implications of these findings).

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 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 *et al.*, 2005; in press, b; Johnson and Roberts, 1995), indicates 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 bipolars' mood episodes. 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 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 behavior (Johnson *et al.*, 2000; Urosevic *et al.*, 2005).

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The social support/EE literature is also reasonably consistent in indicating that positive vs. negative interactions with family and friends has an important impact on the course of bipolar disorder. 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. 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, 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.

Early environment and bipolar disorder: Role of parenting and maltreatment histories

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 body of literature has begun to address potential developmental antecedents of bipolar disorder. Two lines of early environment research have been conducted: one on the parenting practices of bipolar individuals' parents; and the other on the maltreatment histories of bipolar individuals. Both lines of research are characterized by important methodological limitations. 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 cause or a consequence of the bipolarity. Second, only three studies 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, 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 to allow for a determination of whether bipolar individuals' histories differ from those of normals. 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 early environments of bipolar individuals.

Parenting/attachment histories

Eight studies have examined the parenting and attachment histories of individuals with bipolar disorder. 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 depressives (see Alloy *et al.*, 2001 for a review). In an early qualitative study with no control group, Davenport, Adland, Gold, and Goodwin (1979) interviewed six families including both a parent and adult child with bipolar I disorder. These families were characterized by avoidance of affect, the absence of intimate relationships apart from the family, mothers who were domineering, and fathers who were emotionally or physically absent. 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) compared bipolar to unipolar and general practice outpatients and found that only the unipolar depressed group perceived both parents as less caring and their mothers as more over-protective than controls. Similarly, Perris, Arrindell, Eisenmann, Van Der Ende, and Knorr (1986) found that only unipolar depressed patients reported less emotional warmth and greater rejection for both parents and more maternal over-protection than normal controls; the bipolar group did not differ from controls. Although neither Joyce (1984) and Cooke, Young, Mohri, Blake, and Joffe (1999) obtained differences on parenting and family environment between bipolar and normal 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 had more hospitalizations for both depression and mania than those who did not. In Cooke *et al.* (1999), 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. AQ8

Three other methodologically stronger studies did find that bipolar individuals' parenting and attachments differed from those of normal controls. Rosenfarb, Becker and Khan (1994) compared bipolar, unipolar, and normal control participants on measures of parenting and both explicit and implicit measures of attachment. With regard to parenting, both bipolar and unipolar patients reported less maternal affection than controls, but bipolars did not differ from controls on paternal affection or overcontrol from either parent. On the explicit attachment measure, both the bipolar and unipolar groups

reported less attachment to their mothers than controls; bipolars did not differ from controls on either paternal or peer attachment. On the implicit attachment measure, the bipolar group perceived less attachment to their fathers than did the controls. In the only non-retrospective study, Geller *et al.* (2000) compared 7–16-year-old bipolar youths to both ADHD and community control groups on their contemporaneous family and peer characteristics as assessed by both youths and maternal report. Compared to the ADHD and control groups, bipolar youths evidenced greater impairment on parent–child interaction items, indicating less maternal warmth and greater maternal and paternal tension/hostility. In addition, bipolar youths had fewer friends and poorer social skills than the ADHD youths. Finally, in the only study to control for both current depressive and manic symptoms as well as family history of mood disorder, Neeren, Alloy, Abramson, Pieracci, and Whitehouse (2005) compared the reported parenting of bipolar spectrum individuals and demographically matched normal controls. Controlling for current symptoms and family history, the bipolar group reported less warmth/acceptance and greater psychological control for both parents than did the control group.

Maltreatment histories

Eight studies 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 patients with severe mental illness. They found that 98 per cent of the patients reported exposure to at least one traumatic event, but the rate of PTSD was higher among unipolar depressed (58 per cent) than among bipolar (40 per cent) 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, Levitan *et al.* (1997) found that bipolar individuals reported a greater rate of childhood PA than did unipolar depressives, but the groups did not differ on childhood SA. In contrast, in an outpatient sample, Hyun, Friedman, and Dunner (2000) observed that bipolar patients reported a higher incidence of childhood SA than unipolar patients, but the groups did not differ on PA history. 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 per cent) reported higher rates of childhood abuse than bipolar outpatients (5 per cent). 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) studied 96 bipolar patients undergoing a

trial of cognitive-behaviour therapy and found no differences between those with and without childhood SA histories on age of onset or first hospitalization, but bipolar patients with a trauma history predating their bipolar disorder were more likely to have auditory hallucinations than those with no trauma history. However, trauma histories were not systematically assessed in this study; instead, such histories were noted if the patient happened to mention a trauma or abuse during therapy. 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 age of onset (< 14), and faster cycling frequencies. PA and SA were associated with an increased incidence of suicide attempts and a history of PA was associated with an increased severity of mania. Finally, in a subset of bipolar patients followed prospectively for at least one year, those with childhood abuse compared to those with no abuse exhibited a greater percent of time ill.

Two studies did utilize a normal comparison group to examine childhood stressors. Coverdale and Turbott (2002) compared the rate of PA and SA occurring during childhood ($< \text{age } 16$) and adulthood in a sample of bipolar and schizophrenic outpatients to demographically matched medical outpatients with no psychiatric history. Bipolar diagnoses comprised only 15.6 per cent of the patient sample. Combined childhood PA and SA did not differ between patients and controls, but more patients reported combined adult PA and SA than controls. Unfortunately, Coverdale and Turbott did not examine the rates of abuse for bipolar patients specifically. Grandin, Alloy and Abramson (2005) examined childhood stressful events including maltreatment (both PA and SA) in a large sample of bipolar spectrum individuals and demographically matched normal controls. This study was the only one to attempt to rule out report biases and genetic third variables by 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 behaviour and that occurred prior to vs. after the age of onset of bipolar individuals' first mood episode (using the corresponding age cut-off for the matched normal participant). Controlling for current symptoms and family history, only independent events occurring prior to the age of onset were associated with bipolarity, whereas neither independent nor dependent events occurring after the age of onset were related to bipolarity. 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.

Summary of early environment findings

To summarize, 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 over-protection, 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, lack of controls for current mood state, failure to consider family history, and failure to consider whether the developmental variables preceded the bipolar disorder. The methodologically stronger studies provide greater evidence of associations between parenting and maltreatment history 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.

Cognitive styles and bipolar disorder

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 and information processing similar to that observed among unipolar depressives; and whether these cognitive patterns, alone or in combination with life events, predict the expression or course of bipolar disorder (Alloy *et al.*, 2005a, in press, a, b). Studies of cognition in bipolar disorder have been guided by two main theoretical perspectives: logical extensions of the cognitive models of unipolar depression (Abramson, Metalsky, and Alloy, 1989; Beck, 1967, 1987) and the ‘manic defence’ hypothesis, a psychodynamic model (Abraham, 1911/1927; Dooley, 1921; Freeman, 1971; Klein, 1994; Rado, 1928) more recently updated in cognitive-behavioural terms by Neale (1988).

Given the success of cognitive models in contributing to the understanding of the aetiology, course, and treatment of unipolar depression, the logic of these theories has been extended to bipolar disorders (Alloy *et al.*, 2005a, in press a, b; Alloy *et al.*, 1999b; Hammen, Ellicott, and Gitlin, 1992; Newman, Leahy, Beck, Reilly-Harrington, and Gyulai, 2002; Reilly-Harrington, Alloy, Fresco, and Whitehouse, 1999). Cognitive models of unipolar depression focus on maladaptive cognitive patterns (negative styles for inferring causes,

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consequences and self-worth implications in Hopelessness theory [Abramson *et al.*, 1989] and negative self-schemata, dysfunctional attitudes, and socio-tropic and autonomous personality modes in Beck's [1967; 1987] theory) as vulnerabilities for depression when individuals experience stressful life events. Such maladaptive cognitive styles increase the likelihood of negative appraisals and processing of negative life events, 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 of bipolar individuals following negative events. With respect to risk for the manic/hypomanic episodes of bipolar individuals, two types of predictions may follow from an extension of cognitive theories of unipolar depression (Alloy *et al.*, in press, a). On one hand, bipolar individuals may also possess positive cognitive styles that increase risk for mania/hypomania when 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 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.

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Psychodynamic formulations suggesting that the grandiosity of mania is a 'defence' or counter-reaction to underlying depressive tendencies (e.g., Abraham, 1911/1927; Dooley, 1921; Freeman, 1971; Klein, 1994; Rado, 1928) and recent cognitive reconceptualizations of this 'manic defence' hypothesis (Neale, 1988) are consistent with the potential relevance of negative, depressive cognitive styles to mania/hypomania (see Chapter 11 for further discussion of these issues). According to Neale (1988), life events that are 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 are ruled by 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 defence mechanism, depression results. On the other hand, mania results from a reactance to helplessness and threatened self-esteem with a last extreme effort to regain control and mastery. Tests of the manic defence hypothesis depend on a comparison of 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 literature on the cognitive styles associated with bipolar disorders and predictive of the course of bipolar disorders, alone and in combination with life events. Most studies are cross-sectional and only relevant to examining the cognitive styles characteristic of bipolar individuals and their similarity to those of unipolar depressives. Only a few 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 examine the nature of cognitive styles in bipolar individuals independent of mood states and symptoms of the disorder (Alloy *et al.*, in press, a). 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 a depressive vs. manic episode; and by conducting within-subject longitudinal studies of the same bipolar individuals in different mood states. 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 explicit or implicit (Alloy *et al.*, in press, a). Most studies indicate that bipolar individuals exhibit cognitive patterns as negative as those of unipolar depressives (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. Finally, the majority of studies in this area suffer from one or more additional methodological 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.

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Cross-sectional studies: Cognitive styles in a depressed state

Five studies compared bipolar depressed and unipolar depressed groups on cognitive styles and all but one found no differences between the two groups' cognitive styles. Both depressed bipolar and unipolar participants showed equally negative dysfunctional attitudes, automatic thoughts, attributional styles, and self-referent information processing characteristic of depression and more negative than normal comparison groups (Hill, Oei, and Hill, 1989;

Hollon, Kendall, and Lumry, 1986; Reilly-Harrington *et al.*, 1999). Rosenfarb, Becker, Khan, and Mintz (1998) observed that both depressed unipolar and bipolar women were more self-critical than controls, but only the unipolar depressed women were more dependent than controls. 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 are as negative as those of unipolar depressives.

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 and Chapman, 1986), tested the manic defence hypothesis. Failing to support the hypothesis, Thompson and Bentall (1990) found that high levels of hypomania were associated with global attributions for both positive and negative events on an explicit attributional style measure. However, consistent with the manic defence hypothesis, using an implicit emotional Stroop test involving naming the ink colours 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 colour of depression, but not euphoria, words. These findings were replicated by French, Richards, and Scholfield (1999) controlling for the effects of anxiety on Stroop performance.

Cross-sectional studies: Cognitive styles in a remitted/euthymic state

Eleven studies assessed the cognitive styles of remitted or euthymic bipolar individuals. Across these studies, observations of negative cognitive styles among remitted bipolar individuals did not depend on whether the cognitive measures are explicit or implicit. Five studies using primarily explicit measures obtained little evidence of negative cognitions in the remitted state. In two studies of the same sample (Pardo, Bauwens, Tracy, Martin, and Mendlewicz, 1993; Tracy, Bauwens, Martin, Pardo, and 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 depressives. 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 measures of cognition, obtained more support for negative cognitive styles among remitted bipolar individuals. In the only remitted study directly supportive of the manic defence hypothesis, Winters and Neale (1985) found that remitted bipolar patients exhibited higher self-esteem than remitted unipolar patients and normal controls on explicit measures, but provided attributions as negative as the remitted unipolar patients on an implicit inference task. Alloy *et al.* (1999b) reported that euthymic cyclothymic and dysthymic participants did not differ from each other and exhibited more negative attributional styles and dysfunctional attitudes than hypomanic and normal participants. Scott, Stanton, Garland, and Ferrier (2000) found that remitted bipolar patients and normal controls were similar on explicit self-esteem, but the remitted bipolar patients exhibited more explicit dysfunctional attitudes, greater sociotropy and autonomy, fewer solutions on a social problem-solving task, and greater implicit over-general recall on an autobiographical memory task. 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, but only the remitted unipolar women were also more dependent than the controls. Lam, Wright, and Smith (2004) conducted a factor analysis of dysfunctional attitudes in a bipolar I sample to yield goal attainment, achievement, and dependency factors. Euthymic bipolar patients only scored higher than euthymic unipolar patients on goal attainment dysfunctional attitudes. Finally, controlling for concurrent depressive and hypomanic symptoms, Abramson *et al.* (2005) found that euthymic bipolar participants exhibited more negative inferential styles, dysfunctional attitudes (only the perfectionism subscale), autonomy, self-criticism, private self-consciousness, and rumination than did normal controls. Euthymic bipolar participants and normal controls 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 set of negative cognitive styles consistent with the high drive/incentive motivation associated with high BAS sensitivity, but not by

dependency and attachment attitudes typically observed among unipolar depressives.

Cross-sectional studies: Comparisons of cognitive styles across mood states

Five studies compared depressed bipolar to other 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 cognitive measures. Ashworth, Blackburn and McPherson (1982) and Hayward, Wong, Bright, and Lam (2002) both found that bipolar manics had higher explicit self-esteem than bipolar depressives, although in Ashworth *et al.* (1982), neither group's self-esteem differed significantly from nonpsychiatric controls. Scott and Pope (2003) reported that hypomanic bipolar participants had higher levels of both negative and positive self-esteem than depressed or remitted bipolar participants, whereas hypomanic bipolar participants had higher dysfunctional attitudes than remitted bipolars, but lower than depressed bipolars. Lyon, Startup and Bentall (1999) employed both explicit and implicit measures of cognitive style. On the explicit measures, manic 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). However, consistent with the manic defence hypothesis, on the implicit tests, manic patients, like depressed bipolar patients, attributed negative events internally on the Winters and Neale (1985) pragmatic inference task, showed slower colour-naming for depression-related rather than euphoria-related words on the emotional Stroop task, and recalled more negative than positive words on the self-referent incidental recall task. Finally, Murphy *et al.* (1999) administered an emotional-attention-shifting task and found that manic patients exhibited positive mood congruent attentional bias, whereas bipolar depressed patients showed negative mood congruent attentional bias.

Longitudinal studies: Stability of cognitive styles within individuals across moods

Only three studies to date have used longitudinal designs to investigate the actual stability of cognitive patterns across different mood states of the same bipolar individuals. Ashworth *et al.* (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 in both depressed and manic states. Although their design was longitudinal, their analyses were between- rather than within-subjects. Mood dependent recall occurred in both depressed and manic states, but patients generated more positive than negative autobiographical events when manic and more negative than positive events when depressed. Alloy *et al.* (1999b) assessed attributional styles, dysfunctional attitudes, and more state-like self-perceptions in diagnosed cyclothymic, dysthymic, hypomanic, and normal control participants on three separate occasions across different mood states (euthymic, depressed, and hypomanic) characteristic of their disorders. 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. In contrast, self-perceptions varied across moods and were more positive in hypomanic than depressed moods.

Longitudinal studies: Cognitive styles as predictors of bipolar course

Do cognitive styles alone or in combination with life events predict the course of bipolar disorder? Three longitudinal studies examined various cognitive styles as predictors of bipolar course without considering the role of life events. Two of these studies predicted change in symptom severity over time and found that cognitive styles predicted change in depressive but not manic symptoms (Johnson, Meyer, Winett, and Small, 2000; Johnson and Fingerhut, 2004). In a small sample of bipolar I patients, Johnson *et al.* (2000) reported that low self-esteem (assessed six months into the follow-up period) predicted average depressive, but not average manic, symptom severity over an eight 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, but not manic, symptoms over a two-year follow-up, controlling for baseline symptoms. Neither depression nor mania symptom change were predicted by dysfunctional attitudes. In contrast to Johnson *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.

Longitudinal studies: Cognitive vulnerability × stress predictors of bipolar course

Extensions of cognitive theories of unipolar depression to bipolar disorder have led to six efforts to examine the interaction between cognitive styles and life events as predictors of the course of bipolar disorder. Three 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, 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 six 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. The autonomy × negative achievement events interaction did not predict symptom onset or severity. Based on a four month follow-up, Francis-Raniere, Alloy and Abramson (2005) found that among bipolar individuals, controlling for initial depressive symptoms and the total number of negative events experienced, the self-criticism/performance evaluation × congruent negative events interaction predicted increases in depressive symptoms over the four months. Similarly, controlling for initial hypomanic symptoms and the total number of positive events experienced, the self-criticism/performance evaluation × congruent positive events interaction predicted increases in hypomanic symptoms over the follow-up. In contrast, sociotropy/dependency buffered against depressive symptoms following both congruent and non-congruent negative events. That is, bipolar individuals with high sociotropy/dependency experienced smaller increases in depressive symptoms over the four months following negative events than did those with low sociotropy/dependency. In a sample of remitted bipolar patients followed for one 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 obsessiveness and extraversion.

Two studies tested the cognitive vulnerability-stress hypotheses for attributional style and dysfunctional attitudes. In a sample of dysthymic, cyclothymic and hypomanic individuals, consistent with hopelessness theory, Alloy *et al.* (1999b) 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. 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. 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 and, within the bipolar group, manic symptoms. The fact that bipolar individuals with negative cognitive styles responded to the occurrence of negative life events with increases in manic symptoms is consistent with the 'manic defence' hypothesis.

It is interesting that, whereas Alloy *et al.* (1999b) found that positive life events combined with positive attributional styles to predict increases in hypomanic symptoms, Reilly-Harrington *et al.* (1999) found that it was negative events combined with negative cognitive styles that predicted manic symptoms. One possible explanation for this difference may be that Reilly-Harrington *et al.*'s bipolar sample was more severe, including primarily bipolar II and some bipolar I participants, whereas Alloy *et al.*'s sample included milder cyclothymic and hypomanic individuals. Given that bipolar I and II individuals have a course of disorder that includes major depressive episodes, they may be more responsive to negative life events. Alternatively, it may be that the particular types of negative events experienced by participants in the two studies is critical for whether such events would precipitate hypomanic/manic symptoms. Negative events that disrupt social rhythms or the sleep-wake cycle (Malkoff-Schwartz *et al.*, 1998; 2000) or those that act to activate the BAS (e.g., challenges that can be overcome; anger-inducing events) may be more likely to trigger hypomania/mania. Clearly, more work is needed to understand the conditions under which positive vs. negative events and positive vs. negative cognitive styles provide risk for hypomania/mania.

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. Compared to unipolar depressives, 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 depends somewhat on the current mood state of bipolar individuals and whether the measures of cognition are explicit or implicit, although the evidence for the manic defence 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 bipolars' cognitions are specifically BAS-relevant. Finally, 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 mania/hypomania. Further prospective studies are needed to test the cognitive vulnerability–stress hypothesis for bipolar disorder and whether it applies equally well to manic symptom course as it does to depression symptom course. Perhaps greater focus on BAS-relevant cognitive styles will increase predictive power for mania.

Conclusion

Do current or early environmental factors and cognitive styles contribute to the onset, course, or expression of bipolar disorders? Our conclusions must be tentative until such time as the methodological limitations characteristic of the psychosocial risk factors literature are more fully addressed. More prospective studies are needed with adequately sized samples of bipolar individuals, normal comparison groups, controls for initial mood state and symptoms, controls for genetic predisposition or use of genetically informative designs (e.g., prospective twin studies), and separate examination of depressive and manic/hypomanic episodes.

With these caveats in mind, there is fairly consistent evidence from prospective studies that recent life events and social support predict the likelihood of onsets and recurrences of bipolar mood episodes. Further exploration of social rhythm disrupting and BAS-relevant life events as predictors of bipolar mood episodes is clearly warranted. The literature on parenting and maltreatment histories as risk factors is only just developing, contains few prospective studies, and is less consistent overall. But there are promising findings suggesting that early environmental risk factors for bipolar disorder are worth further exploration. The cognitive style literature provides some consistency in suggesting that bipolar individuals' cognitive styles may be as negative as those of unipolar depressives, although more uniquely characterized by styles reflective

of high BAS sensitivity. Moreover, 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. 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 on the onset, course and expression of bipolar spectrum disorders.

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Author Queries:

AQ1: Alloy et al in press b – do you have publication details for this?

AQ2: Brown and Harris 1979 – 1978 in refs, which is correct?

AQ3: Davenport and Adland 1982 – not in refs

AQ4: Johnson et al 2000 – there are two entries in the refs for this author in this Year: I have made them a and b, please change the text citations as necessary

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AQ10: Alloy et al in press a, b – do you have publication details for this?

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AQ14: French et al 1999 – 1996 in refs, which is correct?

AQ15: Johnson et al 2000 – as above

AQ16: Johnson et al 2000 – as above

AQ17: Abramson et al 2005 in preparation – do you have publication details for this?

AQ18: Alloy et al in press a, b – do you have publication details for this?

AQ19: Francis-Raniere et al 2005 in preparation – do you have publication details for this?

AQ20: French et al 1996 – ‘Hypomania, anxiety and the emotional Stroop’ – should this say ‘Stroop test’?

AQ21: Grandin et al 2005 under review – do you have publication details for this?

AQ22: Neeren et al 2005 under review – do you have publication details for this?

AQ23: Nusslock et al 2005 – do you have publication details for this?

AQ24: Shen et al 2005 under review – do you have publication details for this?

AQ25: Urosevic et al 2005 under review – do you have publication details for this?