Attention and memory biases in the offspring of parents with bipolar disorder: indications from a pilot study

Ian H. Gotlib, ¹ Saskia K. Traill, ¹ Rebecca L. Montoya, ¹ Jutta Joormann ² and Kiki Chang ¹

¹Stanford University, USA; ²Ruhr-Universitaet Bochum, Germany

Background: Although children of bipolar parents are at heightened risk for developing emotional disorders, the processes underlying this vulnerability are not well understood. This study examined biases in the processing of emotional stimuli as a potential vulnerability marker of bipolar disorder. Methods: Sixteen children of bipolar parents who did not show any indication of having an emotional disorder at the time of testing and ten children of never-disordered control parents underwent a negative mood induction designed to activate cognitive schemas and were then administered an emotion Stroop task and a self-referent encoding task. Results: Children of bipolar parents were found to exhibit an attentional bias towards social-threat and manic-irritable words. Furthermore, although high- and low-risk children did not differ in their endorsement of positive and negative words as selfdescriptive, the high-risk children demonstrated better recall of negative words than did the low-risk children. Conclusions: Thus, children without a mood disorder who are at high risk for developing a mood disorder were found to exhibit biases in attention and memory that are similar to those found for bipolar and unipolar depressed adults, suggesting that children at increased risk for affective disorder are characterized by potentially pathogenic cognitive structures that can be activated by sad mood. These findings offer insights into mechanisms of cognitive vulnerability for bipolar disorders. Keywords: Children of bipolar parents, vulnerability, depression, risk factors, information processing biases.

Research on affective disorders has begun to focus increasingly on potential markers of risk or vulnerability to these disorders. Because offspring of parents with affective disorders are known to be at heightened risk for developing different types of psychopathology (e.g., Gotlib & Goodman, 1999; Hammen, 1990; Morrison, 1983), the assessment of these children has been an important strategy for elucidating factors associated with increased risk for psychiatric disorder (DelBello et al., 2000; Duffy, 2000). In the specific case of bipolar disorder (BD), Lapalme, Hodgins, and LaRoche (1997) concluded on the basis of a meta-analysis that, compared with children of parents with no mental disorder, children of parents with BD are more than twice as likely to develop any psychiatric disorder, and four times more likely to develop an affective disorder. Recent studies of the offspring of parents with BD have reported a cross-sectional incidence of bipolar spectrum disorders in 14-50% of the offspring (e.g., Chang & Steiner, 2003).

Although there is clearly a substantial heritability component to BD (see Johnson & Kizer, 2002), biological variables alone are not sufficient to explain the wide individual differences observed in the onset, severity, and course of this disorder. Consequently, investigators have increasingly underscored the need to explore psychosocial as well as biological factors that may influence the onset and course of BD (e.g., Johnson & Roberts, 1995; Johnson &

Kizer, 2002). In studies of unipolar depression, cognitive models have proven helpful both for conceptualizing psychological processes that might represent risk factors for the disorder and for developing specific research strategies to identify mechanisms of vulnerability to depression. Cognitive models of depression (e.g., Abramson, Seligman, & Teasdale, 1978; Beck, 1967, 1976; Beck & Young, 1985; Bower, 1981; Teasdale & Dent, 1987) postulate that particular cognitive styles and processes represent vulnerabilities for the development of mood disorders. More specifically, Beck contends that negative schemas that characterize individuals who are at elevated risk for depression are activated in the face of negative life events, leading to a downward spiral into clinically significant depres-

Although early tests of cognitive theories of depression relied on the use of self-report measures of schemas and cognitive functioning, more recent investigations have utilized information-processing paradigms adapted from experimental cognitive psychology. Although there have been exceptions, investigations using information-processing paradigms have generally demonstrated that depressed individuals exhibit negative cognitive biases in both attention to, and memory for, valenced stimuli (see Gotlib & McCabe, 1992, and Gotlib & Neubauer, 2000, for overviews of this research). Indeed, consistent with Beck's (1967,

1976) theory, some investigators have suggested that negatively biased cognitive processing in attention and memory may represent a stable vulnerability factor for unipolar depression (Gotlib & Krasnoperova, 1998).

Cognitive models and research paradigms developed in the study of unipolar depression have recently been extended to investigations of BD (Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999). Consistent with findings of early studies of depression, investigations using self-report measures of cognitive functioning have found that unipolar and bipolar patients exhibit similar patterns of dysfunctional attitudes, negative automatic thoughts, and attributional styles (Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999; Hollon, Kendall, & Lumry, 1986). There are, as yet, very few studies of individuals with BD, or of individuals who are at elevated risk for developing BD, that have used information-processing paradigms to assess cognitive biases; consequently, we know little about the processing of emotional stimuli in these individuals. Lyon, Startup, and Bentall (1999) administered the emotion Stroop task to groups of manic and depressed individuals diagnosed with bipolar I disorder and found greater color-naming interference for depression-related, but not euphoria-related, words in both groups of participants. Similarly, Bentall and Thompson (1990) and French, Richards, and Scholfield (1996) found that a history of hypomanic symptoms among undergraduate students was related to interference for depression-content words on the emotion Stroop task. Lyon et al. (1999) found further that although manic patients endorsed mainly positive words on a self-referent encoding task (SRET), they, like depressed individuals in previous studies, recalled more negative than positive words following the encoding procedure. Finally, among undergraduates with a lifetime diagnosis of either bipolar spectrum disorder or unipolar depression, Reilly-Harrington et al. (1999) found that dysfunctional attitudes, negative attributional styles, and self-referent encoding of negative information interacted with negative events to predict increases in depressive symptoms across a onemonth period. Although Reilly-Harrington et al. did not report separate analyses for participants with bipolar and unipolar disorders, the results are consistent with the formulation that, like unipolar depression, clinical aspects of BD can be predicted from the operation of dysfunctional cognitive pro-

To date, only Jaenicke et al. (1987) have assessed the cognitive functioning of offspring of parents diagnosed with BD. Jaenicke et al. compared the functioning of offspring of four groups of mothers: mothers with BD, mothers with unipolar depression, mothers with a medical illness, and 'normal' mothers. They administered measures of self-concept, attributional style, and, most notably,

the SRET to the children. Jaenicke et al. found that, compared with the offspring of the medically ill and control mothers, the offspring of BD mothers had a less positive self-concept, a more negative attributional style, and less positive (though not more negative) endorsement and recall of words on the SRET. Interestingly, virtually identical results obtained for the offspring of the unipolar depressed mothers. It is important to note, however, that a large proportion of children of both groups of clinically disordered mothers reported high levels of psychological symptomatology (Hammen, 1991), and it is not clear, therefore, whether the findings regarding more negative cognitive functioning among these children are due to their risk status or to their symptomatology.

It is also noteworthy that Jaenicke et al. (1987) made no attempt to activate negative schemas among the children at risk for psychopathology. A number of investigators have underscored the importance of 'priming' or activating cognitive schemas in high-risk individuals (e.g., Miranda & Persons, 1988; Segal & Ingram, 1994). Typically, this priming involves inducing a sad or negative mood in individuals prior to the assessment of schematic functioning. For example, Taylor and Ingram (1999) examined self-referent processing and subsequent recall of positive and negative words in currently nondepressed children of parents with and without MDD. Although no differences were found between the two groups of children in the absence of a sad mood induction, only the high-risk children who were administered a negative mood induction subsequently showed enhanced processing of negative self-referent information and greater recall of negative words. Although these findings suggest that memory biases may represent a risk factor for depression, it is important to note that Taylor and Ingram did not exclude from their study children who had already experienced a depressive episode. Consequently, it is impossible to know whether the biases they found represent a vulnerability to depression or, alternatively, are consequences or 'scars' of having had an episode of depression (Barnett & Gotlib, 1988; Lewinsohn, Steinmetz, Larson, & Franklin, 1981). Nevertheless, Taylor and Ingram's results highlight the utility of using a priming procedure in studies of children at risk for psychopathology.

The present study was designed to investigate biases in attention and memory in young offspring of parents with BD and offspring of parents with no history of psychopathology. All of the children had no history of mood disorders. Following a negative mood induction procedure, children were administered an emotion Stroop task and a self-referent encoding task. We predicted that, compared to the control children, the offspring of parents with BD would demonstrate more negative biases in both attention and memory functioning.

Method

Participants

Participants were 26 English-speaking children between 9 and 14 years of age who had no history of Axis I mood disorders or psychosis. Ten participants (50% female) had biological parents who had no history of Axis I psychopathology, and sixteen participants (60% female) had at least one biological parent diagnosed with Bipolar Disorder (BD). The offspring with at least one bipolar parent were recruited from a larger study of offspring of parents with BD at Stanford University (Chang, Steiner, & Ketter, 2000). Children were identified for eligibility based on their absence of a personal history of mood disorders, and they were asked if they would participate in a separate session. Approximately 90% of eligible children agreed to participate in this study.

The children of parents with no history of psychiatric disorder were recruited through flyers posted in community centers and advertisements placed in newspapers, monthly magazines, and on-line job sites. Those families who were considered likely to be eligible for participation in the study on the basis of a brief telephone diagnostic interview (approximately 35% of those screened) were invited to come to the laboratory to participate in structured clinical interviews (see below). Those parents and children who remained eligible for the study after the clinical interviews (approximately 65% of families interviewed) were asked to return to the laboratory so that the child could participate in the tasks described below. Children and parents were each paid \$20 per session for their participation.

Each group of children included two sets of siblings. In the control group, one set of siblings had three children and one had two children. In the high-risk group, both sibling sets included two children. Although allowing the use of siblings reduces variance in parental psychopathology, the primary purpose of this study was to assess individual differences in information-processing biases among the children. While differences in information-processing biases between siblings are likely to be less than differences between unrelated children, the relatively small number of siblings was not expected to affect the main dependent variables.

Diagnostic evaluation

All parents were interviewed about their current and past psychopathology using the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995), and diagnostic status of the children was assessed with the parents using the Schedule for Affective Disorders for School-Age Children (K-SADS; Puig-Antich & Chambers, 1978). Interviewers were advanced psychology graduate students and post-baccalaureate research assistants, all of whom were trained in the use of the SCID and the K-SADS. Offspring were assessed by the affective module of the Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS) (Geller et al., 2001; Geller, Williams, Zimerman, & Frazier, 1996) and the K-SADS. The WASH-U-KSADS has been shown to reliably distinguish ADHD from BD in child

populations (Geller, Warner, Williams, & Zimerman, 1998a) and to maintain a six-month stability in bipolar diagnosis (Geller et al., 2000). Participants were evaluated either by a child psychiatrist or a trained masterslevel research assistant. Inter-rater reliability was established at the outset by rating videotaped interviews, observing trained rater interviews, and performing interviews with observation by a trained rater, as described by Geller et al. (1998b) (four consecutive patients with 100% agreement on diagnoses). Diagnostic decisions were ultimately made by a child psychiatrist (KC) based on personal interview, discussion with the research assistants, and written notes of parental and subject responses to individual WASH-U-KSADS questions. Current and lifetime diagnoses were established according to DSM-IV criteria. Offspring of parents with BD had at least one parent diagnosed by the SCID with bipolar I or bipolar II disorder. Parents were euthymic at the time of their own and their child's interview. No offspring in this study received a diagnosis of mood disorder, including major depression, dysthymic disorder, cyclothymic disorder, or bipolar I or II disorder.

Demographic information

Demographic information was collected from the parent. All parents with BD were euthymic at the time of completing the questionnaires. Demographic information was collected concerning age of child, gender, ethnicity, household income, both parents' occupations, and levels of education.

Measures

Children's Depression Inventory (CDI). The CDI (Kovacs, 1985) is a 27-item survey measuring cognitive-affective and somatic symptoms associated with depression. Modeled after the BDI, the CDI measures depressive symptoms in children between the ages of 8 and 17. Each item inquires about a depressive symptom within the last 2 weeks and is scored on a 3-point scale. Scores range from 0 to 54. Studies have shown good reliability and validity (Kazdin, French, Unis, & Esveldt-Dawson, 1983).

Assessment of current affect. To measure affect repeatedly throughout the course of the information-processing session, children completed affect sheets. They were asked to rate themselves on four different negative emotions: worried, scared, sad, and unhappy. Ratings were made on a five-point scale, in which increased font size indicated increased level of feeling that emotion. The children were trained on the use of the affect sheet by the experimenter. Our goal was to induce a negative mood and, in particular, sadness. Therefore, we included 'worried' and 'scared' on the affect sheets as control items to evaluate whether we were able to induce a mood that the children would specifically describe as sad and unhappy, rather than worried and scared.

Mood induction. In a meta-analysis of mood induction studies conducted with adults, Westermann, Spies,

Stahl, and Hesse (1996) concluded that film clips with explicit instructions to enter the specified mood state are the most effective form of induction. Importantly, Silverman (1986) analyzed studies using mood manipulations with children and concluded that methodologies used to induce negative mood in adults are also effective with children. Thus, film clips were used to induce negative mood before each information-processing task (emotion Stroop task and self-referent encoding task; see below), and one film clip was used for the positive, or neutralizing, mood induction procedure following the entire information-processing assessment. The first film clip, from Welcome to the Dollhouse (Solondz, 1995), depicted a girl being rejected by her peers in school. The second film clip, from My Girl (Zieff, 1991), depicted a girl's best friend dying and her subsequent grief. The positive mood induction was a section of the film Milo and Otis (Hata, 1989), featuring kittens and puppies frolicking. All of the clips were between three and four minutes long. They were each preceded by instructions to imagine how the characters are feeling, and followed by 30 seconds of guided imagery instructing the children to imagine how they might feel in a similar situation.

Emotion Stroop task. In previous studies, unipolar depressed participants have been found to exhibit interference for depressotypic words (Gotlib & Cane, 1987; Gotlib & McCann, 1984). Consequently, offspring of BD parents, who experience manic and hypomanic episodes, might be expected to exhibit cognitive styles that are more positive than those of unipolar depressed individuals. Therefore, we generated six lists of ten words for this study with the following emotional content: neutral, depressotypic, physically threatening, socially threatening, manic-euphoric, and manic-irritable. We included two types of manic-content words because mania in children may be characterized either by euphoria or by excessive irritability (Findling et al., 2001; Geller et al., 2002). Words were selected from a book of third-grade reading level words (Carroll, Davies, & Richman, 1971), and were rated by psychology undergraduate students on seven-point scales assessing emotional content and specificity. Words were retained for inclusion in the study if they had a mean rating above four for their own category of emotion and a mean rating of less than two for the other categories.

Each category list was printed on separate 81/2" by 11" pieces of paper, which were then laminated. For each list, each word was repeated twice, in a random order, such that all words appeared once in both the first and the second halves of the list. Each word was randomly assigned to be printed in one of four ink colors – red, green, blue, or yellow – with the restrictions that no color appeared more than twice consecutively, and no word appeared twice in the same color. Words were printed in capital letters and in 28-point font so that the list filled the page.

Children were first trained on the task using a practice sheet, and were then timed by a researcher using a stopwatch as they read the colors of the 20 words on each list. The neutral list was presented first to each participant, and the presentation order of remaining five lists was counterbalanced across subjects using a modified Latin square design, such that the depresso-

typic, socially threatening, and physically threatening lists were equally likely to be in the second, third, or fourth positions, and the manic-euphoric and manic-irritable lists were equally likely to be in the fifth and sixth positions. Thus, six different presentation sequences were used in this study, distributed across participants as evenly as possible to avoid any order effects. There were no systematic differences between the groups in the proportion of the various counterbalancing sequences assigned. The later the presentation of a list, the more likely that practice effects would eliminate any Stroop effect. The specific presentation order was chosen, therefore, so that practice effects could not account for the finding of any biases in the processing of the manic-euphoric and manic-irritable lists.

Self-referent encoding task. A set of 40 adjectives was used for this task. These adjectives were all at a third-grade reading level (Carroll et al., 1971), and were rated by research assistants as positive or negative on seven-point scales. Twenty words were identified as positive (rating of greater than four on the positive scale and below two on the negative scale) and twenty as negative (rating of greater than four on the negative scale and below two on the positive scale). The 40 adjectives were presented in a random order to each child using an IBM-compatible computer and a Dell 14-inch color monitor. Micro Experimental Laboratory software and response box (MEL 2.0; Schneider, 1988) were used to control stimulus presentation and record response accuracy and latency for the incidental recall task.

Each child was seated in front of the computer, with the index finger of his/her right hand on a key labeled 'yes' and the third finger of the right hand on a key labeled 'no.' Children were instructed to focus on the cross in the middle of the screen, and were told that they should indicate whether each word that appears on the screen describes them or not. For each trial, the words 'Describes me?' appeared in the center of the screen for 500 msec, followed by a 250 msec pause. Then, one of the stimulus words was presented in capital letters. Children indicated whether the word that appeared on the screen described them by pressing the appropriate key. Following the participant's response, the word disappeared. The inter-trial interval was 1000 msec. Once the practice trials were completed, the experimenter waited outside the room until the participant was finished. The participant was then asked to work on the digit-symbol copying task of the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1939) for three minutes as a distracter task. The experimenter left the room during this time, and then returned with a sheet asking the participant to recall as many words as possible from the previous self-referent encoding task, regardless of whether the words were endorsed as self-descriptive. The experimenter left the room for three minutes, then returned and told the child to stop trying to remember words.

Procedure

In one session, children and their parents completed separate clinical interviews. In a separate second session, children completed the battery of information-

processing tasks. Before the sessions began, the participant and one of his/her parents or legal guardians were asked to sign a consent form allowing participation in the session. In the second session, after the parent left, the child completed the CDI, was trained on how to fill out the affect sheets, and was asked to complete the first affect sheet questionnaire. Then, the child was shown the first film clip and was asked to try to imagine how the main character in the clip was feeling. Immediately after the clip, the child rated his/ her mood on an affect sheet for the second time. Following the mood induction, the participant completed the emotion Stroop task. The child then completed the second mood induction, again rating his/her emotional state after viewing and experiencing the film clip. The child then completed the self-referent encoding task, followed by the distracter task and then the incidental recall task. Finally, the child rated his/her emotional state on an affect sheet and viewed and experienced the positive mood induction film clip.

Results

Participant characteristics

Demographic and clinical characteristics of the participants are presented in Table 1. High-risk and control groups did not differ in age, gender, or ethnicity. They did differ significantly on the Four-Factor Index of Social Position (Hollingshead, 1975), t(19) = 3.72, p < .001), with the control participants obtaining higher scores than the high-risk participants; no group differences were found, however, in reported household income. Importantly, the children in the high-risk group did not differ from their low-risk counterparts on scores on the CDI, t(24) = 1.05; p > .05 (see Table 1). Because of the exclusion criteria, no children had any history of mood disorders. Nevertheless, 5 of the 16 high-risk children were diagnosed with other Axis I disorders: four children of BD parents were diagnosed with Oppositional Defiant Disorder, three were diagnosed with Attention Deficit with Hyperactivity Disorder, two were diagnosed with Generalized Anxiety Disorder, and one was diagnosed with Separation Anxiety Disorder. None of the participants in the control group had a history of any Axis I psychopathology.

Table 1 Demographic and clinical characteristics of the sample

Variable	BDP group $(N = 16)$	Control group $(N = 10)$
Gender, Female N (%) Age, M (SD) Race: Caucasian N (%) Hollingshead SES, M (SD)*	10 (63) 11.14 (1.44) 15 (94) 3.42 (.79) 92.9 (57.8)	5 (50) 11.56 (1.31) 10 (100) 4.56 (.53) 90.6 (18.6)
Household income, M (SD) CDI score, M (SD)	4.25 (4.07)	2.70 (2.79)

Note: BDP = Bipolar Disordered Parent; SES = socioeconomic status; CDI = Children Depression Inventory; *p < .001.

Table 2 Clinical characteristics of parents with bipolar disorder

Variable	Parents with BD	
Only mother BD N (%)	12 (75)	
Only father BD N (%)	3 (19)	
Both mother and father BD N (%)	1 (6)	
Bipolar I Disorder N (%)	9 (56)	
Bipolar II Disorder N (%)	7 (44)	
Age of onset, M (SD)	16.4 (9.36)	

Note: BD = Bipolar Disorder.

The clinical characteristics of the parents in the high-risk group are presented in Table 2. Most of the parents diagnosed with BD were mothers. Mean age of BD onset for the parents was 16.4 years, suggesting that many of our sample of 9–14-year-olds were still at risk for developing a mood disorder.

Mood manipulation check

Our goal with the mood induction was to induce a negative mood, particularly in terms of sadness. Therefore, we averaged the sad and unhappy ratings as a measure of sadness, and we averaged the worried and scared ratings as a measure of anxiety or fear. To test for the effects of our mood manipulation we conducted a repeated-measures analysis of variance (ANOVA) with group (offspring of bipolar parent, offspring of control parent) as the betweensubjects factor, and time (before mood induction, after first mood induction, after second mood induction) and measure (sad vs. anxious) as the within-subjects factors. The ANOVA yielded significant main effects for time, F(2,48) = 13.35, p < .01, and measure, F(1,24) = 7.21, p < .05, which were qualified by a significant interaction of time and measure, F(2,48) = 10.25, p < .01. No significant main effects for group or interactions with group were obtained, all Fs < 2 (see Figure 1 for changes in mood across the session). Post-hoc analyses revealed strong changes in sadness following the first mood induction, t(25) = 4.60, p < .001, which remained stable following the second mood induction, t(25) < 1. No significant changes in worried/scared ratings were obtained, both ts(25) < 1. Thus, we were able to induce a stable and specific mood that the children described as sad/unhappy but not as worried/scared.

Emotion Stroop task

One high-risk child was excluded from these analyses because of experimenter error. Table 3 presents the color-naming times of the two groups of children for the six word lists. A two-way (group repeated over word-list category) analysis of covariance (ANCOVA) was conducted to compare the two groups of children on their color-naming times for the five emotion-content lists, with the color-naming time for the

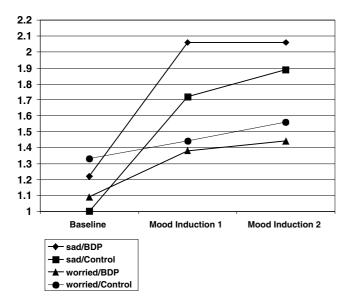


Figure 1 Mean mood ratings on sad/unhappy and worried/scared scales on the affect sheet, separately by group, before mood induction, after first mood induction, and after second mood induction. BDP = Bipolar Disordered Parent

Table 3 Mean reaction times and standard deviations in the emotion Stroop task for high-risk and control children (in ms)

BDP group	Controls
1556 (324)	1598 (294)
` '	1753 (269)
` '	1642 (290)
, ,	1638 (242)
1362 (238) 1454 (286)	1504 (287) 1587 (342)
	1556 (324) 1891 (555) 1582 (347) 1772 (450) 1362 (258)

Note: BDP = Bipolar Disordered Parent; SD in parentheses.

list of neutral words as a covariate to control for individual differences in reading speed. As predicted, the analysis yielded a significant interaction of group and emotion-word category, F(4,88) = 3.01, p < .05. To explore this interaction, we conducted separate one-way ANCOVAs (by group) on the individual emotion-word categories. These analyses revealed that the two groups of children differed in their reaction times to socially threatening,

F(1,22) = 5.52,p < .05, and manic-irritable, F(1,22) = 3.98, p < .05, words; no significant group effects were obtained for the other lists, all Fs < 1. To examine whether the obtained results were due to the presence of children with an Axis I disorder in the high-risk group, we conducted another ANCOVA excluding all children who were diagnosed with an Axis I disorder. Although only nine participants remained in the high-risk group, we nevertheless obtained a significant interaction of group and emotion word category, F(4,60) = 4.36, p < .01. Again, subsequent one-way ANCOVAs (by group) yielded a significant group difference in the color-naming times for the social-threat words, F(1,17) = 7.24, p < .02, and a trend for a group difference in the color-naming times for the manic-irritable words, F(1,17) = 3.58, p < .07.

To present these results graphically, we computed bias scores by subtracting children's reaction times for words in the neutral list from their reaction times for words in the five emotion category lists. Higher bias scores reflect greater interference for the emotional words. These results are presented in Figure 2.

Incidental recall task

One child from the control group was excluded from the analysis because of experimenter error. Table 4 contains the numbers of endorsed positive and negative adjectives, and the number of positive and negative adjectives recalled by the two groups of children. In the first part of the task, children were asked to endorse each of the 20 negative and 20 positive adjectives as self-descriptive or as not selfdescriptive. The two groups of children did not differ in the number of words endorsed within either of the two valence categories, both t(23) < 1. In the second part of the task, children were asked to recall as many of the adjectives as possible. Although children of bipolar parents and the children of control parents recalled an equal number of positive words, t(23) < 1, the high-risk children recalled more negative words than did the control children, t(23) = 2.09, p < .05. These results remained stable when we excluded children diagnosed with an Axis I disorder from the analyses; the two groups of children did not differ in their recall of the positive words, t(17) < 1, but the high-risk children recalled significantly more negative words than did the lowrisk children, t(17) = 2.12, p < .05.

 $^{^1}$ A *t*-test conducted on color-naming speed for words in the neutral list by children in the two groups was not significant, t(23) < 1, indicating that group differences to color-name the words on the various emotion lists were not due to differences in general processing speed. Because our predictions concern the color-naming latencies for emotional words relative to a baseline (i.e., color-naming latencies for neutral words), we used participants' mean color-naming latency for the neutral words as a covariate in our analyses of color-naming latencies for the emotion words. Consistent with the results of these analyses, an alternative single two-way (group by emotion category) repeated-measures ANOVA also yielded a significant interaction of group and emotion category, F(5,115) = 2.30, p < .05.

 $^{^2}$ These planned comparisons supported our *a priori* hypothesis concerning enhanced recall of negative stimuli by highrisk children. A more exploratory two-way (group by emotion category) repeated-measures ANOVA, however, did not yield a significant interaction of group and emotion category for the recall data, F(1,23) < 1.

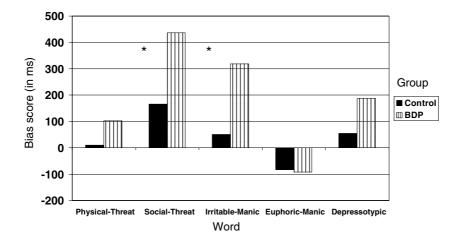


Figure 2 Mean bias scores for reaction times (in msec) to manic-irritable, social-threat, and depression words on the emotion Stroop task separately by group. BDP = Bipolar Disordered Parent; *p < .05

Table 4 Group means and standard deviations of endorsement and recall of valenced adjectives

Valence	BDP group	Controls
Positive adjectives endorsed, M (SD)	` ,	, ,
Negative adjectives endorsed, M (SD) Positive adjectives recalled, M (SD)	, ,	5.44 (2.70)
Negative adjectives recalled, M (SD)	4.13 (1.78)	2.78 (1.39)

Note: BDP = Bipolar Disordered Parent; SD in parentheses.

Discussion

The present study was designed to measure biased cognitive processing of emotional stimuli in the off-spring of parents with bipolar disorder (BD). In order to study cognitive vulnerability as a construct separate from possible prodromal symptomatology, we examined information processing in children who had no history of mood disorders. We predicted that, in a negative mood state, these high-risk children would exhibit a cognitive bias for negative information, demonstrating greater attention to, and better memory for, negative than positive stimuli.

Confirming our predictions, children of BD parents showed greater interference from negative word lists on the emotion Stroop task than did the control children. More specifically, they showed significantly longer color-naming times on the lists containing manic-irritable and socially threatening words; the two groups of children did not differ in their colornaming times on the list of depressotypic words. Finally, although the two groups of children endorsed the same number of negative and positive adjectives on the self-referent encoding task, the high-risk children remembered more of the negative adjectives than did the low-risk children.

Thus, in the present study, children of bipolar parents who are at elevated risk for developing a mood disorder exhibited biases in attention and memory similar to those found for unipolar and bipolar depressed adults. In a negative mood state,

high-risk children exhibited characteristics of a cognitive vulnerability for affective disorder. These data suggest that children at increased risk for affective disorder are characterized by potentially pathogenic cognitive structures that can be activated by sad mood. Therefore, the present results provide further support for the applicability of cognitive theories of unipolar depression to the bipolar spectrum. The present findings are conceptually consistent with studies that have demonstrated biases in attention and memory in response to a negative mood induction in remitted depressed participants, who, like the children of BD parents in the present study, are at elevated risk for the development of an affective disorder (Gotlib & Neubauer, 2000; Hedlund & Rude, 1995). Moreover, the few studies that have been conducted to date examining informationprocessing biases in adult bipolar patients have provided evidence of Stroop interference for depressotypic words (French et al., 1996; Lyon et al., 1999). Lyon et al. (1999) also found that patients diagnosed with mania exhibited better recall of negative than positive words on a self-referent encoding

To our knowledge, no study to date has used a negative mood induction to assess cognitive processes in children of bipolar parents. Taylor and Ingram (1999) found that children of unipolar depressed mothers recalled a higher percentage of the negative words that they endorsed on the SRET than did children of nondepressed control mothers. Interestingly, under a negative mood induction, the highrisk children in Taylor and Ingram's study also endorsed fewer positive self-descriptors than did the low-risk children, a finding not replicated in the present study. Although the differences in the results of these two studies may reflect differences in cognitive vulnerability to unipolar versus bipolar affective disorders, it is also possible that they are a function of an important methodological difference between these two studies. Whereas Taylor and Ingram included children with symptoms of depression in their high-risk sample, that was not the case in the present study. It may be, therefore, that endorsement of fewer positive and more negative adjectives as self-descriptive is related to current symptomatology, a possibility that should be explored more explicitly in future research.

Based on the results of the studies described above using the emotion Stroop test with bipolar adults, we had hypothesized that the children of bipolar parents would exhibit interference for depressotypic words following a negative mood induction procedure. Although the high-risk children exhibited interference for manic-irritable and socially threatening words, they did not exhibit interference for depression-content words. It is possible that the interference for manic-irritable and socially threatening words among the children of BD parents was elicited by one of the two films used to induce negative mood, which depicted teasing, bullying, and aggression in young children. It is also possible that the interference for these two categories of words is due to the poor premorbid social functioning and social adjustment that has been found to characterize children at risk for bipolar disorder (Cannon et al., 1997), or to the comorbid oppositional and anxious characteristics of the children of BD parents in this sample. Because we did not predict interference for these two content categories, it will be important for future studies to replicate these findings.

As we noted earlier, one might have expected offspring of bipolar parents to exhibit cognitive biases that are more positive than those exhibited by children of control parents. The present results indicated, however, that control and high-risk children did not differ with respect to either Stroop interference for manic-euphoric words or recall of positive words on the SRET. It is important to note that all children underwent a negative mood-induction procedure, and it is possible that this procedure is not optimal for assessing vulnerability to mania. Although the results of a number of studies suggest that negative life events can trigger both depressive and manic episodes in people with BD (Johnson & Roberts, 1995; Malkoff-Schwartz et al., 1998; Reilly-Harrington et al., 1999), it is possible that negative mood inductions have more specific effects on the activation of cognitive vulnerabilities. In future research it will be instructive to induce a euphoric mood in highrisk individuals to assess attentional and memory biases for euphoric-mania content. Given the similarity between irritable mania and symptoms of irritability and hostility in depression, gaining a better understanding of cognitive biases for euphoric-manic content may help to distinguish between those high-risk individuals who will go on to develop MDD and those who will develop BD. This distinction might also aid in detecting individuals with MDD who might have an underlying bipolar

diathesis, which would be useful for planning treatment

We should note that there are a number of limitations of this study. For example, with 16 at-risk and 10 control children, the statistical power of this study was not as high as we would have liked. Nevertheless, despite the relatively small sample size, we did obtain statistically significant results that were consistent with our stated hypotheses. Certainly, a replication of the present results with a larger sample is warranted. In addition, because of our focus in this study on vulnerability, we excluded children with a mood disorder from participation. Five of the sixteen high-risk children, however, were diagnosed with at least one other Axis I disorder. We included these children to increase the representativeness of our sample of offspring of parents with bipolar disorder. Importantly, excluding these children from the analyses did not alter the pattern of results obtained in this study. Nevertheless, future research should compare more explicitly the functioning of high-risk children with and without Axis I disorders. Finally, we did include a number of dyads in which the offspring were siblings of participants in other dyads. Although the inclusion of these siblings reduced the independence of the group data, analyses without siblings, too, yielded virtually identical results, albeit with reduced power.

Despite these limitations, the findings of the present study demonstrate that individuals with a parental history of BD who are known to be at elevated risk for developing mood disorders but who are not yet disordered are characterized by negative biases in information processing. These results indicate that cognitive theories that were derived from unipolar depression may be applicable to the bipolar spectrum, and underscore the need for further research assessing cognitive vulnerabilities in samples at elevated risk for the development of bipolar disorder.

Correspondence to

Ian H. Gotlib, Department of Psychology, Bldg. 420, Jordan Hall, Stanford University, Stanford, California 94305, USA; Email: gotlib@psych.stanford.edu

References

Abramson, L.Y., Seligman, M.E., & Teasdale, J.D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology*, 87, 49–74.

Alloy, L.B., Reilly-Harrington, N., 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 depressive and hypomanic mood swings. *Journal of Cognitive Psychotherapy*, 13, 21–40.

- Barnett, P.A., & Gotlib, I.H. (1988). Psychosocial functioning and depression: Distinguishing among antecedents, concomitants, and consequences. *Psychological Bulletin*, 104, 97–126.
- Beck, A.T. (1967). Depression: Clinical, experimental, and theoretical aspects. New York: Harper and Row.
- Beck, A.T. (1976). Cognitive therapy and the emotional disorders. New York: New American Library.
- Beck, A.T., & Young, J.E. (1985). Depression. In D.H. Barlow (Ed.), *Clinical handbook of psychological disorders: A step-by-step treatment manual* (pp. 206–244). New York: Guilford Press.
- Bentall, R.P., & Thompson, M. (1990). Emotional Stroop performance and the manic defence. *British Journal of Clinical Psychology*, 29, 235–237.
- Bower, G.H. (1981). Mood and memory. *American Psychologist*, 36, 129-148.
- Cannon, M., Jones, P., Gilvarry, C., Rifkin, L., McKenzie, K., Foerster, A., & Murray, R.M. (1997). Premorbid social functioning in schizophrenia and bipolar disorder: Similarities and differences. *American Journal of Psychiatry*, 154, 1544–1550.
- Carroll, J.B., Davies, P., & Richman, B. (1971). Word frequency book. New York: American Heritage.
- Chang, K., & Steiner, H. (2003). Offspring studies in child and early adolescent bipolar disorder. In B. Geller & M.P. DelBello (Eds.), *Bipolar disorder* in childhood and early adolescence (pp. 107–129). New York: Guilford Press.
- Chang, K.D., Steiner, H., & Ketter, T.A. (2000). Psychiatric phenomenology of child and adolescent bipolar offspring. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 453–460.
- DelBello, M.A., Soutullo, C.A., Ryan, P., Graman, S.M., Zimmerman, M.E., Getz, G.E., et al. (2000). MRI analysis of children at risk for bipolar disorder. *Biological Psychiatry*, 47(Suppl.), 43.
- Duffy, A. (2000). Toward effective early intervention and prevention strategies for major affective disorders: A review of antecedents and risk factors. *Canadian Journal of Psychiatry*, 45, 340–348.
- Findling, R.L., Gracious, B.L., McNamara, N.K., Youngstrom, E.A., Demeter, C.A., Branicky, L.A., et al. (2001). Rapid, continuous cycling and psychiatric co-morbidity in pediatric bipolar I disorder. *Bipolar Disorders*, 3, 202–210.
- First, M.B., Spitzer, R.L., Gibbon, M., & Williams, J.B.W. (1995). Structured Clinical Interview for DSM-IV Axis I Disorder-Patient Edition. New York: Biometrics Research Department, New York State Psychiatric Institute.
- French, C.C., Richards, A., & Scholfield, E.J.C. (1996). Hypomania, anxiety and the emotional Stroop. *British Journal of Clinical Psychology*, *3*, 617–626.
- Geller, B., Warner, K., Williams, M., & Zimerman, B. (1998a). Prepubertal and young adolescent bipolarity versus ADHD: Assessment and validity using the WASH-U-KSADS, CBCL and TRF. *Journal of Affective Disorders*, *51*, 93–100.
- Geller, B., Williams, M., Zimerman, B., Frazier, J., Beringer, L., & Warner, K.L. (1998b). Prepubertal and early adolescent bipolarity differentiate from ADHD by manic symptoms, grandiose delusions, ultra-rapid or ultradian cycling. *Journal of Affective Disorders*, 51, 81–91.

- Geller, B., Zimerman, B., Williams, M., Bolhofner, K., Craney, J.L., Delbello, M.P., et al. (2000). Six-month stability and outcome of a prepubertal and early adolescent bipolar disorder phenotype. *Journal of Child and Adolescent Psychopharmacology*, 10, 165–173
- Geller, B., Zimerman, B., Williams, M., Bolhofner, K., Craney, J.L., DelBello, M.P., et al. (2001). Reliability of the Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS) mania and rapid cycling sections. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40, 450–455.
- Geller, B., Zimerman, B., Williams, M., Delbello, M.P., Frazier, J., & Beringer, L. (2002). Phenomenology of prepubertal and early adolescent bipolar disorder: Examples of elated mood, grandiose behaviors, decreased need for sleep, racing thoughts and hypersexuality. *Journal of Child and Adolescent Psychopharmacology*, 12, 3–9.
- Geller, B.G., Williams, M., Zimerman, B., & Frazier, J. (1996). WASH-U-KSADS (Washington University in St. Louis Kiddie Schedule for Affective Disorders and Schizophrenia). St. Louis, MO: Washington University.
- Gotlib, I.H., & Cane, D.B. (1987). Construct accessibility and clinical depression: A longitudinal investigation. *Journal of Abnormal Psychology*, *96*, 199–204.
- Gotlib, I.H., & Goodman, S.H. (1999). Children of parents with depression. In W.K. Silverman & T.H. Ollendick (Eds.), *Developmental issues in the clinical treatment of children* (pp. 415–432). Needham Heights, MA: Allyn & Bacon.
- Gotlib, I.H., & Krasnoperova, E. (1998). Biased information processing as a vulnerability factor for depression. *Behavior Therapy*, 29, 603–617.
- Gotlib, I.H., & McCabe, S.B. (1992). An information-processing approach to the study of cognitive functioning in depression. In E.F. Walker, B.A. Cornblatt, & R.H. Dworkin (Eds.), *Progress in experimental personality and psychopathology research* (vol. 15, pp. 131–161). New York: Springer Publishing Company.
- Gotlib, I.H., & McCann, C.D. (1984). Construct accessibility and depression: An examination of cognitive and affective factors. *Journal of Personality and Social Psychology*, 47, 427–439.
- Gotlib, I.H., & Neubauer, D.L. (2000). Information-processing approaches to the study of cognitive biases in depression. In S.L. Johnson, A.M. Hayes, T.M. Field, N. Schneiderman, & P.M. McCabe (Eds.), *Stress, coping, and depression* (pp. 117–143). Mahwah, NJ: Lawrence Erlbaum.
- Hammen, C. (1990). Longitudinal study of diagnoses in children of women with unipolar and bipolar affective disorder. *Archives of General Psychiatry*, *47*, 1112–1117
- Hammen, C. (1991). Depression runs in families: The social context of risk and resilience in children of depressed mothers. New York: Springer-Verlag.
- Hata, M. (director). (1989). Milo and Otis [Film].
- Hedlund, S., & Rude, S.S. (1995). Evidence of latent depressive schemas in formerly depressed individuals. *Journal of Abnormal Psychology*, 104, 517–525.

- Hollingshead, A.B. (1975). *Four-factor index of social status*. New Haven, CT: Yale University Department of Sociology.
- Hollon, S.D., Kendall, P.C., & Lumry, A. (1986). Specificity of depressotypic cognitions in clinical depression. *Journal of Abnormal Psychology*, 95, 52–59.
- Jaenicke, C., Hammen, C., Zupan, B., Hiroto, D., et al. (1987). Cognitive vulnerability in children at risk for depression. *Journal of Abnormal Child Psychology*, 15, 559–572.
- 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* (pp. 141–156). New York: Guilford Press.
- Johnson, S.L., & Roberts, J.E. (1995). Life events and bipolar disorder: Implications from biological theories. *Psychological Bulletin*, 117, 434–449.
- Kazdin, A.E., French, N.H., Unis, A.S., & Esveldt-Dawson, K. (1983). Assessment of childhood depression: Correspondence of child and parent rating. *Journal of the Academy of Child Psychiatry*, 22, 151–164.
- Kovacs, M. (1985). The Children's Depression Inventory (CDI). *Psychopharmacology Bulletin*, 21, 995–1124.
- Kovacs, M., & Beck, A.T. (1977). An empirical-clinical approach toward a definition of childhood depression. In J. Schulterbrandt & A. Riskin (Eds.), *Depression in childhood: Diagnosis, treatment, and conceptual models* (pp. 555–583). New York: Raven.
- Lapalme, M., Hodgins, S., & LaRoche, C. (1997). Children of parents with bipolar disorder: A meta-analysis of risk for mental disorders. *Canadian Journal of Psychiatry*, 42, 623–630.
- Lewinsohn, P.M., Steinmetz, J.L., Larson, D.W., & Franklin, J. (1981). Depression-related cognitions: Antecedent or consequence? *Journal of Abnormal Psychology*, 90, 213–219.
- Lyon, H.M., Startup, M., & Bentall, R.P. (1999). Social cognition and the manic defense: Attributions, selective attention, and self-schema in bipolar affective disorder. *Journal of Abnormal Psychology*, 108, 273–282.
- Malkoff-Schwartz, S.F., Frank, E., Anderson, B., Sherrill, 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: A preliminary investigation. *Archives of General Psychiatry*, 55, 702–707.
- Miranda, J., & Persons, J.B. (1988). Dysfunctional attitudes are mood-state dependent. *Journal of Abnormal Psychology*, 97, 76–79.
- Morrison, H.L. (Ed.). (1983). *Children of depressed parents: Risk, identification, and intervention.* New York: Grune & Stratton.
- Puig-Antich, J., & Chambers, W. (1978). Schedule for Affective Disorders and Schizophrenia for School-Age Children (6–16 years)–KIDDIE-SADS. New York: Psychiatric Institute.
- 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, 567–578.
- Schneider, W. (1988). Micro experimental laboratory: An integrated system for IBM PC compatibles. *Behavioral Research Methods, Instruments and Computers*, 20, 206–217.
- Segal, Z.V., & Ingram, R.E. (1994). Mood priming and construct activation in tests of cognitive vulnerability to unipolar depression. *Clinical Psychology Review*, 14, 663–695.
- Silverman, W.K. (1986). Psychological and behavioral effects of mood-induction procedures with children. *Perceptual and Motor Skills*, *63*, 1327–1333.
- Solondz, T. (director and producer). (1995). *Welcome to the Dollshouse* [Film]. New York: Todd Solondz.
- Taylor, L. & Ingram, R.E. (1999). Cognitive reactivity and depressotypic information processing in children of depressed mothers. *Journal of Abnormal Psychology*, 108, 202–208.
- Teasdale, J.D., & Dent, J. (1987). Cognitive vulnerability to depression: An investigation of two hypotheses. *British Journal of Clinical Psychology*, 26, 113–126.
- Wechsler, D. (1939). The measurement of adult intelligence. Baltimore: Williams & Wilkins.
- Westermann, R., Spies, K., Stahl, G., & Hesse, F.W. (1996). Relative effectiveness and validity of mood induction procedures: A meta-analysis. *European Journal of Social Psychology*, 26, 557–580.

Zieff, H. (director). (1991). My Girl [Film].

Manuscript accepted 14 January 2004