

CHILDHOOD STRESSFUL LIFE EVENTS AND BIPOLAR SPECTRUM DISORDERS

LOUISA D. GRANDIN AND LAUREN B. ALLOY
Temple University

LYN Y. ABRAMSON
University of Wisconsin–Madison

In a cross-sectional study, we investigated the association of reported childhood stressful life events and bipolar spectrum disorders in an undergraduate sample. Specifically, we tested the harsh environment and stress generation explanations of the childhood stress and bipolar disorder relationship. The final sample included 155 bipolar spectrum participants and 155 demographically matched normal controls. Consistent with the harsh environment hypothesis, we found that the total number of reported childhood events, and particularly events independent of one's behavior that occurred prior to bipolar individuals' age of onset (using the same cutoff age for their matched normal controls) were associated with bipolar versus normal control status. Also, in partial support of the harsh environment hypothesis, total number and independent events predicted an earlier onset of bipolar episodes, but inconsistent with the hypothesis, so did dependent events. In contrast to the stress generation hypothesis, a bipolar spectrum diagnosis did not predict the number of total childhood stressors, particularly events dependent on one's behavior occurring after the bipolar individuals' age of onset. These findings suggest that the relationship of childhood stressors and bipolarity may be best explained by a harsh environment effect.

Bipolar disorder has been ranked the sixth leading cause of disability among 15 to 44 year olds (Murray & Lopez, 1996). The prevalence of all subtypes of bipolar disorder combined (Bipolar I, Bipolar II, Cyclothymia,

Louisa D. Grandin and Lauren B. Alloy, Psychology Department, Temple University; Lyn Y. Abramson, Psychology Department, University of Wisconsin - Madison.

This research was supported by National Institute of Mental Health grants MH52617 to Lauren B. Alloy and MH52662 to Lyn Y. Abramson.

Correspondence concerning this article should be addressed to Louisa Grandin, MA, Psychology Department, Temple University, Weiss Hall, 6th Floor, 1701 N. 13th Street, Philadelphia, PA 19122. E-mail: lgrandin@temple.edu

Bipolar NOS) is more than 3% in the United States (American Psychiatric Association, 2000; Wyatt & Henter, 1995). Evidence suggests that individuals diagnosed with bipolar disorder report significantly more difficulties with work-related performance, leisure activities, and family interactions compared to controls. Further, a cost analysis of bipolar disorder estimated the total annual expenditures to be \$45.2 billion for the illness in 1991, and these costs are expected to be much higher today (Wyatt & Henter, 1995). Costs included missing work, inpatient and outpatient treatment, medication, lost income, care-giving costs, and costs to the criminal justice and social welfare systems.

Despite its clear public health significance, bipolar disorder is understudied compared to other mental health disorders (Hyman, 2000). Further research examining psychosocial factors in the onset, course, and progression of the disorder is necessary. The purpose of this study is to examine the potential influence of childhood stress, or life events, on individuals with bipolar disorder. Many studies have documented an association of negative life events with the onset and course of unipolar depression (Monroe & Hadjiyannakis, 2002; Tennant, 2002). Moreover, research has identified a relationship between childhood stressors and both a depressive cognitive style (Crossfield, Alloy, Abramson, & Gibb, 2002; Garber & Flynn, 2001; Gibb et al., 2001; Rose, Abramson, Hodulik, Halberstadt, & Leff, 1994) and the exacerbation of depressive symptoms and the onset of depressive episodes (Gibb et al., 2001, 2006; Hammen, Henry, & Daley, 2000). Despite the consistent finding of a relationship between childhood stressful events and unipolar depression, few rigorous studies have been conducted on the association of childhood stressors with bipolar spectrum disorders.

Recent reviews of the life events and bipolar spectrum disorder association (Alloy et al., 2005; Alloy, Abramson, et al., 2006; Alloy, Reilly-Harrington, Fresco, & Flannery-Schroeder, 2006; Johnson & Kizer, 2002; Johnson, 2005; Johnson & Roberts, 1995) indicate that life events tend to precede both depressive and manic/hypomanic episodes. In addition, Johnson and Miller (1997) found that bipolar individuals who experienced a severe negative life event after the onset of an episode took more than three times longer to achieve full recovery than individuals without a severe negative event. More specifically, several studies have found that bipolar individuals report a higher incidence of childhood abuse than unipolar depressive individuals (Hyun, Friedman, & Dunner, 2000; Levitan et al., 1997; Wexler, Lyons, Lyons, & Mazure, 1997) or than normal controls (Coverdale & Turbott, 2000). Further, several studies have concluded that childhood stressful events prospectively predict unipolar depression (Hammen et al., 2000; Hammen, Adrian, & Hiroto, 1988; Hammen, Burge, & Adrian, 1991).

The aim of the present study was to examine the association of childhood stressful life events with bipolar spectrum disorders. Specifically, we examined two potential explanations of this relationship, the harsh environment and stress generation effects. According to the harsh environment hypothesis, depressed or bipolar individuals report more negative events because they actually have experienced more negative life events that have contributed to the onset of their disorder (Safford, Alloy, Abramson, & Crossfield, 2007). In contrast, the stress generation effect represents the reverse causal direction and suggests that individuals who are depressed or suffer from bipolar disorder generate more negative life events as a result of the characteristics and behaviors associated with their symptoms (Safford et al., 2007; Hammen, 1991). We hypothesized that bipolar spectrum individuals would exhibit evidence for both harsh environment and stress generation effects, given that unipolar depressives have shown both effects (see Figure 1) (Daley et al., 1997; Hammen, 1991; Monroe & Hadjiyannakis, 2002; Simons, Angell, Monroe, & Thase, 1993; Tennant, 2002).

Consistent with prior work designed to test these hypotheses in unipolar depression (Adrian & Hammen, 1993; Hammen, 1991; Safford et al., 2007), we examined the associations between independent (i.e., uncontrollable events independent of an individual's behavior) and dependent (i.e., events at least partly dependent on an individual's behavior) childhood stressful life events and bipolar spectrum diagnosis. If there is a harsh environment effect, we would expect a significant relationship between childhood events that are likely to be independent of an individual's behavior (e.g., death of a grandparent, mother beginning work, had hearing problem) and bipolar spectrum diagnosis. More specifically, given that the harsh environment effect proposes that childhood stressors contribute to the emergence of bipolar symptoms, we examined whether childhood stressful events (particularly independent events) *prior to* the age of onset predict a diagnosis of bipolar spectrum disorder (Hypothesis 1A, see Figure 1). Also, if specific categories of independent events, namely maltreatment, inadequacy, and family deaths, *prior to* the age of onset are related to a bipolar diagnosis, this would further support the harsh environment effect (see Figure 1). Finally, in testing the harsh environment effect, we hypothesized that childhood stressful events would also lead to an earlier age of onset of bipolar disorder (Hypothesis 1B, see Figure 1).

In contrast, if there is a stress generation effect, we would expect to find a significant relationship between childhood events that are at least partially dependent on an individual's behavior (e.g., decrease in acceptance by peers, academic failure, got in trouble with the law), but not independent childhood stressors, and bipolar spectrum diagnosis. Specifically,

given that the stress generation effect proposes that stress arises as a result of one's symptoms, we hypothesized that a bipolar spectrum diagnosis would predict childhood stressful events (particularly dependent events) *after* the age of onset (Hypothesis 2, see Figure 1). Also, if a bipolar spectrum diagnosis predicts specific categories of dependent events, negative emotional and achievement failure events, after the age of onset, this would also support the stress generation effect (see Figure 1).

In all analyses, we controlled for the family history of participants as this has been found to be a significant predictor of bipolarity (Birmaher, Arbelaez, & Brent, 2002; Goodwin & Jamison, 1990; Rush, 2003). We also controlled for current mood state when examining the association between bipolar spectrum diagnosis and reported childhood stressors. This was a conservative approach utilized to decrease the potential biasing effects of a depressive or manic mood state on recall of past experiences. Specifically, individuals who experience depressive or manic/hypomanic episodes may overreport prior stressors in an effort to explain their mood episodes ("effort after meaning;" Brown & Harris, 1979) or as a function of mood state dependent recall (Bower, 1991).

METHODS

PARTICIPANTS

The participants in this study were a subset (those bipolar and normal participants who completed the childhood life events measure and who were exact demographic matches to each other) of those selected for inclusion in the Wisconsin–Temple Longitudinal Investigation of Bipolar Spectrum Disorders (LIBS) Project. This project is a prospective study investigating biological, cognitive and psychosocial predictors of the course of bipolar spectrum disorders as well as of the onset of mania. Given the LIBS project's interest in predicting the first onset of mania, we excluded individuals with bipolar I disorder as they had already experienced mania.

Using the General Behavior Inventory (GBI) (Depue, Krauss, Spont, & Arbisi, 1989), we screened 20,543 students across the two sites, Temple University (TU) and University of Wisconsin (UW). Participants were primarily recruited from undergraduate courses (particularly, Introductory Psychology), but also from flyers advertising the study that were posted around the TU and UW campuses, and received either research credit or \$5 for completing the GBI (Phase I). In Phase II, we conducted 1,730 diagnostic interviews on individuals who met the GBI screening criteria (see Screening Measures below). Of those that met the GBI criteria for a potential bipolar individual ($n = 1042$), 285 met criteria

for a bipolar spectrum diagnosis (12.3% at TU and 6.2% at UW) according to the expanded Schedule for Affective Disorders and Schizophrenic–Lifetime (SADS–L) diagnostic interview (Spitzer, Endicott, & Robins, 1978). Likewise, of the 688 individuals who met the GBI criteria for a potential normal control, 308 met criteria on the SADS–L for a control participant (65.6% at TU and 75.7% at UW).

The final sample for the present study¹ consisted of 155 bipolar spectrum participants (62 men and 93 women), including 39 cyclothymia/bipolar NOS participants (14 men, 25 women), and 116 bipolar II participants (48 men, 68 women), and 155 demographically matched normal control participants (62 men, 93 women). The ages ranged from 18 to 24 years (M age = 19.56, SD = 1.57), which is ideal given that this age range is after the mean onset age of behavioral mood disturbances (14 years), but prior to the mean onset age of bipolar I disorder (24 years) (Akiskal, Djenderedjian, Rosenthal, & Khani, 1977; Depue et al., 1981). The age of onset (M = 12.09, SD = 4.18) for the bipolar participants was defined at the first hypomanic or depressive episode (using the same age for the demographically matched normal control). Given the matching criteria, the ethnic composition of the normal and bipolar spectrum groups was the same, or 74% Caucasian, 12% African–American, 3% Hispanic, 4% Asian, 1% Native American, and 7% Other.

SCREENING MEASURES

Phase I: Self Report Inventory. The GBI (Depue et al., 1981, 1989) is a self-report questionnaire that was used to identify eligible bipolar or normal participants for Phase II of the study. The GBI has good internal consistency (α 's = 0.90–0.96), test-retest reliability (r 's = 0.71–0.74), adequate sensitivity (0.78) and high specificity (0.99) for bipolar spectrum conditions (Depue et al., 1981; 1989). The GBI has been extensively validated in college, psychiatric outpatient, and offspring of bipolar I patient samples (Depue et al., 1981, 1989). Each respondent receives two scores: a total on the depression items (GBI–D) and a total on the hypomania/mania and biphasic items combined (GBI–HB). Cutoff

1. The sample for this study excluded one hundred and twenty six participants primarily due to missing Childhood Life Event Scale (CLES) data. The CLES was added to the LIBS project midway through participant recruitment. However, the sample administered the CLES and included in this study (N = 310) did not differ from the excluded participants on age, gender, BDI scores, HMI scores, or family history (all p 's > .05). The excluded group did have more non-white participants (t = -2.79, p < .05) than the group included in these analyses. This difference is likely because the excluded group was primarily from the urban site (TU) versus the rural site (UW) (t = 3.25, p < .05).

scores were used to determine the two inclusion groups: potential bipolar spectrum participants (GBI-D scale score ≥ 11 and GBI-HB score ≥ 13) and potential normal control participants (GBI-D scale score < 11 and GBI-HB score < 13). These criteria were based on Depue et al.'s (1989) findings and our pilot study in which high and low GBI students, using these cutoffs, were validated against diagnoses derived from SADS-L interviews (Alloy & Abramson, 1999).

Phase II. Diagnostic Interview. The LIBS Project expanded the SADS-L (Endicott & Spitzer, 1978) to accommodate the *Diagnostic and Statistical Manual, Version IV (DSM-IV; APA, 2000)* and Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978). The expanded SADS-L assesses the occurrence, duration, severity and age of onset of symptoms related to mood, anxiety, substance abuse, eating, psychotic, and other disorders over the lifetime. The expanded SADS-L has yielded kappas $\geq .95$ for major depression diagnoses and $\geq .90$ for all unipolar depressive diagnoses based on 80 jointly rated interviews (Alloy & Abramson, 1999; Alloy et al., 2000). An interrater reliability study based on 105 jointly rated SADS-L interviews for this project yielded kappas $> .96$ for bipolar diagnoses (Floyd et al., 2007) and kappa = .89 for the determination of age of onset of bipolar disorder. The training of interviewers consisted of approximately 200 hours of reading and didactic instruction, watching videotaped interviews, discussing case vignettes, and extensive practice conducting live interviews with supervision and feedback meetings. All interviewers were blind to participants' Phase 1 group status and GBI scores. Consensus diagnosis according to the *DSM-IV* and RDC were determined by a 3-tiered standardized diagnostic review procedure involving senior diagnosticians and an expert psychiatric diagnostic consultant.

MEASURES ADMINISTERED AT TIME I

Childhood Stressful Life Events. To identify the occurrence of stressful events during childhood, Crossfield et al. (2002) expanded The Source of Stress Inventory (Chandler, 1981) and called this new measure the Children's Life Events Scale (CLES). The CLES is a checklist composed of 50 moderate-to-major childhood stressful events. The events ranged in severity from less severe but noteworthy events, for example, "poor grades in school," to very severe events, such as "death of a parent" or "experienced sexual abuse, including rape." Participants were also asked their age at the time an event occurred. In the present study, a score on the CLES was derived from the total number of events reported as well as the number of events reported in specific categories. A list of criteria for each category was derived and then, after training with the

categorization scheme, three raters classified events into categories based on these criteria. Interrater reliabilities for the categorization were good to excellent, with intra-class correlation coefficients of .85–.91 (Crossfield et al., 2002). The categories of events represented negative emotional feedback (e.g., “frequent teasing by peers,” “decreased acceptance by peers”), family deaths (e.g., “death of a grandparent,” “death of a parent”), maltreatment (e.g., “experienced physical abuse,” “experienced sexual abuse including rape”), achievement failures (e.g., “academic failure,” “nonacademic failure”), events suggesting inadequacy (e.g., “acquired physical deformity,” “needed special education services”), dependent events (e.g., “became involved in drugs and alcohol,” “got in trouble with the law,” “poor grades in school”) and independent events (e.g., “death of a grandparent,” “mother beginning work,” “loss of a job by a parent,” “vision problem requiring glasses”). Note that the categories of dependent and independent events included events that also appeared in other specific categories.

Chandler (1981) found that the correlation between mothers’ and fathers’ reports on the total number of negative childhood events experienced by their child was $r = .61, p < .01$. We decided to use the CLES because of the wide scope of the events covered on the measure. In the present study, the internal consistency for dependent events and independent events were $\alpha = .63$ and $\alpha = .67$, respectively. Predictive validity of the CLES was demonstrated by Crossfield et al. (2002).

Symptom Measures. The Beck Depression Inventory (BDI) (Beck, Rush, Shaw, & Emery, 1979) is a 21-item self-report inventory that assesses the presence and severity of cognitive, motivational, affective, and somatic symptoms of depression. We used a four month version of the BDI and thus, we averaged across the monthly scores to obtain one score representing participants’ depressive symptoms up until the day that they completed the CLES. The BDI has demonstrated validity for student samples (Bumberry, Oliver, & McClure, 1978; Hammen, 1980). In addition, in a nonclinical population, the internal reliability is good ($\alpha = .81$ to $.86$) and the test–retest reliability ranges from .48 to .86, depending on the interval between re-testing and type of population (Beck, Steer, & Garbin, 1988; Groth–Marnet, 1990).

The Halberstadt Mania Inventory (HMI) (Alloy, Reilly–Harrington, Fresco, Whitehouse, & Zechmeister, 1999; Halberstadt & Abramson, 2007) is a 28-item self-report inventory that measures current manic or hypomanic symptoms. It covers all of the cognitive, motivational, affective and somatic symptoms of mania and hypomania. The HMI was modeled after the BDI format and is thus administered and scored in a similar manner. Halberstadt and Abramson (2007) tested the psychometric properties of the HMI in a sample of 1,282 undergradu-

ates. They found that it had high internal consistency ($\alpha = .82$), adequate convergent validity ($r = .32, p < .001$) with the mania scale of the Minnesota Multiphasic Personality Inventory (MMPI) (Hathaway & McKinley, 1951) as well as discriminant validity ($r = -.26, p < .001$ with the depression scale of the MMPI and $r = -.12, p < .001$ with the BDI). In addition, Alloy et al. (1999) provided evidence of the construct validity of the HMI.

PROCEDURE

Students who met the GBI cutoffs for high GBI and low GBI groups were invited back to participate in a diagnostic interview conducted with the expanded SADS-L. Students who agreed to participate were told that the interview inquired about a broad range of problems and experiences that people sometimes have over their lifetime. All interviews were taped for the purposes of obtaining consensus diagnoses and interrater reliability checks. Students meeting *DSM-IV* and/or RDC criteria for bipolar II, cyclothymia, or bipolar NOS,² but having no lifetime history of mania (or major depressive episodes if diagnosed with cyclothymia or bipolar NOS) were categorized as bipolar spectrum participants. Normal controls with no lifetime history of Axis I disorders (with the exception of one past specific phobia) were recruited and matched to bipolar participants on demographics. Participants were paid \$25 for Phase II. Participants who met all of the above criteria were invited to participate in the LIBS project. All participants in the final sample completed the BDI, HMI, and CLES questionnaires as part of Time 1 of the LIBS project. Participants were paid \$80 for Time 1, which also included other measures not utilized in this study.

RESULTS

PRELIMINARY ANALYSES

Group Differences. We conducted *t*-tests to determine whether the bipolar and normal groups differed significantly in the number of events reported prior to, as well as, after the age of onset (see Table 1). We found that the bipolar group reported significantly more life events prior to, as well as after, the age of onset for most of the event categories, except fam-

2. The bipolar NOS diagnosis included individuals who exhibited recurrent hypomanic episodes without depressive episodes, individuals who exhibited a cyclothymic pattern but with hypomanic and depressive periods that did not meet minimum duration criteria for hypomanic and depressive episodes, and individuals with hypomanic and depressive periods that were too infrequent to qualify for a cyclothymic diagnosis.

TABLE 1. Group Differences on Childhood Stressful Life Events

Events	Bipolar Spectrum Group		Matched Control Group	
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
	Prior to Onset	After Onset	Prior to Onset	After Onset
Total	9.77 (6.60)**	9.39 (7.39)	7.06 (4.75)	8.80 (6.12)
Maltreatment	0.24 (0.63)**	0.15 (0.43)**	0.01 (0.08)	0.00 (0.14)
Family Death	0.52 (0.71)	0.57 (0.73)	0.55 (0.70)	0.39 (0.56)
Negative Emotional	1.15 (1.66)**	0.79 (1.26)**	0.59 (0.93)	0.31 (0.87)
Achievement Failure	0.21 (0.48)**	0.63 (1.25)**	0.13 (0.42)	0.19 (0.48)
Inadequacy	0.66 (0.93)*	0.37 (0.64)	0.59 (1.10)	0.39 (0.66)
Dependent	1.92 (2.49)**	2.98 (2.99)**	1.10 (1.87)	2.57 (2.38)
Independent	7.85 (4.98)**	4.70 (4.16)**	5.96 (3.73)	3.26 (3.14)

Note. *SD* = Standard Deviation. * $p < .05$, in comparison to the same life event category for the normal controls. ** $p < .01$, in comparison to the same life event category for the normal controls.

ily deaths prior to the age of onset and total, family death, and inadequacy events after the age of onset (see Table 1).

Site Differences. T-tests were conducted to determine whether there were differences in the number of childhood stressful life events reported between the sites.³ Given that reports of several event categories differed by site (e.g., achievement failure events prior to the age of onset as well as negative emotional, independent, dependent, and total events after the age of onset), all subsequent analyses with these categories of childhood events controlled for participants' site.

Current Mood State. We performed a series of linear regression analyses to examine whether current levels of depressive and hypomanic symptoms (i.e., BDI and HMI scores) predicted reported childhood stressors that occurred prior to, as well as after, the age of the bipolar participants' first hypomanic or depressive episode. We entered the variables used to match the normal controls to the bipolar spectrum individuals (i.e., age of onset, age, gender, ethnicity) to account for nested effects of our study design as well as family history and site (when necessary). HMI and BDI scores were entered on the second step⁴ to predict childhood stressors.

For events prior to the age of onset, we found that BDI scores signifi-

3. We only examined site differences because we control for the variables that participants were matched on (i.e., ethnicity, age, gender, age of onset) in each analysis. This is a recommended technique when analyzing data matched on specific variables (Campbell & Kenny, 1999; Cohen, Cohen, West, & Aiken, 2003).

4. We performed a Pearson correlation to test the collinearity of current hypomanic and depressive symptoms. Given that these two variables were not significantly collinear ($r = -.05, p = .45$), we entered them on the same step of our linear regression equations predicting childhood stressful life events.

cantly predicted total ($t = 4.05, p < .01$), maltreatment ($t = 5.09, p < .01$), negative emotional ($t = 3.99, p < .01$), dependent ($t = 2.14, p = .03$) and independent ($t = 3.88, p < .01$) childhood stressful events prior to the age of onset.⁵ Therefore, participants with higher depressive symptom scores reported more total, maltreatment, negative emotional, dependent, and independent events prior to the age of onset than individuals with lower depressive symptom scores. HMI scores marginally predicted achievement failure events prior to the age of onset ($t = -1.83, p = .07$). Participants with greater hypomanic symptoms reported fewer achievement failures than individuals with fewer hypomanic symptoms.

For events after the age of onset, we found that BDI scores significantly predicted maltreatment ($t = 2.76, p < .01$), negative emotional ($t = 3.70, p < .01$), and achievement failure ($t = 4.14, p < .01$) childhood stressful events after the age of onset.⁶ Participants with higher depressive symptom scores reported more maltreatment, negative emotional, and achievement failure childhood stressors after the age of onset than individuals with lower depressive symptom scores. HMI scores marginally predicted inadequacy ($t = -1.86, p = .06$) and dependent ($t = 1.83, p = .07$) events after the age of onset. Participants with more hypomanic symptoms reported fewer inadequacy events, but tended to report more dependent events, than individuals with fewer hypomanic symptoms.

Given that current BDI and HMI scores were associated with certain categories of childhood stressful life events, BDI and HMI scores were included as covariates for all subsequent analyses examining the association between childhood stressful events and bipolar spectrum diagnoses. By including the BDI and HMI scores as covariates, we hoped to minimize the effect of reporting biases associated with current mood in our main analyses.

ASSOCIATION OF BIPOLAR SPECTRUM DIAGNOSIS AND CHILDHOOD STRESSFUL LIFE EVENTS

Harsh Environment Hypothesis: Childhood Stressful Life Events Prior to the Age of Onset. To test Hypothesis 1A, we regressed diagnosis on the total

5. We also performed these analyses without incorporating any covariates, because these variables may include variance truly related to the outcome variable (e.g., category of events prior to the age of onset). We found that BDI scores no longer significantly predicted dependent events prior to the age of onset ($\beta = 0.11, t = 1.78, p = .08$). The significance level for the other categories of events prior to the age of onset, predicted by HMI and BDI scores, did not change.

6. We repeated these analyses without any covariates and we found that BDI scores significantly predicted independent events after the age of onset ($\beta = 0.15, t = 2.34, p = .02$). The other categories of events after the age of onset, predicted by HMI and BDI scores, did not change in their level of significance.

number of childhood stressors that occurred prior to the age of the bipolar participants' first hypomanic or depressive episode onset using logistic regression analyses. We entered family history, gender, ethnicity, age, age of onset, current hypomanic and depressive symptoms on the first step and the category of life event prior to the age of onset (as well as site when necessary) on the second step to predict bipolar spectrum diagnosis. We found that the total number, and specifically, independent events prior to the age of onset predicted bipolar spectrum diagnosis (see Table 2).⁷ Thus, consistent with Hypothesis 1A, higher numbers of pre-onset childhood stressors, as well as stressors independent of one's behavior, were associated with a bipolar vs. normal diagnosis (see Figure 1).

Second, to test Hypothesis 1B, we performed a Cox regression (survival analysis) to predict the time (in years) to the bipolar participants' first depressive or hypomanic episode. We entered age of onset of the first episode as the outcome variable and the number of childhood stressors reported prior to the age of onset as the predictor variable. We included family history, age, gender, ethnicity, current hypomanic and depressive symptoms as covariates as well as site when necessary. We found that total, negative emotional, achievement failure, dependent, and independent events significantly predicted the age of first episode onset for the bipolar participants (see Table 3).⁸ Thus, the more events in these categories that the bipolar participants experienced, the younger they were when they had their first bipolar episode (see Figure 1).

Stress Generation Hypothesis: Childhood Stressful Life Events after the Age of Onset. To test the stress generation hypothesis, we conducted linear regression analyses predicting numbers of childhood stressful life events in various categories that occurred after the age of onset from participants' diagnostic status (bipolar versus normal). We entered ethnicity, age, age of onset, gender, family history, site (when necessary), current hypomanic and depressive symptoms on the first step and diagnosis on the second step. We found that bipolar spectrum diagnosis

7. We also performed these analyses without incorporating any covariates. In these analyses, we found that maltreatment events ($Exp(\beta) = 20.66$, $Wald = 8.80$, $p < .01$), negative emotional ($Exp(\beta) = 1.40$, $Wald = 11.70$, $p < .01$), and dependent ($Exp(\beta) = 1.20$, $Wald = 9.37$, $p < .01$) events prior to the age of onset significantly predicted bipolar spectrum diagnosis. The other categories of events prior to the age of onset did not change their level of significance (see Table 2). We report these results to illustrate that we may be overly conservative in our approach.

8. We repeated these analyses without incorporating any covariates. We found no difference between these results and those reported in Table 3.

TABLE 2. Analyses Predicting Bipolar Spectrum Diagnosis from Life Events Prior to the Age of Onset (Harsh Environment Hypothesis) and Predicting Life Events After the Age of Onset from Bipolar Spectrum Diagnosis (Stress Generation Hypothesis)

Events	Prior to Age of Onset ¹			After Age of Onset ²		
	β	Wald	Exp (β)	β	R^2	t
Total	0.09	6.13	1.10**	0.01	0.27	0.18
Maltreatment	1.20	2.21	3.32	0.17	0.13	2.30*
Family Death	-0.08	0.10	0.93	0.17	0.21	2.41*
Negative Emotional	0.04	0.05	1.04	0.07	0.20	1.05
Achievement Failure	0.42	1.25	1.52	0.15	0.12	1.99*
Inadequacy	0.08	0.26	1.09	-0.06	0.20	-0.91
Dependent	0.10	1.38	1.11	0.06	0.07	0.81
Independent	0.12	5.91	1.12*	0.14	0.52	2.48**

Note. ¹ = Analyses conducted with logistic regression. ² = Analyses conducted with hierarchical regression. R^2 = Indicates how well the model fits the data or the proportion of variation explained by model (i.e., the covariates entered on the last step). * $p < .05$. ** $p < .01$.

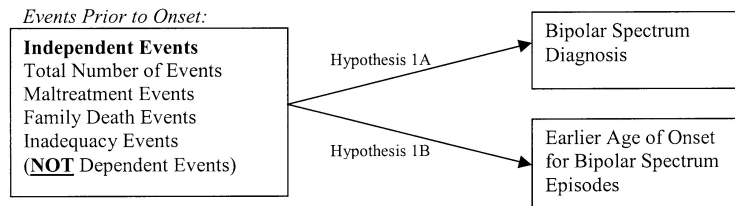
significantly predicted maltreatment, family death, achievement failure, and independent events after the age of onset (see Table 2).⁹

DISCUSSION

The aim of this study was to examine whether the stress generation and the harsh environment hypotheses explained the relationship between reported childhood stressful life events and bipolar spectrum disorders. We found strong support for Hypothesis 1A of the harsh environment effect (see Figure 1). Specifically, we found that the total number of events, and particularly, independent, but not dependent events, prior to the age of onset significantly predicted a bipolar spectrum versus normal diagnosis, even when controlling for current depressive and hypomanic symptoms, match variables, and family history of mood disorders. We obtained mixed support for Hypothesis 1B. Although independent events prior to the age of onset predicted an earlier time to first episode onset among bipolar spectrum individuals as expected (see Figure 1), so did several, unexpected categories of events (i.e., negative emotional, achievement failure, dependent events). These event catego-

9. We repeated these analyses without incorporating any covariates. In these analyses, we found that bipolar spectrum diagnosis significantly predicted negative emotional events after the age of onset ($\beta = 0.22$, $t = 3.88$, $p < .01$). The other categories of events after the age of onset did not change their level of significance (see Table 2).

Harsh Environment Hypothesis



Stress Generation Hypothesis

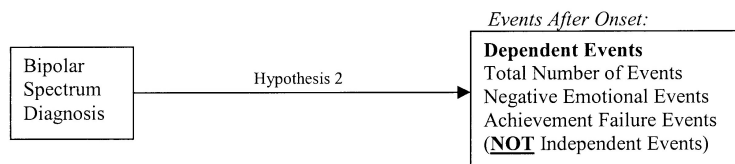


FIGURE 1. Harsh Environment and Stress Generation Hypotheses.

ries were not expected to predict time to first onset of a bipolar episode given that these events are believed to represent the stress generation effect, as they are likely to be dependent on one's behavior. It is possible that prodromal or early occurring symptoms of bipolarity led to the occurrence of these dependent types of events via stress generation mechanisms and then, these events, in turn, increased the likelihood of the earlier onset of a full-blown affective episode.

We found little pure support for Hypothesis 2 or the stress generation effect (see Figure 1). Specifically, we unexpectedly found that a bipolar diagnosis predicted events thought to be independent of one's behavior (harsh environment; i.e., maltreatment, family death, independent) after the age of onset. The only exception was that achievement failure events, considered to be dependent on one's behavior (stress generation), also were predicted by a bipolar spectrum diagnosis. Thus, there was not very strong support for the stress generation hypothesis in explaining the relationship of childhood stress and bipolarity.

These results are slightly perplexing as we expected to find support for both the harsh environment and stress generation effects, given the support for them in the unipolar depression literature (Daley et al., 1997;

TABLE 3. Summary of Cox Regression Analyses for Childhood Stressful Life Events Prior to the Age of Onset

Events	β	SE	Wald	Exp (β)
Total	-0.08	0.02	17.05	0.93**
Maltreatment	-0.04	0.17	0.05	0.96
Family Death	-0.13	0.14	0.95	0.88
Negative Emotional	-0.18	0.07	6.52	0.83**
Achievement Failure	-0.74	0.24	9.71	0.48**
Inadequacy	0.02	0.12	0.02	1.02
Dependent	-0.20	0.05	14.89	0.82**
Independent	-0.07	0.02	8.35	0.94**

Note. ** $p < .01$.

Hammen, 1991; Monroe & Hadjiyannakis, 2002; Simons et al., 1993; Tennant, 2002). However, another study also found that bipolar individuals did not report more dependent events than normal controls (Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999). Yet, there are perhaps other explanations for the absence of a stress generation effect in our bipolar sample. First, the lack of association between dependent events after the age of onset and a bipolar spectrum diagnosis (or Hypothesis 2, see Figure 1) may be due to the possible interaction of the harsh environment and stress generation effects. For example, a teenager with an abusive alcoholic father (harsh environment) may go on a shopping spree with her father's credit card while in a hypomanic episode (stress generation) and, as a result, be beaten by her father (the interaction of both effects). Thus, the effect of dependent stress may be better characterized by an interaction effect.

Another possibility for the lack of an association between dependent stress after the age of onset and bipolarity is that our regression equations examining this relationship were overly conservative. For example, it is possible that our covariates accounted for too much of the overall variance in this relationship or the true variance associated with the independent variable in predicting the dependent variable. This is supported by our post hoc analyses that dependent events, as well as, a specific category of dependent events after the age of onset (i.e., negative emotional) were significantly predicted by a bipolar diagnosis when the covariates were removed from the regression analysis.

However, it is perhaps not surprising that we found more compelling evidence for the harsh environment effect, given other recent findings in the bipolar literature. For example, research has found that stressful environments disrupt bipolar individuals' social rhythms which may, in

turn, lead to dysregulation of their circadian rhythms and onset of symptoms (Frank, Swartz, & Kupfer, 2000; Malkoff-Schwartz et al., 1998). More specifically, Malkoff-Schwartz et al. (2000) found that manic episodes, more so than depressive episodes, are associated with social rhythm disruption brought about by negative life events. In addition, psychotherapy that regularizes bipolar individuals' social rhythms has been effective in improving their mood (Frank et al., 1997, 2005). Thus, individuals predisposed to developing bipolar spectrum disorders in harsh environments may be more apt to develop symptoms because they are more vulnerable to the social and biological disruptions associated with negative life events.

There are several limitations to this study. First, as previously mentioned, we do not account for the possible interaction of independent and dependent events in our analyses. Second, we measured childhood stressful events retrospectively. Therefore, it is possible for participants to forget some of their past events. In addition, reporting biases associated with participants' current mood states could influence their event reporting. We controlled for participants' age and current mood in our analyses to mitigate these confounding variables, but as discussed, this approach may have been overly conservative. Moreover, other possible sources of reporting bias were not controlled. Future longitudinal, prospective studies on the association of childhood stress with bipolar disorder are needed to address these limitations.

It is also important to note that this study did not examine the link between childhood stressors and bipolar I disorder. There may be different relations with stress for individuals with more severe bipolar disorder. Likewise, our sample was derived from a college population, which may reduce the generalizability of our results. Thus, it is important to replicate this study in more representative community samples. We also excluded potential bipolar spectrum participants, as we did not include participants that reported primarily hypomanic symptoms and very few depressive symptoms (i.e., participants who scored high on the GBI-HB scale and low on the GBI-D scale). This design may create selection bias, but we were concerned that such individuals would not be likely to receive a bipolar spectrum diagnosis, as the disorder is rarely manifested without depressive symptoms.

This study is also limited by its method of assessing life events, as the CLES does not account for the context or severity of the events experienced by participants. Brown and Harris (1979) highlight the importance of such information to adequately assess the "contextual threat" or objective impact of the event on the individual. We were apprehensive that participants could accurately identify the context and severity of events that happened years ago and, thus, we opted for the checklist to

minimize further memory bias. Moreover, it is questionable whether events occurring in one's early childhood should be considered contributors to a diagnosis several years later. Several studies assessing life events utilize three months prior to an episode as the critical period by which life events are thought to impact that episode. This study assessed the cumulative impact of such events; further, this is one of the first studies to assess life events after the age of onset. Further, the LIBS project was not designed to assess risk factors from early childhood and thus, parents of our participants were not interviewed. As a result, we do not have an external source of corroboration; however, research suggests that self-reported childhood events correlate highly with (Williamson et al., 2003), or do not significantly differ from (Thaper & McGuffin, 1996), parental report of total, independent, severe, and non-severe events (see Grant, Compas, Thurm, McMahon, & Gipson, 2004, for a review). Further, several of the events on the CLES may have occurred without the parents' knowledge (i.e., became involved in drugs and alcohol, decreased acceptance by peers, frequent teasing by peers). In short, future studies should utilize life events interviews to prospectively assess the impact of life events on bipolar symptomatology.

In sum, our findings suggest that exposure to a harsh environment (uncontrollable stressors) may explain the relationship between childhood stressful events and a bipolar spectrum diagnosis. These results are consistent with the literature on unipolar depression indicating a precipitating role for stressful events (Monroe & Hadjiyannakis, 2002; Tennant, 2002). This study also yields some evidence, although less convincing, for the stress generation effect as well. Taken together, our findings suggest that uncontrollable childhood stressful events may contribute to the emergence and expression of bipolar spectrum disorders.

REFERENCES

- Adrian, C., & Hammen, C. (1993). Stress exposure and stress generation in children of depressed mothers. *Journal of Consulting and Clinical Psychology, 61*(2), 354–359.
- Akiskal, H. S., Djenderedjian, A. H., Rosenthal, R. H., & Khani, M. K. (1977). Cyclothymic disorder: Validating criteria for inclusion in the bipolar affective group. *American Journal of Psychiatry, 134*, 1227–1233.
- Alloy, L. B., & Abramson, L. Y. (1999). The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Conceptual background, design, and methods. *Journal of Cognitive Psychotherapy: An International Quarterly, 13*, 227–262.
- Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, et al. (2000). The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 109*, 403–418.
- Alloy, L. B., Abramson, L. Y., Neeren, A. M., Walshaw, P. D., Urosevic, S., & Nusslock, R.

- (2006). Psychosocial risk factors for bipolar disorder: Current and early environment and cognitive styles. In S. Jones & R. Bentall (Eds.), *The psychology of bipolar disorder and new developments and research strategies* (pp. 11–46). Oxford, UK: Oxford University Press.
- Alloy, L. B., Abramson, L. Y., Urosevic, S., Walshaw, P. D., Nusslock, R., & Neeren, A. M. (2005). The psychosocial context of bipolar disorder: Environmental, cognitive, and developmental risk factors. *Clinical Psychology Review, 25*, 1043–1075.
- Alloy, L. B., Reilly–Harrington, N. A., Fresco, D. M., & Flannery–Schroeder, E. (2006). Cognitive styles and life events as vulnerability factors for bipolar spectrum disorders. In L. B. Alloy & J. H. Riskind (Eds.), *Cognitive vulnerability to emotional disorders* (pp. 93–124). Hillsdale, NJ: Erlbaum.
- Alloy, L. B., Reilly–Harrington, N. A., Fresco, D. M., Whitehouse, W. G., & Zechmeister, J. S. (1999). Cognitive styles and life events in subsyndromal unipolar and bipolar disorders: stability and prospective prediction of depression and hypomanic mood swings. *Journal of Cognitive Psychotherapy: An International Quarterly, 13*, 21–40.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th Ed.–Text Rev.). Washington DC: Author.
- Beck, A. T., Rush, R. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.
- Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty–five years of evaluation. *Clinical Psychology Review, 8*, 77–100.
- Birmaher, B., Arbelaez, C., & Brent, D. (2002). Course and outcome of child and major depressive disorder. *Child and Adolescent Psychiatric Clinics of North America, 11*, 619–638.
- Bower, G. H. (1991). Mood congruity of social judgments. In J. P. Forgas (Ed.) *Emotion and social judgments* (pp. 31–53). Elmsford, NY: Pergamon Press, Inc.
- Brown, G. W., & Harris, T. O. (1979). *The Bedford College Life–Events and Difficulty Schedule: Directory of severity for long–term difficulties*. London: Bedford College, University of London.
- Bumberry, W., Oliver, J. M., & McClure, J. N. (1978). Validation of the Beck Depression Inventory in a university population using psychiatric estimate as the criterion. *Journal of Consulting and Clinical Psychology, 46*, 150–155.
- Campbell, D. T., & Kenny, D. A. (1999). *A primer on regression artifacts*. New York: The Guilford Press.
- Chandler, L. A. (1981). The Source of Stress Inventory. *Psychology in the Schools, 18*, 164–168.
- Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral science* (3rd ed.). Hillsdale, NJ: Lawrence Erlbaum.
- Coverdale, J. H., & Turbott, S. H. (2000). Sexual and physical abuse of chronically ill psychiatric outpatients compared with a matched sample of medical outpatients. *Journal of Nervous and Mental Disease, 188*, 440–445.
- Crossfield, A. G., Alloy, L. B., Gibb, B. E., & Abramson, L. Y. (2002). The development of depressogenic cognitive styles: The role of negative childhood life events and parental inferential feedback. *Journal of Cognitive Psychotherapy, 16*, 487–502.
- Daley, S. E., Hammen, C., Burge, D., Davila, J., Paley, B., Lindenderg, N., & Herzberg, D. S. (1997). Predictors of the generation of episodic stress: A longitudinal study of late adolescent women. *Journal of Abnormal Psychology, 104*, 251–259.
- Depue, R. A., Krauss, S., Spoont, M. R., & Arbisi, P. (1989). General Behavior Inventory identification of unipolar and bipolar affective conditions in a nonclinical university population. *Journal of Abnormal Psychology, 98*, 117–126.
- Depue, R. A., Slater, J., Wolfstetter–Kausch, H., Klein, D., Goplerud, E., & Farr, D. (1981). A behavioral paradigm for identifying persons at risk for bipolar depressive disorder:

- A conceptual framework and five validation studies (Monograph). *Journal of Abnormal Psychology*, 90, 381–437.
- Endicott, J., & Spitzer, R. A. (1978). A Diagnostic Interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, 35, 837–844.
- Floyd, T. D., Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Flannery-Schroeder, E., & Donovan, P. (2007). *Ethnic and gender differences in depressive and hypomanic symptoms and disorders*. Manuscript under editorial review.
- Frank, E., Hlastala, S., Ritenour, A., Houck, P., Tu, X. M., Monk, T. H. et al. (1997). Inducing lifestyle regularity in recovering bipolar disorder patients: Results from the maintenance therapies in bipolar disorder protocol. *Biological Psychiatry*, 41, 1165–1173.
- Frank, E., Kupfer, D. J., Thase, M. E., Mallinger, A. G., Swartz, H. A., Fagiolini, A. M. et al. (2005). Two-year outcomes for interpersonal and social rhythm therapy in individuals with bipolar disorder. *Archives of General Psychiatry*, 62, 996–1004.
- Frank, E., Swartz, H. A., & Kupfer, D. J. (2000). Interpersonal and social rhythm therapy: Managing the chaos of bipolar disorder. *Biological Psychiatry*, 48, 593–604.
- Garber, J., & Flynn, C. (2001). Predictors of depressive cognitions in young adolescents. *Cognitive Therapy & Research*, 25, 353–376.
- Gibb, B. E., Alloy, L. B., Abramson, L. Y., Rose, D. T., Whitehouse, W. G., Donovan, P. et al. (2001). History of childhood maltreatment, negative cognitive styles, and episodes of depression in adulthood. *Cognitive Therapy and Research*, 25, 425–446.
- Gibb, B. E., Alloy, L. B., Walshaw, P. D., Comer, P. D., Shen, G. H. C., & Villar, A. G. (2006). Predictors of attributional style change in children. *Journal of Abnormal Child Psychology*, 34(3), 425–439.
- Goodwin, F. K., & Jamison, K. (1990). *Manic-depressive illness*. New York: Oxford University Press.
- Grant, K. E., Compas, B. E., Thurm, A. E., McMahon, S. D., & Gipson, P. Y. (2004). Stressors and child and adolescent psychopathology: Measurement issues and prospective effects. *Journal of Clinical Child and Adolescent Psychology*, 33, 412–425.
- Groth-Marnat, G. (1990). *The handbook of psychological assessment* (2nd ed.). New York: John Wiley & Sons.
- Halberstadt, L. J., & Adramson, L. Y. (2007). *The Halberstadt Mania Inventory (HMI)*. Madison, WI: University of Wisconsin.
- Hammen, C. (1980). Depression in college students: Beyond the Beck Depression Inventory. *Journal of Consulting and Clinical Psychology*, 48, 126–128.
- Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology*, 100, 555–561.
- Hammen, C., Adrian, C., & Hiroto, D. (1988). A longitudinal test of the attributional vulnerability model in children at risk for depression. *British Journal of Clinical Psychology*, 27, 37–46.
- Hammen, C., Burge, D., & Adrian, C. (1991). Timing of mother and child depression in a longitudinal study of children at risk. *Journal of Consulting and Clinical Psychology*, 59, 341–345.
- Hammen, C., Henry, R., & Daley, S. E. (2000). Depression and sensitization to stressors among young women as a function of childhood adversity. *Journal of consulting and Clinical Psychology*, 68, 782–787.
- Hathaway, S. R., & McKinley, J. C. (1951). *Minnesota Multiphasic Personality Inventory; manual* (Revised). San Antonio, TX: Psychological Corporation.
- Hyman, S. E. (2000). Goals for research on bipolar disorder: The view from NIMH. *Biological Psychiatry*, 48, 436–441.
- Hyun, M., Friedman, S. D., & Dunner, D. L. (2000). Relationship of childhood physical and sexual abuse to adult bipolar disorder. *Bipolar Disorders*, 2, 131–135.
- Johnson, S. L. (2005). Life events in bipolar disorder: Towards more specific models. *Clinical Psychology Review*, 25, 1008–1027.

- Johnson, S. L., & Kizer, A. (2002). Bipolar and unipolar depression: A comparison of clinical phenomenology and psychosocial predictors. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression*. New York: Guilford Press.
- Johnson, S. L., & Miller, I. (1997). Negative life events and time to recovery from episodes of bipolar disorder. *Journal of Abnormal Psychology, 106*, 449–457.
- Johnson, S. L., & Roberts, J. R. (1995). Life events and bipolar disorder: Implications from biological theories. *Psychological Bulletin, 117*, 434–449.
- Levitan, R. D., Parikh, S. V., Lesage, A. D., Hegadoren, K. M., Adams, M., Kennedy, S. H., & Goering, P. N. (1997). Major depression in individuals with a history of childhood physical or sexual abuse: Relationship to neurovegetative features, mania, and gender. *American Journal of Psychiatry, 155*, 1746–1752.
- Malkoff-Schwartz, S., Frank, E., Anderson, B. P., Hlastala, S. A., Luther, J. F., Sherril, J. T. et al. (2000). Social rhythm disruption and stressful life events in the onset of bipolar and unipolar episodes. *Psychological Medicine, 30*, 1005–1016.
- Malkoff-Schwartz, S., Frank, E., Anderson, B., Sherril J. T., Siegel, L., Patterson, D., & Kupfer, D. J. (1998). Stressful life events and social rhythm disruption in the onset of manic and depressive bipolar episodes. *Archives of General Psychiatry, 55*, 702–707.
- Monroe, S. M., & Handjiyannakis, K. (2002). The social environment and depression: Focusing on severe life stress. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression*. New York: Guilford Press.
- Murray, C. J., & Lopez, A. D. (Eds.). (1996). *The global burden of disease: A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020*. Cambridge: Harvard University Press.
- Reilly-Harrington, N. A., Alloy, L. B., Fresco, D. M., & Whitehouse, W. G. (1999). Cognitive styles and life events interact to predict bipolar and unipolar symptomatology. *Journal of Abnormal Psychology, 108*, 1–12.
- Rose, D. T., Abramson, L. Y., Hodulik, C. J., Halberstadt, L., & Leff, G. (1994). Heterogeneity of cognitive style among depressed inpatients. *Journal of Abnormal Psychology, 103*, 242–247.
- Rush, A. (2003). Toward an understanding of bipolar disorder and its origin. *Journal of Clinical Psychiatry, 64*, 4–8.
- Safford, S. M., Alloy, L. B., Abramson, L. Y., & Crossfield, A. G. (2007). Negative cognitive style as a predictor of negative life events in depression-prone individuals: A test of the stress generation hypothesis. *Journal of Affective Disorders, 99*, 147–154.
- Simons, A. D., Angell, K. L., Monroe, S. M., & Thase, M. E. (1993). Cognition and life stress in depression: Cognitive factors and the definition, rating, and generation of negative life events. *Journal of Abnormal Psychology, 102*, 584–591.
- Spitzer, R. L., Endicott, J., & Robins, E. (1978). Research Diagnostic Criteria: Rationale and reliability. *Archives of General Psychiatry, 35*, 773–782.
- Tennant, C. (2002). Life events, stress, and depression: a review or recent findings. *Australian and New Zealand Journal of Psychiatry, 36*, 173–182.
- Thaper, A., & McGuffin, P. (1996). Genetic influence on life events in childhood. *Psychological Medicine, 26*, 813–820.
- Wexler, B. E., Lyons, L., Lyons, H., & Mazure, C. M. (1997). Physical and sexual abuse during childhood and development of psychiatric illness during adulthood. *Journal of Nervous and Mental Disease, 185*, 522–534.
- Williamson, D. E., Birmaher, B., Ryan, N. D., Shiffrin, T. P., Lusk, J. A., Protopapa, J. et al. (2003). The stressful life events schedule for children and adolescents: Development and validation. *Psychiatry Research, 119*, 225–241.
- Wyatt, R. J., & Henter, I. (1995). An economic evaluation of manic-depressive illness: 1991. *Social Psychiatry and Psychiatric Epidemiology, 30*, 213–219.

Copyright of Journal of Social & Clinical Psychology is the property of Guilford Publications Inc. and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.