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Longitudinal Predictors of Bipolar Spectrum Disorders: A Behavioral Approach System (BAS) Perspective

Lauren B. Alloy
Temple University

Lyn Y. Abramson and Snezana Urosevic
University of Wisconsin-Madison

Rachel E. Bender and Clara A. Wagner
Temple University

Abstract

We review longitudinal predictors, primarily psychosocial, of the onset, course, and expression of bipolar spectrum disorders. We organize our review along a proximal – distal continuum, discussing the most proximal (i.e., prodromes) predictors of bipolar episodes first, then recent environmental (i.e., life events) predictors of bipolar symptoms and episodes next, followed by more distal psychological (i.e., cognitive styles) predictors, and ending with the most distal temperament (i.e., Behavioral Approach System sensitivity) predictors. We then present a theoretical model, the Behavioral Approach System (BAS) dysregulation model, for understanding and integrating the role of these predictors of bipolar spectrum disorders. Finally, we consider the implications of the reviewed longitudinal predictors for future research and psychosocial treatments of bipolar disorders.

Introduction

Bipolar disorder is at the same time puzzling and fascinating to researchers and laypeople alike because extreme contrasts in mood (hypomanic/manic euphoria and irritability vs. depressive sadness) and behavior (supercharged energy and excessive goal-striving vs. extreme lethargy and hopelessness) occur within the same individual. Indeed, bipolar disorder has been associated both with high achievement and artistic creativity on the one hand (e.g., Andreasen, 1987; Kutcher, Robertson, & Bird, 1998), and serious functional impairment including lower academic achievement, erratic work history, divorce, substance abuse, and increased suicide on the other hand (e.g., Angst, Stassen, Clayton, & Angst, 2002; Conway, Compton, Stinson, & Grant, 2006; Goodwin & Jamison, 1990; Lagace & Kutcher, 2005; Nusslock, Alloy, Abramson, Harmon-Jones, & Hogan, 2008c; Strakowski, DelBello, Fleck, & Arndt, 2000). Bipolar disorder has been ranked as the 6th leading cause of disability among both physical and psychiatric disorders worldwide (Murray & Lopez, 1996). Despite being quite prevalent (4.4% of a nationally representative U.S. sample were affected by a bipolar spectrum disorder; Merikangas et al., 2007) and sometimes disabling, bipolar disorder has been understudied relative to other mental health disorders (Hyman, 2000).

Bipolar disorders appear to form a continuum or spectrum of severity 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). Moreover, milder forms of bipolar disorder often progress to the more severe forms (e.g., Akiskal et al., 1977; 1979; Shen, Alloy, Abramson, & Sylvia, 2008), providing support for the spectrum concept of bipolar disorder. Thus, we consider the full range of bipolar spectrum disorders in this article.

Although bipolar disorder has a strong genetic predisposition (Merikangas et al., 2002; McGuffin et al., 2003) and important neurobiological underpinnings (Clark & Sahakian, 2006), there is a recognition that genetic and neurobiological factors cannot fully account for the timing, expression, and polarity of symptoms. Thus, in the past two decades, there has been increasing interest in the role of psychosocial processes in the onset, course, and treatment of bipolar spectrum disorders (see Alloy et al., 2005; 2006a,b,d,e,f for reviews). In this article, we review empirical research on longitudinal predictors, primarily psychosocial in nature, of the onset, course, and expression of bipolar spectrum disorders. We organize our review of these longitudinal predictors along a proximal – distal continuum (Abramson, Metalsky & Alloy, 1989), considering the most proximal or immediate (i.e., prodromes) predictors of bipolar episodes first, then recent environmental (i.e., life events) predictors of bipolar symptoms and episodes next, followed by more distal psychological (i.e., cognitive styles) predictors, and ending with the most distal temperamental (i.e., Behavioral Approach System [BAS] sensitivity) predictors. We then present a theoretical model, the Behavioral Approach System (BAS) dysregulation model (e.g., Depue, Krauss, & Spoont, 1987; Depue & Iacono, 1989; Johnson, 2005; Urosevic, Abramson, Harmon-Jones, & Alloy, 2008b), for understanding and integrating the role of these longitudinal predictors of bipolar spectrum disorders (see Figure 1). Finally, we end with a consideration of the implications of the reviewed longitudinal predictors for psychosocial treatments of bipolar disorders and directions for future research.

We note that there are other potential longitudinal predictors of bipolar disorder that we do not cover in this review, either because of lack of longitudinal data on these predictors or to other reviews providing extensive summary of them. For example, the role of supportive and non-supportive interpersonal relationships in bipolar disorder are covered in the Miklowitz et al. article in this special issue and in Alloy et al. (2005), but are not addressed in this review. Similarly, impaired executive functions (e.g., attention, working memory, cognitive perseveration) have been associated with bipolar disorder, have been found to be stable across mood states (see Alloy et al., 2006d; Walshaw & Alloy, 2008 for reviews), and may influence the longitudinal course of the disorder. These are not addressed in this review as neurocognitive functioning in bipolar disorder is covered in the Henin et al. article in this special issue. Early developmental experiences (e.g., parenting styles, family functioning, maltreatment history) have been associated with bipolar disorder, but these studies almost exclusively use retrospective or cross-sectional designs, rather than longitudinal ones, and, therefore, we do not review them here (but see Alloy et al., 2005; 2006a,b,d for reviews). The present review is unique in its focus on the temporal sequence of predictors of bipolar episodes, its integration of this set of predictors, and its provision of a theoretical model that may explain this temporal sequence. To this end, we highlight longitudinal and high-risk design studies and outline future directions that would further elucidate this temporal sequence.

General Methodological Issues and Challenges

In reviewing the literature on prodromes, recent environmental factors, and more distal cognitive and temperamental predictors of the onset, course, and expression of bipolar spectrum disorders, we chose to focus on the longitudinal and prospective studies. Longitudinal and prospective studies are better able to establish the potential predictor as independent of bipolar symptoms and to determine its temporal distance from the bipolar mood episodes or symptoms it is hypothesized to predict (Alloy et al., 2005). However, we also reviewed cross-sectional studies that compare bipolar individuals in a euthymic or remitted state to normal

controls on potential psychosocial predictors. We did so because such studies serve to demonstrate that the potential predictor under examination is independent of the symptoms of bipolar disorder, although they cannot establish that the psychosocial variable is a risk factor for bipolar disorder, rather than its consequence (Just, Abramson, & Alloy, 2001). Naturalistic longitudinal or prospective studies of psychosocial predictors of bipolar disorder are not without methodological limitations, such as potential third-variable explanations (in particular, the effects of genetic predisposition; Alloy et al., 2005). Few studies try to control for genetic vulnerability by controlling for family history of bipolar disorder (which, of course, also controls for family environment associated with having a relative with bipolar disorder). The very nature of bipolar spectrum disorders also presents a methodological challenge in that these disorders are recurrent with significant inter-episode symptoms. Studies wishing to establish predictor status for psychosocial variables need to ensure that potential predictors are independent of bipolar symptoms by controlling for mood and symptoms at the time the psychosocial variable under examination is assessed.

We also include in our review studies with high-risk research designs, in which the offspring of bipolar parents are compared to offspring of normal control parents (genetic high-risk design) or individuals at high vs. low risk for bipolar disorder based on a behavioral characteristic (behavioral high-risk design) are compared. We include even those high-risk study designs that are actually cross-sectional. Given that offspring of parents with bipolar disorder are at significantly higher risk for bipolar disorder themselves (Jones & Bentall, 2008), any psychosocial factors they experience at higher rates than the offspring of controls are potential predictors of bipolar disorder, even if not established longitudinally. Similarly, if the behavioral characteristic leading to designation of individuals as “high-risk” in behavioral high-risk studies has been shown to be a longitudinal predictor of bipolar mood episodes or symptoms in other studies, then by the same logic, any psychosocial factor these behavioral high-risk individuals show at higher levels than low-risk individuals can be considered a potential predictor of bipolar disorder. We turn now to our review of psychosocial predictors of bipolar spectrum disorders.

Immediate Predictors: Prodromes

A “prodrome,” from the Greek word *prodromos*, meaning forerunner of an event, is defined as the early symptoms and signs that precede the acute clinical phase of an illness (Fava & Kellner, 1991). Thus, by definition, prodromes precede episodes of disorder and are short-term longitudinal predictors of those episodes. However, there are methodological difficulties involved in studying prodromes of bipolar disorders. First, it is difficult to identify prodromes for mental disorders in general because there can be disagreement about what constitutes the acute phase of a psychological disorder. Second, although prodromes precede acute mood episodes in bipolar disorder by definition, most studies of bipolar disorder prodromes involve retrospective reporting of prodromes, increasing the possibility of questionable accuracy of the reports. Finally, a balance is needed between sensitivity and specificity in assessing prodromes. The assessment instruments need to be sensitive enough to identify small changes in signs and symptoms characteristic of the prodromal period, but not so sensitive as to identify normative mood and behavioral fluctuations as prodromal symptoms (Fava & Kellner, 1991). With these methodological caveats in mind, we review studies of the prodromes of bipolar hypomanic/manic and depressive episodes.

Research suggests that both bipolar individuals and their relatives are able to report prodromes reliably (Keitner et al., 1996; Lam, Wong, & Sham, 2001). Four studies examined the prodromes of manic episodes. Lam and Wong (1997; Wong & Lam, 1999) found that reduced sleep (58.3%) and increased goal directed activity (55.5%) were the most frequently reported prodromal symptoms of an impending manic episode among individuals with bipolar I disorder

(see Figure 1). In addition, compared to bipolar I individuals with poor coping skills, those with good coping strategies for prodromes decreased their goal directed activity (e.g., took extra time to rest, restrained themselves, engaged in calming activities) in order to deal with hypomania/mania. And, this behavioral deactivation coping strategy was successful. In a prospective study, Lam et al. (2001) found that over 18 months, bipolar I individuals who actively decreased their goal directed activity in response to manic prodromes were less likely to have a manic relapse (12.5%) than those who didn't cope in this manner (45.5%). Based on these findings, Lam et al. (2003) modified traditional cognitive therapy to target excessive goal striving and found that this led to significantly reduced relapse and hospitalization rates for bipolar episodes over one year.

Seven studies (Altman et al., 1992; Bauer et al., 2006; Keitner et al., 1996; Lam & Wong, 1997; Molnar, Feeney, & Fava, 1988; Smith & Tarrier, 1992; Wong & Lam, 1999) have examined prodromes of bipolar depressive episodes. Across these studies, decreased pleasure or anhedonia (45–82%), decreased motivation and goal directed activity (82%), decreased energy (71–86%), sad mood (21–86%), and decreased self-confidence (88%) were the most commonly reported prodromal symptoms for bipolar depression (see Figure 1). In addition, Bauer et al. (2006) found that hypersomnia predicted depressed mood the next day; but did not investigate prodromal symptoms other than sleep disturbances. Similar to their findings for coping strategies for manic prodromes, Lam et al. (2001) found that bipolar I individuals who coped with depressive prodromes by increasing their goal directed activity (e.g., keeping busy and getting organized, becoming more social) were less likely to experience depressive relapses (8.3%) over an 18-month follow-up than those who did not increase their behavioral activity (46.2%). Thus, studies of prodromes in bipolar disorder suggest that increased goal-striving and decreased pleasure/goal-directed activity are important immediate harbingers of impending manic and depressive episodes, respectively.

Recent Environmental Predictors: Life Events

Much evidence suggests that aspects of a person's current environment influence the onset, course, and expression of bipolar spectrum disorders (Alloy et al., 2005; 2006a,d). Specifically, we review the role of recently experienced life events as longitudinal predictors of bipolar disorder mood episodes and symptoms. As discussed in detail by Alloy et al. (2005), there are a variety of methodological limitations in many of the life events studies in bipolar disorder. The most problematic limitation of longitudinal studies of environmental predictors of bipolar disorder is failure to control for bipolar individuals' mood state at the time they report on life events and, thus, for mood-associated report biases. Second, some studies rely on self-report measures of life events, which can produce different interpretations across participants of what experiences count as an instance of a particular life event category. Such differences in self-reported life events could potentially introduce systematic differences in reporting as a function of symptoms or vulnerability factors. Thus, studies that employ structured interviewer assessments of life events should be given greater weight. Third, many studies don't examine life event predictors of hypomanic/manic and depressive episodes separately to determine if there are polarity-specific relationships. Finally, some studies define mood episode onset using markers that frequently do not correspond to the actual time of episode onset, such as admission to the hospital or start of treatment. With these caveats in mind, we review work on life events and bipolar disorder.

Recent life events as predictors of bipolar symptoms/episodes

Recent reviews of the association between life events and bipolar disorder (Alloy et al., 2005; 2006a,d,e; Johnson, 2005; Johnson & Kizer, 2002) indicate that individuals with bipolar spectrum disorders experience increased life events prior to first onsets and recurrences of mood episodes. Whereas negative life events precede the depressive episodes of bipolar

individuals, both negative and positive life events precede hypomanic/manic episodes. We briefly discuss the more methodologically limited retrospective studies first, followed by a more detailed review of the stronger longitudinal and prospective studies. We then examine whether specific types of life events are particularly likely to trigger hypomanic/manic and depressive mood episodes and whether bipolar mood episodes and symptoms, in turn, lead to the generation of specific types of life events as well.

Retrospective studies of life events have found that bipolar individuals' first and subsequent episodes are preceded by the occurrence of negative events, including events rated as independent of their behavior (see Alloy et al., 2005; 2006a,d,e for detailed reviews). The three retrospective studies of adolescent offspring of bipolar parents (Hillegers et al., 2004; Petti et al., 2004; Wals et al., 2005) found that affected offspring experienced significantly higher levels of negative life events than unaffected offspring. Wals et al. (2005) observed that it was only life events dependent on a participant's behavior that were related to increased risk of a mood episode (39% vs. 10%) in the offspring of the bipolar parents. This difference was no longer significant when prior anxious or depressive symptoms in the offspring were controlled. None of the retrospective studies investigated whether particular types of negative events are more relevant as triggers of manic vs. depressive episodes.

A smaller number of prospective, longitudinal studies of life events in bipolar disorder have been conducted to date. Three of these studies used questionnaire assessments of life events in bipolar samples. In an early study, Hall, Dunner, Zeller, and Fieve (1977) reported that bipolar patients with a hypomanic relapse had greater numbers of work-related stressors than did non-relapsers, although the overall number of events did not differ between bipolar patients who relapsed and non-relapsers. Christensen et al. (2003) assessed life events every 3 months for 3 years via questionnaire and found that bipolar women, but not men, had a greater number of events in the 3 months prior to a depressive episode compared to a control period. But, they failed to track relapses between the 3-month assessments. Lovejoy and Steuerwald (1997) tracked stressful events for 21 days in a small sample of cyclothymic, intermittent depressive, and control undergraduates and found that the cyclothymic group experienced more stressful events than the intermittent depressive group, which, in turn, had more events than the controls.

There are six prospective studies of bipolar samples that used interview assessments of life events. Most of these studies did not include a control group. Ellicott, Hammen, Gitlin, Brown, and Jamison (1990) observed that bipolar outpatients with high levels of stressful events had a 4.5-fold greater relapse rate over a 2-year follow-up period than those with lower stress. These findings could not be explained by differences in treatment adherence or medication levels. Similarly, Hammen and Gitlin (1997) found that bipolar patients who relapsed during a 2-year follow-up had more total stress and more severe events during the preceding 6 months than those who did not relapse. Hunt, Bruce-Jones, and Silverstone (1992) found that 19% of 52 relapses among bipolar patients were preceded by a severe event in the previous month compared to 5% of patients with a severe event at other times; manic and depressive relapses did not differ in the rate of prior severe events. Another study followed recovered bipolar patients, unipolar depressed patients, and normal controls for 1 year with interview assessments of life events and symptoms every 2 months (Pardoen et al., 1996). Pardoen et al. reported that bipolar patients with a hypomanic/manic relapse had more marital stressors prior to the relapse than other bipolar patients, although bipolar and unipolar patients who relapsed did not experience more life events overall in the 2 months prior to the relapse than patients who did not relapse. McPherson, Herbison, and Romans (1993) also did not find a significant difference in moderately severe, independent events in the month prior to relapse as compared to control periods among bipolar patients. However, these findings' interpretation is limited by a high attrition rate and the absence of a required well period prior to study entry. Finally, Johnson and Miller (1997) found that bipolar inpatients who reported a severe, independent event during

the index episode took 3 times longer to recover from that episode than those who did not have a severe independent event. This effect was not mediated by medication compliance.

In a “kindling” model, Post (1992) hypothesized that mood episodes become more autonomous with each recurrence such that life events are less likely to precipitate later than earlier episodes of mood disorder. Monroe and Harkness (2005) hypothesized two distinct, alternate models that might explain Post's kindling effect. The “stress sensitization” model postulates that major life stress is important in initiating first episodes, but decreases in unique importance for subsequent episodes. Although major life stress continues to be capable of triggering an episode, stress sensitization implies that lower and lower levels of stress may trigger episodes with subsequent recurrences, so that events that would not have been sufficient to trigger an initial episode acquire the power to precipitate recurrent episodes. Thus, the impact of major life events should increase with each episode as the individual becomes increasingly sensitized to stress. However, the relative frequency of major life events should decrease over time, as episode recurrences are increasingly triggered by more commonly occurring minor stressors. The impact and relative frequency of minor life events becomes stronger with episode recurrences. In contrast, the “stress autonomy” model postulates that major life stressors are important in initiating the first episode, but that later episodes are increasingly independent of psychosocial factors to the point where recurrences eventually take place fully independently of psychosocial triggers. This model postulates that the association between major life stress and initial mood episodes contributes to the development of another (potentially neurobiological) process that eventually takes over and fully accounts for episode recurrences. According to the stress autonomy model, the impact and frequency of both major and minor life events should decrease with each episode, as processes other than life stress increasingly trigger episode recurrences.

Although five retrospective studies (see Alloy et al., 2005; 2006a for reviews) claimed to support the kindling effect, these studies did not distinguish between the stress sensitization and stress autonomy models of kindling. They did not examine major and minor events separately or the impact vs. frequency of these events relative to the onset of bipolar mood episodes. Further, Hlastala et al. (2000) found that age, rather than the number of previous mood episodes, predicted higher levels of stressful events in pre-onset than control periods. Also, the one prospective study that examined the kindling effect in bipolar disorder (Hammen & Gitlin, 1997) found that contrary to the hypothesis, a greater proportion of bipolar patients with multiple lifetime mood episodes, rather than patients with few mood episodes, experienced a severe negative event prior to relapse. Thus, the evidence for a greater triggering effect of stressful events on earlier than later mood episodes in bipolar disorder is not strong at present. More longitudinal studies are needed that examine major and minor events, and the impact and frequency of these events, separately.

Role of specific types of life events

Recent evidence suggests that hypomanic/manic and depressive episodes of bipolar spectrum disorders may be triggered by specific types of life events, i.e., social rhythm disruption and BAS-relevant events¹ (see Figure 1). Four prospective, longitudinal studies found that life events involving goal striving or goal attainment that activate the BAS specifically trigger hypomanic/manic symptoms and episodes among bipolar individuals. In two separate studies,

¹BAS activation-relevant events have been defined to involve approach to (i.e., goal-striving) or attainment of rewards/goals, whereas BAS deactivation-relevant events involve failure to obtain or loss of pertinent rewards/goals and cessation of approach (see Urosevic et al., 2008b). Certain types of negative events (e.g., anger-inducing events, such as obstacles to goals and insults) have also been defined as BAS activation-relevant, because such events tend to elicit approach emotions and behaviors (Carver, 2004; Harmon-Jones & Allen, 1998; Harmon-Jones et al., 2002; 2003; Harmon-Jones & Sigelman, 2001). Behavioral Inhibition System (BIS)-relevant events, or events that involve threats without definitive failure or loss, have not been specifically studied in relation to bipolar disorders and are not reviewed here.

Johnson et al. (2000b;2008) reported that events involving attainment of goals predicted increases in manic, but not depressive, symptoms among bipolar I patients over prospective follow-up, whereas general positive life events did not. Similarly, Nusslock, Abramson, Harmon-Jones, Alloy, and Hogan (2007) found that students with bipolar spectrum disorders (bipolar II, cyclothymia) were significantly more likely to develop a new onset of hypomania, but not depression, following a goal striving life event (studying for and taking final exams) compared to other bipolar participants without this event (42% vs. 4%). Finally, Alloy et al. (2008c) found that BAS activation-relevant events (e.g., goal striving and attainment events) and BAS deactivation-relevant events (e.g., failures and losses) prospectively predicted increases in hypomanic and depressive symptoms, respectively, among bipolar spectrum participants over a 1-year follow-up. Some studies have also found that negative life events can trigger hypomanic or manic episodes (e.g., work-related stressors in Hall et al., 1977), but it is not known whether it is particular kinds of negative events that are relevant to predicting hypomania/ mania. Given that hypomania/mania sometimes presents with irritable mood, it is of interest that anger-inducing events that also are BAS-activating (e.g., goal obstacles, insults) have been associated with increased hypomanic symptoms (Carver, 2004;Harmon-Jones et al., 2002). However, prospective, longitudinal studies have not yet examined whether anger-provoking events predict onset of hypomanic/manic episodes in bipolar individuals.

Just as BAS-relevant events may trigger mood episodes in bipolar individuals, bipolar mood episodes and prodromes may also increase the likelihood of experiencing such events through processes of stress generation (Hammen, 1991; see Figure 1). In a prospective study, Urosevic et al. (2008a) found that bipolar spectrum individuals generated both BAS-activating and deactivating events at significantly greater rates than normal controls. Similarly, in another prospective study, Bender, Alloy, Abramson, Sylvia, and Urosevic (2008) reported that hypomanic symptoms predicted greater subsequent occurrence of BAS-related positive and negative achievement events in males, whereas depressive symptoms predicted greater occurrence of positive and negative interpersonal events in females.

Another specific type of life event that has been found to trigger bipolar mood episodes in five of seven studies is events that disrupt daily social rhythms (e.g., meal times, sleep-wake times, etc.). Such events are hypothesized to trigger bipolar mood episodes through their effects on destabilizing circadian rhythms (see Ehlers, Frank, & Kupfer, 1988; Grandin, Alloy, & Abramson, 2006; Healy & Williams, 1988 for reviews). Two studies (Ashman et al., 1999; Jones, Hare & Evershed, 2005) found that bipolar patients had lower social rhythm scores than age- and sex-matched controls, but the relationship between daily social rhythms and mood was not significant. However, the Ashman et al. study was underpowered due to small sample size. In two retrospective studies with structured interview assessments of life events in bipolar I patients, Malkoff-Schwartz et al. (1998, 2000) found that manic episodes were significantly more likely to be preceded by social rhythm disruption (SRD) events than depressive episodes. Kadri, Mouchtaq, Hakkou, and Moussaoui (2000) found that 45% of 20 Muslim bipolar patients relapsed during Ramadan (a fasting month with no meals and, thus, involving social rhythm disruption), and 71.4% of those relapses were manic episodes. In a prospective study of 206 bipolar spectrum individuals and 206 matched normal controls, Shen et al. (2008) reported that bipolar individuals had less regular social rhythms than the controls at Time 1. Moreover, lower Time 1 social rhythm regularity predicted a shorter time to onset of hypomanic/manic and depressive episodes during an average of 33 months of follow-up. Finally, in another 1-year prospective study of 101 bipolar spectrum participants and 100 matched normal controls, with structured interview assessments of life events and bipolar symptoms every 4 months, Sylvia et al. (in press) found that SRD events at each assessment predicted increased depressive symptoms at the next assessment. In addition, bipolar participants experienced more depressive symptoms after rather than before an SRD event occurred, and they were more likely to experience an SRD event prior to a depressive episode

than during a control period. Thus, SRD events appear to increase the likelihood of both hypomanic/manic and depressive episodes.

Psychological Predictors: Cognitive Styles

Given the success of cognitive models of unipolar depression (e.g., Abramson et al., 1989; Beck, 1987), the logic of these theories has been extended to bipolar disorders. Maladaptive cognitive styles (e.g., negative inferential styles, dysfunctional attitudes) found to increase vulnerability to unipolar depression also may contribute to the onset and course of bipolar mood episodes. Cognitive styles may be defined as people's typical patterns of perceiving, interpreting, and reacting to events in their lives. Negative inferential styles consist of the tendency to infer stable (enduring) and global (widespread) causes, negative consequences, and negative self-characteristics in response to a negative life event (Abramson et al., 1989). Dysfunctional attitudes involve maladaptive beliefs that one's self-worth depends on being perfect or on others' approval (Beck, 1987). In this section, we review the literature on cognitive styles alone and in combination with life events as predictors of the course of bipolar disorders, with specific emphasis on identifying cognitive styles uniquely characteristic of and important to bipolar disorders² (see Figure 1).

A central methodological issue in this literature is the need to establish cognitive predictors of bipolar mood episodes independent of potential mood state biases on the assessment of cognition. Studies have addressed this issue in several ways: by 1) controlling statistically for mood and symptoms at the time of cognitive measurement, 2) examining cognitions in remitted or euthymic bipolar individuals, 3) comparing bipolar individuals in depressive vs. manic episodes, or 4) conducting within-subject longitudinal studies of the same bipolar individuals in different mood states. Other study limitations in this literature include failure to take treatment status into account, absence of control groups, un-validated cognitive style measures, undiagnosed samples, and small sample sizes (see Alloy et al., 2005; 2006a,d,e,f).

Cognitive styles as predictors of bipolar symptoms/episodes

We briefly review the more limited cross-sectional studies of cognitive styles first, followed by a more detailed review of longitudinal studies. Cross-sectional studies of bipolar individuals in a depressed state find that their cognitive styles are as negative as those of unipolar depressed individuals and more negative than those of normal comparison groups. Also, in cross-sectional studies, current hypomania is associated with positive cognitive styles on explicit (direct) measures of cognition, but with negative cognitive styles on implicit (indirect) measures. Cross-sectional studies that compared bipolar individuals in different mood states found more positive cognitive styles in manic than depressed bipolar individuals with explicit measures, but more negative cognitive styles in hypomanic/manic than remitted bipolar individuals (see Alloy et al., 2005; 2006a,d,e,f for more detailed reviews).

Studies of bipolar individuals in a remitted or euthymic state, although also cross-sectional, are at least able to assess cognition independent of potential mood-state related biases. The results of fourteen such studies of cognitive styles have been mixed. Six studies (Hollon, Kendall & Lumry, 1986; Lex, Meyer, Marquart & Thau, in press; MacVane, Lange, Brown & Zayat, 1978; Pardoen, Bauwens, Tracy, Martin & Mendlewicz, 1993; Reilly-Harrington,

²As our review shows, the maladaptive cognitive styles characteristic of bipolar disorder, BAS-relevant cognitive styles, are a subset of those found to provide vulnerability to unipolar depression. But, they are unique in exhibiting a combination of characteristics involving high drive and incentive motivation (e.g., perfectionism, goal-striving, autonomy, and self-criticism), but not the approval-seeking and dependency typically observed in unipolar depression. Consequently, cognitive styles in unipolar depression and bipolar disorder can be assessed using the same instruments, and although there would be overlap in endorsed items, a combination of items endorsed consistent with BAS-relevant cognitive styles would emerge for bipolar disorders. For example, this has been shown in factor analysis of the Dysfunctional Attitudes Scale (Lam, Wright, & Smith, 2004).

Alloy, Fresco & Whitehouse, 1999; Tracy, Bauwens, Martin, Pardoën & Mendlewicz, 1992) using primarily explicit measures obtained little evidence of negative cognitive styles in the euthymic state. However, Lex et al. (2008) did find a trend ($p = .06$) for remitted bipolar individuals to have more negative dysfunctional attitudes than normal controls. In contrast, eight other studies (Alloy et al., 1999; 2008b; Goldberg, Gerstein, Wenzel, Welker & Beck, 2008; Knowles et al., 2007; Lam, Wright & Smith, 2004; Rosenfarb, Becker, Khan & Mintz, 1998; Scott, Stanton, Garland & Ferrier, 2000; Winters & Neale, 1985), also using mostly explicit measures, did find negative cognitive styles among euthymic bipolar individuals.

Cross-sectional studies of individuals at genetic or behavioral risk for bipolar disorder have also obtained evidence for maladaptive cognitive and coping styles. In a genetic high-risk study, adolescent offspring of bipolar parents exhibited greater ruminative response styles for negative affect, but not greater dysfunctional attitudes, than offspring of control parents (Jones, Tai, Evershed, Knowles & Bentall, 2006). In addition, three studies of individuals at behavioral high risk for bipolar disorder found that high-risk participants exhibited both a greater ruminative style in response to negative and/or positive affect and more dysfunctional attitudes than low-risk participants (Carver & Johnson, in press; Knowles, Tai, Christensen & Bentall, 2005; Thompson & Bentall, 2002).

Two types of longitudinal studies have examined cognitive styles in bipolar individuals. The first type investigated the stability of cognitive styles across different mood states within the same bipolar individuals over time. Two studies found that inferential styles and dysfunctional attitudes were stable and relatively negative over time and across untreated bipolar spectrum participants' different mood swings (Alloy et al., 1999; Gerstein, Alloy & Abramson, 2008). In contrast, Ashworth et al. (1985) found that explicit self-esteem reverted to normal levels when previously manic or depressed bipolar patients recovered. Thus, there is some evidence of stable cognitive styles in bipolar individuals across mood states, but further longitudinal studies of this kind are needed. In particular, it is important for such studies to control for treatment status, because treatment could remediate bipolar individuals' cognitive styles.

The second kind of longitudinal study examined general cognitive styles as predictors of the course of bipolar disorder. Scott and Pope (2003) found that low self-esteem was the most robust predictor of relapse at 12-month follow-up among hypomanic bipolar patients. Two other studies found that negative automatic thoughts (Johnson & Fingerhut, 2004) and low self-esteem (Johnson, Meyer, Winett, & Small, 2000a) predicted depressive, but not manic, symptoms over prospective follow-up.

Bipolar disorder-specific cognitive styles

Recent evidence suggests that particular types of cognitive styles may be especially relevant to bipolar disorder and prediction of bipolar mood episodes and symptoms. Five remitted design studies found that euthymic bipolar individuals exhibited a unique profile of maladaptive cognitive styles characterized by perfectionism, autonomy, self-criticism, and goal-striving and not by maladaptive dependency, approval-seeking, or attachment attitudes typically observed among unipolar depressed individuals (Alloy et al., 2008b; Goldberg et al., 2008; Lam et al., 2004; Rosenfarb et al., 1998; Scott et al., 2000). The bipolar individuals' types of maladaptive cognitive styles are consistent with the high drive and incentive motivation associated with high BAS sensitivity² (see Figure 1). In addition, seven behavioral high-risk studies found that individuals at increased risk for bipolar disorder exhibited specific cognitive styles with high BAS-relevance. In particular, individuals prone to hypomania/mania exhibited overly ambitious goal-striving and goal-setting, even controlling for current hypomanic and depressive symptoms (Carver & Johnson, in press; Gruber & Johnson, in press; Johnson & Carver, 2006; Johnson, Ruggero & Carver, 2005; Meyer & Krumm-Merabet, 2003) and more positive appraisals of major personal goals (Meyer, Beevers & Johnson, 2004). They also

showed greater cognitive reactivity and positive generalization in response to success experiences (Carver & Johnson, in press; Eisner, Johnson & Carver, 2008, Study 2; Johnson et al., 2005).

Finally, two longitudinal studies specifically examined whether BAS-relevant cognitive styles predicted bipolar mood symptoms or episodes over time. Lozano and Johnson (2001) reported that the BAS-relevant style of high achievement-striving predicted increases in manic symptoms over 6 months in a bipolar I sample. Similarly, Alloy et al. (2008b) reported that BAS-relevant cognitive styles of self-criticism and autonomy, but not cognitive styles involving approval-seeking, dependency and attachment, predicted prospective onsets of hypomanic/manic and depressive episodes among individuals with bipolar spectrum disorders.

Cognitive styles X life event interactions as predictors of bipolar symptoms/episodes

Six longitudinal studies tested the cognitive vulnerability-stress hypothesis for prediction of bipolar mood symptoms and episodes. Swendsen, Hammen, Heller and Gitlin (1995) found that stressful events interacted with obsessiveness and extraversion to predict relapse among remitted bipolar patients. Alloy et al. (1999) reported that among individuals with cyclothymic and hypomanic disorders, Time 1 (euthymic state) negative attributional style for negative events interacted with negative events to predict subsequent increases in depressive symptoms, and Time 1 positive attributional style for positive events combined with positive events to predict later increases in hypomanic symptoms. The interaction of dysfunctional attitudes and life events did not predict subsequent symptoms. In a unipolar and bipolar I and II sample, Reilly-Harrington et al. (1999) found that controlling for initial symptoms, Time 1 negative attributional styles, dysfunctional attitudes, and negative information processing about oneself each interacted significantly with negative life events to predict subsequent increases in depressive symptoms and, among the bipolar participants, manic symptoms as well. Note that in the Alloy et al. (1999) study, hypomanic symptoms were predicted by positive life events combined with positive attributional styles, whereas in Reilly-Harrington et al. (1999), manic symptoms were predicted by negative events combined with negative cognitive styles. Given that bipolar I and II individuals (in Reilly-Harrington et al.'s study) have a course of disorder that includes major depressive episodes, they may be more responsive to negative life events than the bipolar individuals without major depression in the Alloy et al. study. It's also possible that the particular types of negative events (e.g., BAS activation-relevant anger-inducing events) experienced by participants in the two studies may determine whether such events would precipitate hypomanic/manic symptoms.

Longitudinal studies have also investigated sociotropic (i.e., dependent) and autonomous cognitive styles in interaction with congruent life events (interpersonal events for sociotropic/dependent individuals and achievement events for autonomous individuals) as predictors of bipolar symptoms. Hammen et al. (1989; 1992) found that sociotropy and negative interpersonal events interacted to predict symptom severity, but not symptom onset, in bipolar individuals (although only a trend in Hammen et al., 1989). Autonomy combined with achievement events did not predict symptom severity. However, Hammen et al. (1989; 1992) did not examine prediction of depressive and hypomanic/manic symptoms separately. Finally, controlling for initial symptoms and the total events experienced, Francis-Raniere, Alloy and Abramson (2006) reported that a BAS-relevant self-critical, perfectionistic cognitive style interacted with style-congruent negative or positive events to predict prospective increases in depressive and hypomanic symptoms, respectively, among bipolar spectrum individuals.

Psychobiological Predictors: Temperament/Personality

Given that bipolar disorder has a strong genetic predisposition (Merikangas et al., 2002; McGuffin et al., 2003), and temperament is considered to be genetically-influenced,

researchers have become interested in temperamental factors involved in bipolar spectrum disorders. One temperamental characteristic that has figured prominently in theorizing and research about bipolar disorder is BAS sensitivity (approach motivation and reward responsiveness). We review its role as a predictor of bipolar mood episodes and symptoms here. Only some of the studies in this area control for mood symptoms at the time of assessment of BAS sensitivity, and none control for genetic predisposition itself.

BAS sensitivity as a predictor of bipolar symptoms/episodes (see Figure 1)

Investigators suggest that two fundamental psychobiological systems are critical in regulating behavior (Davidson, 1999; Gray, 1981; 1982), the BAS and the Behavioral Inhibition System (BIS). The BAS regulates approach behavior to attain rewards and goals, whereas the BIS regulates inhibition of behavior in response to threat and punishment.³ We focus on the BAS because theory and recent research underscore its importance in bipolar disorder. Activation of the BAS by signals of reward causes a person to increase approach motivation and movement toward attainment of goals, as well as cognitive processes (e.g., hope, self-efficacy, planning) aimed at promoting goal attainment. BAS activation is also hypothesized to be associated with happiness and elation (Depue & Iacono, 1989; Gray, 1994). Recent work also documents a link between anger and BAS activation (Carver, 2004; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001), especially when people have a high expectancy of success for rectifying the anger-provoking situation (Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003). BAS sensitivity and state activation levels have been assessed in three main ways: 1) with self-report questionnaires (e.g., BIS/BAS Scales [Carver & White, 1994] and Sensitivity to Punishment Sensitivity to Reward Questionnaire [SPSRQ; Torrubia, Avila, Molto & Caseras, 2001]); 2) with behavioral tasks involving rewards (e.g., Card Arranging Reward Responsivity Objective Test [CARROT; Powell, Al-Adawi, Morgan & Greenwood, 1996]); and 3) with relative left vs. right prefrontal cortical activation as measured with EEG, both in the resting state and in response to rewards. Greater relative left frontal cortical activation on EEG has been found to reflect higher BAS sensitivity and activation (e.g., Coan & Allen, 2004; Davidson, Jackson & Kalin, 2000; Harmon-Jones & Allen, 1997; Sobotka, Davidson & Senulis, 1992; Sutton & Davidson, 1997).⁴

Cross-sectional studies have found that even controlling for bipolar mood symptoms, individuals with bipolar spectrum disorders exhibit higher levels of self-reported BAS sensitivity, as well as greater reward responsiveness on behavioral tasks and greater relative left frontal cortical activity on EEG than relevant controls. Studies of bipolar individuals in a remitted or euthymic state, although also cross-sectional, are of interest because they assess BAS sensitivity or responsiveness independent of potential mood-state related biases. Salavert et al. (2007) found that controlling for concurrent depressive and hypomanic/manic symptoms, euthymic bipolar I patients exhibited higher BAS sensitivity on the SPSRQ than normal

³Whereas BIS activation involves inhibition of behaviors in order to avoid threats and punishments, BAS deactivation involves an extreme reduction in goal-directed activity following inability to obtain a reward (as signaled by unequivocal failure or loss). Someone with high BIS activation would be quicker than most to withdraw/inhibit behavior in response to the threat of a negative consequence. BIS activation has been associated with both anxiety and depression. Instead, someone with BAS deactivation would show a reduction in approach behavior following an actual, irrevocable failure or loss. Thus, BAS deactivation is associated only with depression (Kasch, Rottenberg, Arnou, & Gotlib, 2002; McFarland, Shankman, Tenke, Bruder, & Klein, 2006; Pinto-Meza et al., 2006). Typically, the correlation between BAS and BIS sensitivities on self-report questionnaires is quite low (e.g., Alloy et al., 2008a; Carver & White, 1994).

⁴Although self-report, behavioral task, and EEG measures of BAS sensitivity and activation have been found to correlate with each other, in some studies their convergence is relatively weak. One possible reason for this is that BAS sensitivity may include three components, supported by factor analyses of the BIS/BAS scales (e.g., Carver & White, 1994): BAS Drive (persistence in pursuit of reward), BAS Fun-seeking (willingness to approach novel and rewarding stimuli), and BAS Reward Responsiveness (positive reactivity to rewards). Some of these BAS components may relate to behavioral and EEG measures of BAS activation more strongly than others; this has not been investigated well to date. Similarly, across studies, there are inconsistent findings with respect to which of these BAS subscales are related more strongly to bipolar disorder. Thus, although it is important to continue to explore potential differential effects of these BAS components, at present, there is insufficient evidence to determine which are more implicated in BAS dysregulation and bipolar disorder.

controls. Hayden et al. (2008) reported that euthymic bipolar I patients exhibited higher reward responsivity on the CARROT card sorting task than normal controls, but they didn't exhibit higher self-reported BAS sensitivity on the BIS/BAS Scales or greater relative left frontal cortical activation on EEG in the resting state. In a 28-day daily diary study, Wright, Lam, and Brown (2008) found that euthymic bipolar I individuals and controls did not differ on recovery of behavioral activation to baseline following rewarding or frustrating life events. However, time taken for behavioral activation to recover from rewarding events increased with increasing numbers of previous manic episodes, and time taken to recover following frustrating events increased with increasing numbers of both previous manic and depressive episodes. Moreover, both BAS sensitivity and BAS-related dysfunctional attitudes remain stable across fluctuations in clinical symptoms and despite positive mood induction (Meyer, Johnson & Winters, 2001; Urošević et al., 2008c; Wright, Lam & Newsom-Davis, 2005).

With one exception, individuals at genetic or behavioral risk for bipolar disorder have shown higher BAS sensitivity than controls. Although Jones et al. (2006) did not observe higher self-reported BAS sensitivity in offspring of bipolar parents than offspring of control parents, Chang, Blasey, Ketter and Steiner (2003) did find that children of bipolar parents displayed a greater tendency to approach novel, potentially rewarding situations on a temperament scale than controls. In addition, Nurnberger et al. (1988) reported greater self-reported sensation-seeking in offspring of bipolar parents than controls. In behavioral high-risk studies, high BAS sensitivity has also been associated with risk for bipolar disorder. Individuals prone to hypomanic symptoms exhibited higher self-reported BAS sensitivity than controls (Carver & Johnson, in press; Meyer, Johnson & Carver, 1999) and greater relative left frontal cortical activation on EEG in response to a BAS-activating event (an anger-provocation scenario; Harmon-Jones et al., 2002). Finally, Alloy et al. (2006c) selected participants with high vs. moderate levels of BAS sensitivity on both the BIS/BAS Scales and the SPSRQ and administered structured diagnostic interviews. They found that individuals with high BAS sensitivity were 6 times more likely to meet diagnostic criteria for a lifetime bipolar spectrum disorder than those with moderate BAS sensitivity.

Four longitudinal studies have examined BAS sensitivity as a predictor of subsequent bipolar symptoms or episodes. Meyer et al. (2001) found that higher self-reported BAS sensitivity at recovery predicted greater manic symptoms over time in bipolar I patients. Similarly, controlling for initial depressive and manic symptoms, Salavert et al. (2007) found that over an 18-month follow-up, bipolar I patients who relapsed with a hypomanic/manic episode had higher, and those who relapsed with a depressive episode had lower, BAS sensitivity at Time 1 than patients who remained asymptomatic. Also controlling for initial depressive and hypomanic symptoms, Alloy et al. (2008a) found that higher Time 1 self-reported BAS sensitivity predicted a shorter time to onset of hypomanic/manic episodes among individuals with bipolar spectrum disorders over a 3-year follow-up. In addition, higher BAS Reward Responsiveness predicted shorter time to onset of major depressive episodes (with marginal significance). Finally, among bipolar spectrum individuals, Nusslock et al. (2008b) found that greater relative left frontal cortical activity on EEG in the resting state predicted shorter time to onset of hypomanic/manic episodes, whereas decreased relative left frontal cortical activation in response to reward incentives predicted an increase in the number of major depressive episodes over follow-up.

A Behavioral Approach System (BAS) Dysregulation Perspective on Longitudinal Predictors

How can we understand the evidence regarding the prodromal, environmental, cognitive, and temperamental predictors of the course and expression of bipolar disorders? A BAS dysregulation perspective on bipolar disorders may allow us to integrate these predictors of

bipolar disorder into a more comprehensive, explanatory model (see Figure 1). According to the BAS dysregulation model of bipolar disorder (Depue & Iacono, 1989; Depue et al., 1987; Johnson, 2005; Urosevic et al., 2008b), individuals vulnerable to bipolar spectrum disorders exhibit an overly sensitive BAS that is hyper-reactive to relevant cues and, thus, becomes dysregulated easily. Such BAS hypersensitivity may lead individuals to experience great variability in their state levels of BAS activation over time and across situations in response to BAS activating and deactivating stimuli. Thus, a hyper-responsive BAS can lead to excessive BAS activity in response to BAS activation-relevant events involving goal striving and attainment, reward incentive, and anger-evocation. In vulnerable individuals, this excessive BAS activation is hypothesized to lead to hypomanic/manic symptoms such as elation, excessive goal seeking behavior, decreased need for sleep, irritability, distractibility, excessive self-confidence and optimism (Figure 1, top). In contrast, in response to BAS deactivation-relevant events involving definite failure and non-attainment of goals, excessive BAS deactivation or shutdown of behavioral approach should occur, leading to depressive symptoms of sadness, low energy, anhedonia, motor retardation, hopelessness, and low self-confidence (Figure 1, bottom). In essence, individuals vulnerable to bipolar disorder are unable to effectively regulate their emotions and behavior because their proneness to BAS dysregulation renders them excessively responsive to BAS-relevant events. Indeed, even quite minor BAS-relevant events may be sufficient to trigger bipolar mood episodes in individuals with highly sensitive BAS temperaments⁵ (i.e., stress sensitization as a function of higher vulnerability). Such BAS hypersensitivity and vulnerability to dysregulation may be an endophenotype that mediates the effects of the genetic predisposition to bipolar disorder.

This BAS dysregulation model provides an integrative biopsychosocial perspective for understanding the effects of the predictors of bipolar disorder reviewed here (see Figure 1). As reviewed in the section on prodromes, and consistent with the BAS dysregulation model, the most frequent prodromal signs of impending hypomanic/manic episodes are reduced sleep and increased goal-directed activity, both indicators of increased approach motivation mediated by BAS activation. Similarly, decreased motivation, goal-directed activity, and pleasure, indicators of BAS deactivation, are frequent prodromal signs of impending depressive episodes. Moreover, strategies for coping with prodromes involving decreasing goal-directed activity when it starts to increase, or increasing such activity when it begins to decrease, were found to reduce the likelihood of manic and depressive relapses, respectively.

Our review of the life events literature indicated that negative events precipitate bipolar depressive episodes and both negative and positive events precipitate hypomanic/manic episodes. However, consistent with the BAS model, studies that investigated specific types of events that trigger bipolar mood episodes found that goal-striving and goal attainment events that activate the BAS precipitate hypomania/mania, whereas failures and losses that deactivate the BAS precipitate bipolar depression. Some evidence suggests that negative events involving anger-provocation also trigger hypomania/mania, consistent with the BAS model. Thus, the BAS model provides a plausible explanation for the seemingly inconsistent findings of negative life events sometimes predicting onset of hypomania/mania as well as depression, because certain types of negatively-valenced events (e.g., goal obstructions, insults) should activate the BAS rather than deactivate it. Finally, our review indicated that events that disrupt daily social rhythms (SRD events) also precipitate both hypomanic/manic and depressive episodes in bipolar individuals. It's possible that SRD events exert their effect on bipolar symptoms through

⁵From a BAS dysregulation model perspective, at least some cases of bipolar mood episodes that appear to arise “out of the blue” may actually have environmental triggers – relatively minor BAS-relevant events that are over-reacted to by individuals with a hypersensitive BAS. The stronger an individual's BAS sensitivity, the less environmental input would be required to trigger a bipolar mood episode. But, it is also possible that some subtypes of bipolar disorder represent a more pure expression of the high BAS temperament (e.g., hyperthymic personality).

their BAS-relevant characteristics. For example, events involving goal-striving may trigger a dysregulated BAS response with social rhythm disruption as one component of this dysregulated response (e.g., a bipolar individual starts a new job, becomes over-involved in projects, and as a result, loses hours of sleep). Future studies assessing both the BAS-relevance of events (e.g., the extent of goal-striving) and the events' SRD characteristics are needed to determine whether SRD is related to and mediates dysregulated BAS response in predicting bipolar mood symptoms.

The evidence regarding cognitive styles and bipolar disorder, although mixed, indicated that individuals with bipolar spectrum disorders frequently exhibit maladaptive cognitive styles, even in the euthymic state, as negative as those seen in unipolar depressed persons. Moreover, these cognitive styles predict subsequent bipolar mood symptoms and episodes alone or in interaction with life events. However, a growing number of studies suggest that it is cognitive styles involving perfectionism, excessive goal-striving, self-criticism, and autonomy (i.e., styles that are relevant to the high drive and incentive motivation associated with high BAS sensitivity) that are uniquely characteristic of risk for bipolar disorder and predictive of bipolar mood symptoms and episodes. Finally, research on BAS sensitivity itself indicated that individuals with or at increased risk for bipolar disorders exhibit high BAS sensitivity assessed via self-report, behavioral tasks, and neurophysiology, and that high BAS sensitivity predicts bipolar mood symptoms and episodes longitudinally. Consequently, the BAS dysregulation theory of bipolar disorders, a model that integrates psychosocial and neurobiological (e.g., reward system circuitry) underpinnings of bipolar disorder, may provide a useful conceptual framework for understanding a diverse set of empirical findings regarding longitudinal predictors of bipolar disorder.

The BAS model suggests novel predictions about the course of bipolar spectrum disorders (see Urosevic et al., 2008b). According to a BAS dysregulation perspective, both a hypersensitive BAS temperament and BAS-relevant life events should interact to predict the course and prognosis of bipolar disorders, yet no studies have examined this prediction to date. The frequency of BAS activation-relevant and deactivation-relevant events also should predict the relative predominance of hypomania/mania vs. depression, respectively, in the course of bipolar disorder. Moreover, an individual's level of BAS activation prior to the occurrence of a BAS-relevant life event, along with their general degree of BAS sensitivity, should influence the magnitude of their dysregulated response to that event and thus, the severity and duration of bipolar symptoms.

Clinical Implications

The BAS dysregulation model also suggests new directions for improving treatment for bipolar disorders (see Nusslock, Abramson, Harmon-Jones, & Alloy, 2008a). Given the evidence that environmental factors (e.g., life events) play an important role in the onset and course of bipolar disorders and the limitations of pharmacotherapy alone in treating these conditions (Prien & Rush, 1996), promising psychosocial therapies have been developed as adjuncts to medication (e.g., cognitive behavioral therapy [CBT] and Interpersonal Social Rhythm Therapy [IPSRT]) in an attempt to reduce the likelihood of relapse of mood episodes. The evidence reviewed here on longitudinal predictors of bipolar disorder and the BAS dysregulation perspective for integrating these predictors have implications for increasing the effectiveness of these psychosocial interventions (see Nusslock et al., 2008a for a detailed explication of the therapeutic implications of the BAS model).

As an adjunctive intervention for bipolar disorder, CBT teaches bipolar individuals to recognize prodromes for depressive and manic episodes and then modify their cognitions and behavior to prevent the onset of full-blown episodes (Lam et al., 2003; Newman et al., 2002).

Thus, CBT focuses on recognizing proximal predictors of impending episodes (i.e., prodromes) and modifying more distal cognitive styles to prevent mood episodes. Overall, the small outcome literature on CBT for bipolar disorder suggests that it has a positive prophylactic effect (see Alloy et al., 2005 and Nusslock et al., 2008a for reviews). Based on the evidence that the cognitive styles of bipolar individuals are characterized by perfectionism and extreme goal-striving tendencies and the prodromes of manic and depressive episodes involve increased and decreased goal-directed activity, respectively, CBT based on a BAS dysregulation perspective should aid bipolar clients in recognizing the relationship between ambitious goal-setting and onset of manic episodes and giving up on goals and onset of depressive episodes. In addition, CBT cognitive techniques in which surges of confidence and ambitious goal-striving on the one hand, or reduced efficacy and decreased goal-striving on the other hand, are challenged and reframed as early harbingers of impending episodes should be beneficial. Further, behavioral strategies designed to reduce goal-directed activity when the client recognizes a surge in success expectancies and goal-striving, and to increase goal-oriented behaviors when the client notes a decrease in confidence and goal-striving, should reduce the likelihood of manic and depressive episodes, respectively, as found by Lam et al. (2001; 2003).

Based on the social/circadian rhythms theory of bipolar disorder and supporting evidence, IPSRT was also designed as an adjunct to pharmacotherapy for bipolar disorder (Frank, Swartz & Kupfer, 2000). IPSRT encourages bipolar patients to notice the association between life events and their moods, the importance of maintaining regular daily schedules, and to manage interpersonal stressors that may precipitate SRD, and thus, mood episodes. Thus, IPSRT focuses almost entirely on recent environmental triggers of mood episodes. The small outcome literature on IPSRT finds that it may also have promise for improving the course of bipolar disorder (e.g., Frank et al., 2005; see Alloy et al., 2005 and Nusslock et al., 2008a for reviews). To date, IPSRT has focused on the disruptive effects that interpersonal events can have on social and circadian rhythms. However, the BAS model and supportive evidence reviewed here suggest that BAS-relevant events in the achievement domain are also likely to precipitate social and circadian rhythm disruption in bipolar individuals, because work, school, and goal-striving are also important social zeitgebers ("time-givers"). For example, when working on a project or faced with a deadline, many people lose sleep, miss meals, and significantly alter their usual daily routine. BAS deactivation events may also precipitate SRD. Therapeutic strategies designed to address the effect of interpersonal events on SRD could be applied to BAS-relevant events as well. For example, the therapist and client could identify the extent to which various BAS-relevant events are likely to induce social and circadian rhythm disruption and develop strategies for regulating rhythms when these events occur. Thus, added therapeutic benefit might result from an expansion of IPSRT's focus to strategies for coping with the effects of both independent and self-generated BAS-relevant events on social and circadian rhythms (see Nusslock et al., 2008a for details).

No psychosocial interventions have been developed yet designed to modify or decrease the likelihood of activation of a hypersensitive BAS temperament, the proposed distal vulnerability for bipolar spectrum disorders. Recent evidence suggests that it is possible to manipulate neurophysiological indicators of BAS activation in the laboratory (Harmon-Jones, 2006; Peterson, Schackman, & Harmon-Jones, 2008). Thus, future work on the therapeutic implications of the BAS dysregulation model might also address the development of strategies for more directly reducing BAS hyper-responsivity.

Conclusions

More prospective, longitudinal studies are needed with adequately sized samples, controls for initial mood state and symptoms, separate examination of depressive and hypomanic/manic episodes, standardized and well-validated measures of psychosocial and neurobiological

variables, and controls for genetic predisposition or genetically informative designs (e.g., longitudinal twin studies) to further our understanding of important predictors of bipolar disorders. Despite these methodological caveats and the long-standing assumption that bipolar disorders are primarily caused by genetic predisposition and resulting neurobiological dysfunction, the evidence reviewed here indicates that psychosocial factors also serve as distal and proximal predictors of the onset, course, and expression of bipolar disorders. Specifically, a temperament involving high BAS sensitivity and related cognitive styles characterized by perfectionism, self-criticism, autonomy, and excessive goal-striving predict risk for bipolar symptoms and episodes. In addition, life events that activate and deactivate the BAS, as well as events likely to disrupt daily social rhythms, predict subsequent mood episodes, and prodromes involving changes in goal-directed activity, sleep, and self-confidence serve as immediate harbingers of the impending mood episodes. Future studies are needed to further delineate the temporal relationships of these psychosocial predictors and bipolar symptoms (e.g., as seen in Figure 1). Finally, a BAS dysregulation perspective on bipolar disorder may provide a comprehensive and integrative psychobiological model for understanding the role of these empirically identified predictors of bipolar course and expression. As Akiskal (1986, p. 671) speculated, "...what is transmitted are these affectively dysregulated temperaments and that the progression to full-blown bipolar illness is due to environment."

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