Social Support and the Course of Bipolar Disorder

Sheri L. Johnson
Department of Psychology University of Miami
Carol A. Winett
Department of Psychology University of Miami
Björn Meyer
Department of Psychology University of Miami
William J. Greenhouse
Department of Psychology University of Miami
Ivan Miller
Department of Psychiatry and Behavior Brown University

ABSTRACT

The current study prospectively examined the impact of social support on symptom severity and recovery from episodes in bipolar disorder, both as a direct influence and as a buffer of life events. Fifty-nine individuals with Bipolar I disorder were followed longitudinally with monthly symptom severity interviews. Social support was measured by the Interpersonal Support Evaluation List and the Interview Schedule for Social Interaction, and life events were assessed using the Life Events and Difficulties Schedule. Individuals with low social support took longer to recover from episodes and were more symptomatic across a 6-month follow-up. Results suggest a polarity-specific effect, in that social support influences depression but not mania. Discussion focuses on theoretical implications of a series of polarity-specific findings within the field.

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Correspondence may be addressed to Sheri L. Johnson, Department of Psychology, University of Miami, Coral Gables, Florida, 33124—0721.

Electronic mail may be sent to sjohnson@miami.edu

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Bipolar disorder is severe, with devastating consequences for affected individuals and for society. Of individuals hospitalized for mania, 30% remain unemployed for 6 months (Dion, Tohen, Anthony, & Waternaux, 1988) and 23% for 1 year (Harrow, Goldberg, Grossman, & Meltzer, 1990). Across physical and psychiatric disorders, bipolar disorder is ranked as the sixth leading cause of disability (Murray & Lopez, 1996). In 1991, costs for bipolar disorder among adult Americans totaled $45 billion (Wyatt & Henter, 1995). The personal costs of this disorder are emphasized by the alarming
finding that as many as 19% of bipolar individuals die from suicide (Isometsa, 1993).

Beginning with Cade's discovery of the mood-stabilizing effects of lithium in 1949, remarkable advances have been made in pharmacotherapy of bipolar disorder. Double-blind randomized trials demonstrate the efficacy of lithium in reducing the severity and frequency of episodes (Sachs, Lafer, Truman, Noeth, & Thibault, 1994). Newer medications, such as valproate and carbamazepine, have shown promise as well (Ketter et al., 1998; McElroy, Keck, Pope, & Hudson, 1992). The efficacy of pharmacotherapy has contributed to a biological zeitgeist, which has been bolstered by twin studies demonstrating an extremely high concordance for bipolar disorder (Bertelsen, Harvald, & Hauge, 1977). For many years, it was assumed that bipolar disorder was understood and controlled using these biological approaches.

Despite the gains from pharmacological approaches, many individuals still experience poor outcomes. Even with blood serum levels of lithium between 0.8 and 1.0, 32% of bipolar individuals relapsed over a 3-year period (Keller et al., 1992). Treatments are less effective for bipolar depression than mania (Hlastala et al., 1997). Within naturalistic studies, noncompliance contributes to even higher relapse rates: In one study, only 4% of patients who were prescribed lithium monotherapy sustained remission for 1 year (Sachs et al., 1994). Beyond the poor outcomes, biological models are limited in predicting course of disorder. Several factors, such as seasonality, previous history, and family history predict course within some studies; however, these effects tend to explain a relatively small proportion of the variance in outcome (cf. Coryell, Endicott, & Keller, 1992; Silverstone, Romans, Hunt, & McPherson, 1995). In summary, biological indexes do not fully account for the timing and severity of episodes.

Noting that these gaps exist, researchers have recognized the need for multifactorial models of treatment and etiology (Prien & Potter, 1990). Recent research has demonstrated the importance of psychosocial treatments as adjuncts to medication (Miklowitz, 1996). In tandem with shifts in treatment approaches, psychosocial predictors of the course of the disorder have been examined more carefully. Life events (Hammen, Ellicott, & Gitlin, 1992; Johnson & Roberts, 1995) and personality (Swendsen, Hammen, Heller, & Gitlin, 1995) predict course. Most relevant to social relationships, expressed emotion appears highly predictive of relapse (Miklowitz, Goldstein, & Nuechterlein, 1987; Miklowitz, Simonen, Sachs-Ericsson, Warner, & Sudhaff, 1996; O'Connell, Mayo, Flatow, Cuthbertson, & O'Brien, 1991; Priebe, Wildgrube, & Muller-Oerlinghausen, 1989). Even though negative aspects of relationships appear so important, relatively little empirical attention has been paid to positive aspects of relationships, such as social support.

Social support has been shown to have robust effects on a broad range of psychiatric and biological outcomes outside of bipolar disorder. Within unipolar depression, Brown and his colleagues have shown that women experiencing a severe event without support from a confidant had a 40% risk of developing depression; in contrast, those with a confidant's support had a 4% risk (Brown & Andrews, 1986). Other studies have replicated the importance of social support, both directly and as a stress buffer, within depression as well as other psychiatric disorders (Kessler, Price, & Wortman, 1985; Monroe & Johnson, 1992).

The biological vulnerability to bipolar disorder makes the physiological effects of social support particularly intriguing. Social support is strongly linked with both morbidity and mortality (cf. Cohen & Syme, 1985; House, Landis, & Umberson, 1988). Social support correlates with immune functioning, cortisol levels (Uchino, Cacioppo, & Kiecolt-Glaser, 1996), and susceptibility to the common cold (Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997). Intervention and laboratory
studies document social support effects on cardiovascular functioning (Uchino et al., 1996). In short, social support appears to influence a broad range of biological and psychological processes.

For individuals with bipolar disorder, average levels of social support appear low (Cecil, Stanley, Carrion, & Swann, 1995; Romans & McPherson, 1992). Even during remission, bipolar individuals reported fewer contacts with friends than controls or unipolar depressed individuals (Bauwens, Tracy, Pardoen, Vander Elst, & Mendlewicz, 1991; Romans & McPherson, 1992). Nonetheless, not all individuals with bipolar disorder display impairments in social relationships; 55% describe their core relationships as adequate (Romans & McPherson, 1992). The profound psychological difficulties involved in adjusting to this disorder, with its unpredictable symptom fluctuations and difficulties in social functioning, are likely to accentuate the need for social support.

To our awareness, only two published studies have examined the impact of social support on the course of bipolar disorder. In a retrospective study of support preceding a manic episode, bipolar individuals were more likely than orthopedic controls to report the absence of a confiding relationship (Kennedy, Thompson, Stancer, Roy, & Persad, 1983). In the only prospective study, social support at the time of hospitalization predicted better overall functioning and fewer weeks of affective episodes across 1 year (O'Connell, Mayo, Eng, Jones, & Gabel, 1985). The social support measure used in this study, the Personal Resources Inventory (Clayton, Hirschfeld, & Larkin, 1977), however, included domains not typically defined as social support, including household finances, religion, hobbies, and pets. Despite methodological limitations, these studies suggest that social support is an important topic for further investigation within bipolar disorder.

Within this article, we extend previous literature on social support in bipolar disorder. Drawing on the evidence for social support as a stress buffer with other psychiatric conditions, we hypothesized that social support would operate both directly and as a buffer of severe negative life events. Our goal was to understand the role of social support on two different clinical parameters, assuming that social support might not operate uniformly across clinical processes (Monroe & Johnson, 1992). We examined (a) changes in symptom severity and (b) time to recovery from an index episode. Our hypotheses were that social support would predict less severe symptoms of mania and depression and a shorter time to recovery. We also hypothesized that social support would exert more impact among individuals who experienced severe life events.

**Method**

**Design**

Data for this investigation are drawn from an ongoing, naturalistic, longitudinal study of Bipolar I disorder. For the full study, participants are followed for up to 2 years with monthly interviews. Within this article, we include 59 individuals who had completed at least 4 months of follow-up interviews. On average, individuals had 15.15 months ($SD = 7.88$) of data available.

**Participants**

Participants were recruited from hospitals, outpatient clinics, support groups, and community advertising in South Florida and Rhode Island. Sample selection criteria included a *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV; American Psychiatric Association, 1994) diagnosis of bipolar disorder as assessed by the Structured Clinical Interview for DSM (SCID; First, Spitzer, Gibbon, & Williams, 1996) and participant age between 18 and 65 years. Exclusion
criteria included mood disorders secondary to a general medical condition, alcohol abuse or dependence in the past year, substance abuse or dependence in the past year, and inability to speak English or independently complete self-report measures.

Of the current sample, 53% were men. Age ranged from 25 to 65, with a mean of 41.9. The mean number of years of education was 14.76, with a range from 9 years to advanced degrees. Thirty-six percent were employed full-time, 10% were employed part-time, 6% were students, 18% were on disability, 6% were retired, and 24% were unemployed. Using Hollingshead (1957) occupational criteria, 45% had most recently held a job meeting criteria for higher executives, business managers, and administrative personnel.

At the time of study entry, 90% were experiencing a mood episode. Forty-seven percent entered the study in a manic episode, 25% in a depressed episode, 11% cycling from one polarity to another without recovery, and 7% in a mixed episode. Severity of episode was variable. Of those entering the study in an episode, 33% had mild or moderate episodes, and 29% had severe episodes without psychosis. Twenty-one percent had mood-congruent psychosis, and 17% had mood-incongruent psychosis. On average, individuals had experienced 23 lifetime episodes, with only 1 individual entering the study in their first episode.

Procedures

Prior to approaching any potential participant in a treatment center, permission was obtained from the attending physician. All interested individuals completed written informed consent procedures. Potential participants were interviewed using the SCID to determine if they met criteria. After study entry, assessments were completed up to a 2-year period. Symptom severity and treatment status measures were completed monthly. Although these were completed by telephone for the majority of participants, all participants were invited to complete these measures face-to-face if they preferred. At 2-, 6-, 12-, 18- and 24-month interviews, staff who were kept unaware of symptom levels completed life event measures. Social support measures were completed at the 2-month interview. We chose this time period to allow for remission of acute symptoms. We also felt that this time period would allow us to prospectively examine symptom recovery and duration. Any individual too ill to complete an assessment at a given time was rescheduled, so that psychotic, tangential, or disorganized thinking would not interfere with the quality or validity of assessments. Execution of follow-up has been highly successful, with an attrition of only 2 participants for loss of contact and 2 voluntary dropouts. Five others moved from the area. A total of 8 were ruled out early in the study (3 because of ambiguous diagnostic factors, 2 because of mental status, and 3 because of language difficulties). These individuals are not included in the count of 59 participants. To examine the generalizability of our sample, we conducted t tests to compare these noncompleters with the 59 participants. No significant mean differences emerged for the Interpersonal Support Evaluation List (ISEL; Cohen, Mermelstein, Kamarck, & Hoberman, 1985), the Adequacy of Attachment subscale (ADAT; Henderson, Byrne, & Duncan-Jones, 1981), the 2-month Modified Hamilton Rating Scale for Depression (MHRSD), medication adequacy, severity of index episode, or number of lifetime mood episodes.

Measures ISEL.

The ISEL is a 40-item self-report questionnaire developed to assess direct and stress-buffering effects of perceived social support. Scale development was guided by a comprehensive theoretical review of social support (Cohen & Wills, 1985). ISEL subscales capture tangible assistance (material aid),
appraisal (someone to talk to about one's problems), self-esteem support (both positive appraisal of self by others as well as positive self-comparison to others), and belonging (people with whom one can do things). ISEL items include, "If I needed a ride to the airport very early in the morning, I would have a hard time finding anyone to take me" (tangible); "If a family crisis arose, few of my friends would be able to give me good advice about handling it" (appraisal); "Most people I know think highly of me" (self-esteem); and "I often meet or talk with family or friends" (belonging). Each item is answered on a 4-point Likert scale ranging from definitely false to definitely true. In community studies, the ISEL has obtained 6-month test—retest stability coefficients of .74 and high internal consistency (α = .90; Cohen et al., 1985). Within our sample, the ISEL demonstrated strong internal consistency (α = .93). We also obtained high 4-month test—retest stability (r = .83; N = 20, p < .0005), suggesting that individuals remain consistent in their evaluation of social support across time. In a series of longitudinal studies, the ISEL has predicted a variety of psychiatric outcomes, including changes in depression and well-being, as well as stress-buffering effects (Cohen et al., 1985).


Whereas the ISEL measures the general availability of resources, the ADAT measures satisfaction with the resources offered by core relationships. The ISSI includes four subscales: Availability of Social Integration, Adequacy of Social Integration, Availability of Attachment, and Adequacy of Attachment. Of the four ISSI subscales, the ADAT has demonstrated the most robust prediction of psychiatric symptoms following major adversity (Henderson, Byrne, & Duncan-Jones, 1981). ADAT items include, "Is there anyone you'd like to comfort you more in this way (by being held in someone's arms) or is it all right the way it is?" and "Do you think those at home really appreciate you?" Four-month test—retest reliability for the ADAT was high (r = .80, N = 221), and internal consistency was also high (α = .81, N = 756; Henderson, Duncan-Jones, Byrne, & Scott, 1980). Additionally, the ADAT scale has been shown to differentiate bipolar individuals from nonpsychiatric controls (Romans & McPherson, 1992). Within our sample, internal consistency (α = .77; n = 40) and 4-month test—retest reliability (r = .74; n = 24, p < .0005) were adequate. Because the ADAT was added later in the study, data are available for a subset of 37 participants. As one would expect from the different constructs measured, there was a moderate correlation between the ISEL and the ADAT in our sample (r = .48; n = 35; p < .004, two-tailed).

Bedford College Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978).

The LEDS is a semistructured interview and rating system focused on negative life events. The LEDS controls several confounds in life stress measurement. First, because individuals are biased toward shifting the dating of events to help explain the onset of symptoms, interviewers use a timeline with anchors to facilitate accurate event dating (Brown & Harris, 1982; Loftus & Marburger, 1983). Second, because mood symptoms influence how individuals perceive stressors, the LEDS provides objective ratings of long-term threat—the severity for the average person 10—14 days after the event unfolds. Finally, because symptoms or poor coping could provoke life events, each event is rated for independence—whether personality or psychopathology characteristics contributed to the genesis of each stressor. The LEDS achieves increased reliability and predictive validity compared with other life event measures (Gorman, 1995; McQuaid et al., 1992).

To obtain threat ratings, the LEDS interviewer presents each life event and its context to a team of three to four raters who are unaware of symptom severity or the participants' emotional reactions to
events. Although contextual factors will influence threat, severe events generally include the loss of a confidant or immediate family member, threat to relationships in the immediate family, or loss of a close friend. Typically, events such as resolvable arguments, minor car accidents, and smaller financial losses are considered nonsevere. In previous research, nonsevere events have been shown to have little impact on the course of unipolar depression (Brown & Harris, 1978), and we have shown that nonsevere events impact the course of bipolar disorder significantly less than severe events (Johnson et al., 1997).

To rate independence, raters are presented detailed descriptions of how each event transpired. For example, difficulties with lateness or attendance would be considered in evaluating supervisor conflicts. Only events rated independent of personality and psychopathology are included in primary analyses. We conducted separate ratings of whether an event could be attributed to family history of mood disorders. To be congruent with previous research (Johnson & Miller, 1997), current analyses focus on whether individuals experienced at least one severe, independent life event (n = 9) between symptom onset and recovery. Life events that occurred following recovery were excluded.

The first author, Sheri L. Johnson, who trained and supervised all raters for this study, received LEDS interview training through George Brown and Ellen Frank. Brown's "dictionaries," which provide tens of thousands of rating examples (Brown & Harris, 1978), were used to anchor all ratings of threat and independence. High interrater reliability for LEDS threat and independence ratings has been documented by prior research. Reliability for the LEDS raters in this study was monitored on an ongoing basis, and intraclass correlations (Shrout & Fleiss, 1979) were consistently at or above .76 for independence and .77 for threat. Consensus judgments were made where interrater discrepancies occurred. Analyses that focus on the direct effects of life events have been published separately (Johnson & Miller, 1997).

Diagnosis.

Diagnosis was assessed using the SCID. Although data collection began using the DSM-III-R version of the SCID (American Psychiatric Association, 1987), all early interviews were reviewed to confirm that they met DSM-IV criteria, which are comparable for mania. The SCID achieves strong interrater reliability; kappa for bipolar disorder has been shown to be .84 for bipolar disorder (Williams et al., 1992). Within our team, interrater reliability has been high (κ = 1.0 for mania in 7 interviews evaluated by 2 raters, r = .94 for the specific symptoms of mania, N = 74, p < .0001).

Symptom severity.

The MHRSD was used to assess the severity of depressive symptoms (Miller, Bishop, Norman, & Maddever, 1985). The MHRSD was developed to allow paraprofessionals to make reliable and valid ratings of depression severity. This modification of the HRSD includes a standardized interview format and behavioral references for ratings. The 17-item index achieves strong correlations with the traditional HRSD, and we used this index for all analyses. Interrater reliability for the scale is high (intraclass correlation = .93). The MHRSD has been widely used and is sensitive to change in clinical status. Severity of manic symptomatology was assessed using the Bech-Rafaelsen Mania Scale (BRMS; Bech, Bolwig, Kramp, & Rafaelsen, 1979), an interview-based measure of manic symptoms with strong psychometric properties; interrater reliability as assessed using Spearman correlation coefficients has ranged from .97 to .99. The scale is widely used for the detection of changes in clinical status. Our interrater reliability was high (intraclass correlations = .95 for the MHRSD and .92 for the BRMS using methods defined by Shrout & Fleiss, 1979). Each scale assessed
symptoms during the most severe week within the month.

On the basis of standards established by a consensus panel (Frank et al., 1991), recovery was defined as minimal or no symptoms (a MHRSD score less than or equal to 7 and a BRMS score of less than 7) and no hospitalizations for 2 consecutive monthly interviews. Time to recovery was operationally defined as the number of days from symptom onset to recovery. The date of symptom onset was initially established using a Longitudinal Interview Follow-up Evaluation (Keller et al., 1987), then wherever possible it was verified through collateral sources, including family interviews and reviews of medical records. This definition of recovery has been empirically validated; these criteria are associated with longer time to relapse than definitions varying on degree or duration of symptom remission (Rosenberg, Winett, & Johnson, 1998).

Pharmacotherapy adequacy.

Outpatient treatment was categorized on a 6-point scale using the Somatotherapy Index, a modification of the Pharmacotherapy Adequacy Scale used in the NIMH Collaborative Program on the Psychobiology of Depression Clinical Studies Project. The Somatotherapy Index was developed specifically to evaluate treatment for bipolar disorder. Data on medications was drawn from monthly interviews covering the types, dosages, and compliance for each medication and from medical records of blood serum levels. The scale has achieved high interrater reliability (intraclass correlaton calculated using $\alpha = .96$; Bauer, McBride, Shea, & Gavin, 1996). Complicated or unusual treatments were consensually rated. Within this sample, 61% of individuals were maintained on adequate levels of pharmacotherapy, defined as demonstrated blood serum levels of >.50 mEq of lithium, >50 µg/ml of depakote, >4 µg/ml of tegretol, or >200 mg/day of imipramine or an equivalent antidepressant dosage for 4 consecutive weeks.

Training of interviewers.

For training, interviewers were assigned didactic materials, role-plays, and co-interviews. Before completing any interviews, all personnel were required to meet interrater reliability (correlation coefficients and percent agreements of above .90) with a trained interviewer's ratings. Initial interviews were audiotaped and supervised. Regular supervision of interviews was conducted to maintain reliability. Psychiatric consultation was used for differential diagnosis of organic brain syndrome. Routine team meetings were held to consensus ratings and to protect against rater drift.

Results

We first examined clinical characteristics of the study sample and bivariate correlations of the monthly symptom measures with ISEL score. Because this is an ongoing longitudinal study, not all participants have completed each follow-up interview to date. Pairwise deletions of cases were thus employed in subsequent analyses. Standard deviations suggested considerable variability for both depression and mania during the follow-up period (see Table 1). The ISEL mean in this sample, 116.50 ($SD = 21.33$), was substantially lower than the mean of 140 obtained in a normative sample ($t = -8.24$, $df = 55$, two-tailed $p < .0005$; Schonfeld, 1991). Despite these low levels, cross-sectional analyses indicated that social support was negatively correlated with depression ($r_{\text{ISEL-MHRSD month 2}} = -.38$, $N = 56$, $p = .004$). This inverse correlation was not observed between social support and symptoms of mania ($r_{\text{ISEL-BRMS month 2}} = -.12$, $N = 56$, $p = .39$).
Previous literature suggested that social support was unrelated to previous number of episodes and psychiatric severity (Bauwens et al., 1991; Romans & McPherson, 1992). To establish this in the present sample, we conducted a set of preliminary analyses. Pearson correlation coefficients between illness and treatment parameters (severity of index episode, number of hospitalizations, number of depressive and manic episodes, age of illness onset, pharmacotherapy adequacy) and social support (measured by the ISEL and ISSI) were not significant at the .05 level. Similarly, 2-month BRMS and HRSD scores were not significantly correlated with any of these potential confounds. We therefore did not include these variables as covariates in subsequent analyses.

**Survival Analyses**

Analyses were conducted to examine the impact of life events, social support, and their interaction on time to recovery. Cox regression survival analyses were used to conjointly examine the percentage of the sample obtaining recovery as well as the timing of recovery. Individuals who had not obtained recovery by the end of the follow-up period could still be included as censored observations. For these analyses, we excluded individuals who were not experiencing a full-blown episode at the time they began the study, yielding a final sample size of 52.

Time to recovery was the dependent variable for all analyses. Before conducting these analyses, we used survival analyses to examine whether medication level or number of previous episodes predicted time to recovery. As these were not significant predictors, we did not include them as covariates. We first entered the presence of a severe, independent life event. Social support (ISEL) was entered next as a continuous variable. Finally, the interaction of life events and social support was entered. Results revealed that life events functioned as a significant predictor, $\chi^2 (1, N = 52) = 5.12$, one-tailed $p < .01$, as did social support, $\chi^2 (1, N = 52) \text{ change} = 5.89$, one-tailed $p < .01$. Contrary to our hypotheses, the interaction between life events and social support was not significant, $\chi^2 (1, N = 52) \text{ change} = .08$, one-tailed $p = .78$.

Regardless of life events, the median time to recovery for individuals with high social support was 238.27 days, compared with over 1 year for those with low social support. Among individuals who had experienced a severe event, median time to recovery was more than a year regardless of social support, and social support did not even demonstrate a trend toward a buffering effect. Unfortunately, our power to examine social support was limited by the small number of individuals with life events who achieved recovery during the follow-up period. In fact, across the entire sample, 39% of individuals failed to achieve recovery.

To understand the nature of this prolonged nonrecovery, we examined symptoms among individuals who did not recover. Among these individuals, we found a BRMS mean of 6.93 and a MHRSD mean of 11.88 across 6 months, suggesting that nonrecovery often involved chronic subsyndromal symptoms of depression. Furthermore, participants who did not achieve recovery nevertheless may have experienced a considerable range of symptom severities. By using a dichotomous outcome variable—as in survival analyses—these subtle but potentially important differences in symptom severity are obscured.

**Regression Analyses**

To examine the effects of life events and social support on symptoms over time (measured on a
continuum), we conducted two hierarchical multiple regression analyses (see Table 2), following procedures outlined by Cohen and Cohen (1983). One-tailed tests were used for all regression analyses. In predicting depression, the dependent variable was each participant's score on the MHRSD for the 6 months following the social support assessment (Month 3 through Month 8). At Step 1 in this hierarchical regression analysis, the standardized baseline (2-month) MHRSD score was entered, to control for bias due to initial depression levels. In Step 2, five dummy variables representing the time of the assessment were entered, to account for the six time points at which symptom severity was assessed.

In Step 3, the life events and social support variables (standardized) were entered as a set. The life events effect was coded as a dichotomous variable, whereas the social support effect was coded as a continuous variable. In the final step, the interaction between life events and social support was entered. This interactive term was formed by cross-multiplying the standardized life events and social support variables (Aiken & West, 1991; West, Aiken, & Krull, 1996).

In this analysis, significant main effects were observed for baseline MHRSD, life events, and social support (see Table 2). As predicted, high baseline depression, the occurrence of a life event, and low social support were associated with higher depression over time. Time of assessment, represented by the set of dummy variables, did not exert a main effect on MHRSD scores. The interaction between life events and social support was not significant. Contrary to the buffering hypothesis, social support did not appear to mitigate symptom severity among those who had experienced events.

To predict mania symptoms over time, a parallel hierarchical multiple regression analysis was conducted. The dependent variable for this analysis was each participant's score on the BRMS measured from Month 3 through Month 8. Again, baseline (2-month) BRMS scores were entered in a first step, followed by a set of dummy variables that represented time of assessment. At Step 3, life events and social support were entered as a set. In the final step, the interaction of life events and social support was entered. As before, all predictors except dummy-coded variables were standardized. Again, the interactive term was formed by cross-multiplying the standardized life event and social support variables. In this analysis, only baseline BRMS scores exerted a significant main effect on follow-up BRMS scores (see Table 2). Time of assessment, life events, social support, and the interaction of life events and social support were not significant predictors of follow-up BRMS scores.

To assess specific dimensions of social support, we conducted parallel analyses using each of the four subscales of the ISEL as predictors of symptoms over time. Results were entirely congruent with the above regression analyses; whereas three of four subscales (all except "appraisal") were linked significantly with subsequent depression ($r > .36, p < .01$), none interacted with life events in predicting depression, and none were linked with subsequent mania.

In summary, these analyses indicated that although psychosocial risk factors were potent predictors of depression, they did not predict mania over time.

**Interview-Based Measures of Social Support**

We were interested in determining the effects of social support as assessed by the ADAT interview. Although we had only obtained the ISSI on more recent study participants ($n = 37$), we conducted survival analyses and regression analyses to assess the direct effects of the ISSI ADAT scale, similar to those conducted with the ISEL. The effects of life events and the Life Event × Social Support
interaction were not examined because of insufficient power.

For survival analysis, the ADAT scale was the independent variable and time to recovery was the dependent variable. Congruent with the ISEL results discussed above, ADAT was significantly related to time to recovery, $\chi^2 (1, N = 37)= 2.68$, one-tailed $p < .05$. Hierarchical multiple regression analyses of changes in depression and mania were also congruent with ISEL results. In the prediction of depression (MHRSD across 6 months), baseline depression exerted a significant effect ($b = 2.02$, one-tailed $p < .001$), time of assessment was not a significant predictor, and the effect of social support was significant in the predicted direction ($b = -1.49$, one-tailed $p < .01$). In the prediction of mania (BRMS across 6 months), only baseline mania scores exerted a significant effect ($b = 3.13$, one-tailed $p < .0005$). In short, social support appeared to predict depression but not mania, regardless of whether an interview or a self-report measure was employed.

**Discussion**

The current study suggests that individuals with high social support recover more quickly from mood episodes and are less vulnerable to increases in depression over time. Although there has been limited available data on social support in bipolar populations, these findings are congruent with the impact of social support on other psychiatric and medical illnesses.

Contrary to hypotheses, social support did not buffer the effects of life stress. Whereas it is possible that our nonsignificant interaction is secondary to the small number of individuals with a severe life event, trends were not in the anticipated direction. In part, the absence of a stress-buffering effect could be related to the low support in this sample. Although social support seemed to have a direct beneficial effect, more intensive social support may be needed to overcome severe life events. In other words, the level of social support that bolsters daily mood regulation may not suffice when individuals face family deaths, job losses, and other devastating events. Within this highly vulnerable population, the presence of any psychosocial adversity, be it low social support or high life stress, seems to contribute to greater risk for depression and chronic episodes.

Although the current study suggests that social support is important, little is known about the mechanisms linking social support with bipolar symptoms. Several models of social support within unipolar depression have emphasized effects on self-esteem, hopelessness, stress appraisal and coping, as well as biological pathways (Cohen & Wills, 1985; Stroebe & Stroebe, 1996). Clarification of the components and mechanisms of social support would be an important goal for ongoing research.

Current results suggest that social support is not merely a reflection of psychiatric severity, history, or medication compliance, and that it was stable across time. Our study is limited for examining how illness impacts support, however, in that relationships may be most damaged by the first several episodes; most individuals in the current study had experienced multiple episodes. Further, even though we were able to rule out a range of potential confounds, our naturalistic design makes it difficult to exclude the possibility that social support and symptoms both reflect some unmeasured illness or personality parameter. Research on bipolar disorder could benefit from experimental approaches to disentangling social support from other variables, including manipulating emotional support during laboratory-based stressors (Spitzer, Llabre, Ironson, Gellman, & Schneiderman, 1992) and providing social support interventions (Helgeson & Cohen, 1996).

Regression analyses reveal a polarity-specific pattern, in which social support predicts changes in
depressive symptoms but not manic symptoms. This seemed paradoxical, given the vast range of psychiatric and physiological processes protected by social support. Mania appears to be relatively unique in not being affected by social support. At first glance, these results appear contradictory to earlier social support findings, but neither of the previous studies on social support and course actually contrast manic versus depressive symptoms.

Before turning to theoretical interpretations of this finding, we examined several methodological possibilities. We were reassured by the strong psychometric support for the mania measure and the adequate variability in manic symptoms. We queried whether manic symptoms might prompt more aggressive pharmacological treatment than depressive symptoms; however, neither manic nor depressive symptoms were strongly related to treatment adequacy. In the absence of a methodological explanation, we turned toward a more theoretical understanding of this discrepancy.

Our polarity-specific finding is congruent with clinical lore, as well as findings that psychotherapy may be more influential for depressive than manic symptoms (Miklowitz, Simoneau, & Richards, 1997). However, lithium is more effective for manic than depressive symptoms (Hlastala et al., 1997; Sachs et al., 1994), and sleep deprivation provokes mania but not depression (Healy and Williams, 1989; Malkoff-Schwartz et al., 1998; Wehr, 1991). Within genetic studies, phenotype definition on the basis of manic rather than depressive symptoms appears to receive support across a range of methodologies (Simpson & DePaulo, 1998).

One possible framework for understanding the more specific and biological set of predictors for mania than depression is a recent biopsychosocial model of mood, in which manic symptoms can be viewed as markers of excess activity in the behavioral facilitation system (Depue & Zald, 1993) or the behavioral activation system (BAS; Gray, 1994). This system may correspond to dopaminergic pathways in the ventral tegmental area (Depue, Collins, & Luciana, 1996). Excess activity within this neurobehavioral emotional system may be linked with intense affective and behavioral responsiveness to incentives and heightened positive affect and behavioral activation in general (Carver & White, 1994). Despite criticism (Gardner & Wenegrat, 1993), this BAS model is receiving increasing attention (Fowles, 1993; Winters, Scott, & Beevers, in press). One consequence of adopting this BAS dysregulation model is the idea that mania is explained by a relatively narrow physiological process. Conceivably, then, only psychosocial factors that directly impact BAS function may influence mania, whereas this is not a prerequisite for depression. Given that social support and life events influence depression more than mania, these ideas seem worthy of further empirical examination.

Does this model suggest that all psychosocial factors will fail to predict mania? We believe that some psychosocial factors will be explanatory in the genesis of manic episodes. Those factors that relate more specifically to behavioral activation and related paths, such as sleep, are hypothesized to be more powerful.

In contrast, depression may not be explained by a single mechanism: A wider range of pathways may promote the onset and maintenance of depressive symptoms, including psychosocial and biological mechanisms. For example, depressive episodes that follow a manic episode have long been hypothesized to reflect a physiological opponent process (Kukopulos & Reginaldi, 1973), and the biological nature of these episodes is supported by greater medication responsivity compared with other episodes (Maj, 1990). In contrast to these biologically driven episodes, current results indicate that depressive symptoms are influenced by life stress and social support. In short, tentative but somewhat convergent lines of evidence suggest that bipolar depression could be conceptualized as
more etiologically heterogeneous than mania.

One might ask whether these heterogeneous etiological mechanisms correspond to specific symptom patterns. There is a long history of attempts within the unipolar depression literature to identify symptom profiles associated with differential etiologies (Jackson, 1986), such as hopelessness depression (Abramson, Metalsky, & Alloy, 1989; Alloy, Just, & Panzarella, 1997). Further research is necessary to understand whether symptom profiles can be linked with etiological factors within bipolar disorder.

In summary, current results suggest that social support is an important aspect of course within bipolar disorder. Coupled with results from the expressed emotion literature, it appears that both positive and negative aspects of relationships are important determinants of bipolar symptoms. Multifactorial models of illness need to be tested, carefully integrating social with biological factors to compare and contrast impact for differing aspects of illness. Most importantly, however, these results suggest that social support is a meaningful target for intervention, particularly for alleviating specific phases of disorder, such as depression. Interventions focused on interpersonal relationships, such as family therapy and interpersonal psychotherapy, may be particularly important to consider within this context.

References

Brown, G. W. & Harris, T. (1982). Fall-off in the reporting of life events. Social Psychiatry, 17, 23-
28.


1

We conducted parallel analyses including both independent and dependent severe life events (n = 14). Results were entirely comparable.

Clinical Characteristics of Study Sample

<table>
<thead>
<tr>
<th>Category of Life Event</th>
<th>n</th>
<th>Male</th>
<th>Female</th>
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<td>Total</td>
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<td>22</td>
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<tr>
<td>Severe</td>
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<td>7</td>
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<tr>
<td>Dependent</td>
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<tr>
<td>Independent</td>
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Hierarchical Multiple Regression Results