J Abnorm Child Psychol (2008) 36:849–863
DOI 10.1007/s10802-008-9217-9

The Role of Family Functioning in Bipolar Disorder in Families

Tina D. Du Rocher Schudlich · Eric A. Youngstrom · Joseph R. Calabrese · Robert L. Findling

Published online: 13 February 2008

Abstract Investigated the association between family functioning and conflict and their links with mood disorder in parents and with children's risk for bipolar disorder. Participants were 272 families with a child between the ages of 5–17 years. Parents' history of psychiatric diagnoses and children's current diagnoses were obtained via semi-structured interviews. Parent report on the Family Assessment Device and the Conflict Behavior Questionnaire measured family functioning and conflict, respectively. Results revealed a small but significant indirect pathway from parental diagnosis of mood disorder to child bipolar disorder through impaired family functioning, via increased family conflict. Parental mood disorders were also significantly related to other negative outcomes in children, including unipolar depression and oppositional defiant disorder. Associations between parent diagnoses and family functioning changed depending on youth age, but not youth sex.

T. D. Du Rocher Schudlich (✉)
Department of Psychology, Western Washington University,
516 High Street, MS 9089, Bellingham, WA 98225-9089, USA
e-mail: Tina.Schudlich@wwu.edu

E. A. Youngstrom
Department of Psychology,
University of North Carolina at Chapel Hill,
Chapel Hill, NC, USA

E. A. Youngstrom · J. R. Calabrese · R. L. Findling
Department of Psychiatry, Case Western Reserve University,
Clevelend, OH, USA

J. R. Calabrese · R. L. Findling
Department of Psychiatry,
University Hospitals Case Medical Center,
Clevelend, OH, USA

Keywords Bipolar disorder · Family functioning · Family conflict · Child outcomes · Parental mood disorders

Children of parents with bipolar spectrum disorders are at heightened risk for a broad range of psychiatric disorders (e.g. attention-deficit hyperactivity disorder and disruptive behavior disorders) and adjustment problems, including social and academic difficulties and poor overall functioning (Singh et al. 2007). Furthermore, they are at specific risk for developing mood disorders, such as major depressive disorder and bipolar disorders (DelBello and Geller 2001; Lapalme et al. 1997). Although rates vary depending on methodology, the most recent meta-analyses indicate that having a parent with a bipolar disorder increases risk of bipolar disorder in children by a factor of at least five (Hodgins et al. 2002).

However, many children with bipolar parents do not develop adjustment or mental health problems, demanding further explication of the processes that modify children's adjustment (Cummings et al. 2000).

Genetic influences, as well as environmental influences including family processes such as marital difficulties, parenting problems, and chronicity and severity of parental illness, are established risk factors in children of affectively disordered parents (Hammen 2002; Hodgins et al. 2002). Because of the family environment's pervasive influences on child development, family functioning may be of particular importance. Whereas evidence supporting the role of family processes related to parental depression in children's risk for adjustment problems is substantial (for reviews see Cummings and Davies 1994; Downey and Coyne 1990; Hammen 2002; Schwoeri and Sholevar 1994; Miklowitz 2004), little work has been done exploring the role of family processes related to parental bipolar disorder in children's risk for problems. Because much of the burden

associated with bipolar illness comes from depressed mood states in adults (Judd et al. 2005), it is likely that findings from the parental depression literature hold important implications for family processes in families with bipolar disorder (Keitner et al. 1985; Miklowitz 2004).

Families with depressed parents are often under great marital and parent–child stress (Hammen 1991). High levels of family stress predict children's subsequent depression (Hammen 1988; Hammen et al. 1991). Negative family interactions are more predictive of children's outcomes than is the depression itself (Caplan et al. 1989; Fergusson et al. 1995; Hammen et al. 1987; Murray et al. 1999). Parents' depressive symptoms are associated with multiple differences in interaction patterns, including increased angry and depressive marital conflict tactics, verbal hostility, defensiveness, withdrawal, and insults, as well as more negative affect displays of anger and sadness (Du Rocher Schudlich et al. 2004). Affected families also show diminished positive or productive strategies, such as calm discussion, positive affect and conflict resolution. Thus, the conflict strategies utilized by couples with a dysphoric partner not only hinder problem resolution, but could also seriously impair relations and communication between the couple. Such interaction styles linked with dysphoric affect may be particularly detrimental to children's outcomes (Davis et al. 2000; Du Rocher Schudlich and Cummings 2003). This is especially true when considering effects of genetic vulnerability for negative affect, which may undermine children's coping resources for effectively managing the stress of such negative family environments (Rice et al. 2002).

Research addressing bipolar disorder's role in family functioning has been scarce and inconsistent regarding the impairment these families face. Findings to date potentially support three plausible suppositions: (a) mania and mixed states further disrupt family processes and are associated with worse outcomes, (b) the unique aspects of bipolar illness, mania and hypomania, do not add substantially to the disruption of family processes compared to the burden of depression, or (c) bipolar disorder might be associated with periods of higher functioning, possibly including hypomanic periods, that actually reduce the effects of depression on family functioning.

The most intuitive expectation would be that bipolar disorder leads to greater disruptions of family processes. Families with children with bipolar disorder acknowledged significantly more minor conflicts with family members than either unipolar or control group families (Robertson et al. 2001). Families with parent mood disorders also reported significantly less positive shared activities and communication between siblings. Another recent study found that families reported more conflict, less organization, and less cohesion compared to controls when one or both parents had bipolar disorder (Chang et al. 2001). These features of family interaction were in turn associated with the presence of bipolar disorder and other axis I disorders in children. Given the already increased biological risk for disorder that bipolar offspring face (Hodgins et al. 2002; McGuffin et al. 2003), these disruptions in family functioning may place children at even greater risk for developing adjustment and psychiatric problems.

However, other studies have found that families with parents with bipolar disorder do not differ from control families regarding the severity of family dysfunction (Anderson and Hammen 1993; Cooke et al. 1999) or in several specific areas of family functioning (e.g. problem solving, communication, roles, affective responsiveness, affective involvement, behavior control and general functioning; Miller et al. 1986). Children of unipolar mothers may be under even greater levels of family stress than children of bipolar mothers (Adrian and Hammen 1993). Thus, family impairment may be due more to something specific about depression rather than affective disorders in general. Furthermore, evidence is beginning to suggest that parental bipolar disorder and disturbed family functioning may be associated with a more general risk for psychiatric disorders and maladjustment in children, rather than bipolar disorder specifically (Chang et al. 2001; Radke-Yarrow 1998; Zahn-Waxler et al. 1988). Thus, it is important to examine other disorders in children as potential alternative negative outcomes.

The goal of this study is to further explicate the relations between parental mood disorder, family functioning/conflict and children's risk for disorder. Thus, we are focusing on both the risk that children of parents with affective disorders face and the predictors of bipolar disorder in children, which is reflected in our sample where participants are recruited based on children's disorders, with additional information obtained about parents' disorders. Based on the possible predictions described above, our tests are two-tailed. Our first hypothesis was that a history of bipolar spectrum disorders in either parent would be associated with impaired family functioning, including poorer problem solving and communication (Fristad et al. 2002; Miklowitz et al. 2005). We also hypothesized that poor communication, problem solving, and general family functioning would predict increased levels of conflict in the family. This expectation was based on therapeutic models that target deficient problem solving and hostile communication as processes (Danielson et al. 2004; Fristad and Goldberg-Arnold 2004; Miklowitz 2004). A third hypothesis was that family functioning and conflict would partially mediate the association between mood disorder in the parent and in the youth. We expected to find evidence of mediation because of the prior evidence reviewed above about parental mood disorder being linked with poorer

family functioning and increased conflict, and also because of findings that poor family functioning is associated with age of onset and risk of relapse in pediatric bipolar disorder (Geller et al. 2004). We expected that mediation would be only partial because of the well-established genetic risk associated with bipolar spectrum disorders (McGuffin et al. 2003).

An alternate model of clinical interest would be a "child effects" model (Forehand and McCombs 1988; Lytton 1990), suggesting that the externalizing behavior problems frequently associated with pediatric bipolar disorder (Kowatch et al. 2005; Youngstrom et al. 2004) might result in an excess of conflict above what might be predicted just on the basis of poor family functioning. This would be consistent with evidence about coercive processes, where youths' aversive behavior can contribute to increased conflict in the family and more ineffective parenting (Patterson et al. 1989), as well as the high degree of stress reported in parents of youths with bipolar diagnoses (Hellander et al. 2003). Past work by Hammen et al. (1991) demonstrated that dysfunctional youths were as likely to contribute to maternal mood episodes as much as the reverse, and that child characteristics contribute to maternal functioning in a cycle of negative mutual influence (e.g., Conrad and Hammen 1989; Ge et al. 1995). Thus, path analyses also examined whether a child effects or a bidirectional model provided a better fit to the data.

Four additional sets of secondary questions were also explored in this study. First, we compared whether specific types of conflict and family functioning had different effects on the risk of disorder in youths. Second, we examined whether any particular category of youth diagnosis (bipolar spectrum, unipolar, ADHD, or disruptive behavior disorders such as ODD or CD) was uniquely associated with family functioning even after accounting for comorbidity in a highly mixed outpatient sample. Third, we examined whether youths' age moderated any of the patterns of association between parental mood status, family processes, and youth risk of bipolar spectrum disorders. Age of onset appears to be an important prognostic variable, with earlier onset potentially signifying a worse course of the disorder (e.g. Geller and Luby 1997; Geller et al. 2004; Perlis et al. 2004). Therefore, it is important to examine whether family processes and models for their effects differ as a function of child age. Finally, we examined whether there were sex differences in the patterns of association. Prior research has found significant but complicated sex effects regarding family functioning in families with and without affective disorders (see Cummings and Davies 1994, and Snyder 1998 for reviews). Also, there are sex differences in the rate of depression, but not of bipolar I disorder (Biederman et al. 2004).

Method

Participants

The Institutional Review Board of University Hospitals of Case Medical Center approved all procedures used here. Participants for this study were 272 families with a child between the ages of 5–17 who were recruited to a general clinical research center conducting research in treatment of disruptive behavior disorders, attention-deficit/hyperactivity disorder, mood disorders, and schizophrenia. The entire sample was recruited from this one clinical infrastructure, and all subjects received the same assessment protocol. Recruitment included three referral streams: Parents contacting the Division of Child and Adolescent Psychiatry because of concerns about their youth's functioning (the most common source); referrals from the adult mood disorders program for evaluation of youths who had a parent receiving treatment for bipolar disorder; and a smaller group of children (n=31) who were recruited as a non-treatment seeking comparison group. Data collection pre-dated the STARD guidelines for describing patient recruitment and representativeness (Bossuyt et al. 2003), so patient flow information was not documented to that degree of detail (see Youngstrom et al. 2004, for a STARD flow diagram prepared for the larger cohort from where this sample was drawn).

Assessments took place at an outpatient clinic in an urban Midwestern city. Inclusion criteria were: (a) youths between the ages of 5 years 0 months and 17 years 11 months of age, (b) of either sex, (c) of any ethnicity, (d) the youth providing written assent and the guardian providing written consent for participation, and (e) both the youth and the primary caregiver presenting for the assessment. Exclusion criteria included: (a) either the youth or the parent being unable to communicate orally at a conversational level in English in order to complete the interview; (b) having a pervasive developmental disorder, as determined by psychiatric history, psychiatric interview, or having an Autism Screening Questionnaire score of 15 or higher (Berument et al. 1999); or (c) suspected mental retardation—documented via educational history, standardized cognitive ability test scores <70, or a *Peabody Picture Vocabulary Test-Third Edition* (Dunn and Dunn 1997) score <70 as a screener. Parents provided demographic information regarding themselves and their family, including age, marital status, family composition and living arrangements, occupational status, employment type, educational level, and total income.

One hundred and eighty-three of the mothers (69%) were married. Thirty-five percent of the children were only children; the median number of additional siblings was 1, with an average of 1.3 additional sibs (SD=1.5). Fifty-one

percent of parents were living together, 38% were living apart, and 9% never lived together. Children lived primarily with both parents (52%), 43% lived with their mothers only, and 5% with their fathers only. Seventy-nine percent of the target children had one to three siblings and 21% had four or more siblings. Mean age of the index child participating in the study was 11.57 years (SD=3.24). There were 158 boys (58%) and 114 girls (42%) in the sample.

Mothers averaged 39 years of age (SD=6.4) and fathers averaged 42 years of age (SD=7.0). Summary information gathered from the parents' occupational status, employment, education and income indicated that the families were primarily middle to upper middle-class. Approximately 3% of mothers and 12% of fathers obtained less than a high school education, 28% of mothers and 33% of fathers obtained a high school education, 40% of mothers and 21% of fathers attended some college, 12% of mothers and 16% of fathers graduated from college, and 18% of mothers and 13% of fathers obtained a post-graduate education. Approximately 82% of families were Caucasian, 10% were African American, 3% were Hispanic, 1% were Native American, and 4% were other or did not specify.

Table 1 presents the distribution of bipolar diagnoses in offspring stratified by biological parent history of mood disorder. Diagnostically, 100 youths (37%) met criteria for bipolar I, 50 (18%) met for other bipolar spectrum diagnoses (7 for bipolar II, 13 for cyclothymia, and 30 for bipolar NOS—typically presenting with a sufficient number and severity of symptoms to warrant a diagnosis of mania, but with insufficient duration of the index mood episode to meet DSM-IV criteria), 63 (23%) met for unipolar depression or dysthymia, 28 (10%) met for ADHD or disruptive behavior disorders without a comorbid mood diagnosis, and 31 (11%) did not meet criteria for any axis I disorder. The median number of axis I diagnoses was two, with 60% of the sample meeting criteria for anywhere from two to six diagnoses. ADHD was the most frequent comorbidity, affecting 51% of the sample, including 66% of the cases with bipolar spectrum diagnoses.

Table 1 Distribution of bipolar diagnoses in offspring stratified by biological parent history of mood disorder

Father diagnosis	Mother diagnosis				Total
	Child bipolar status	Bipolar	Unipolar	Neither	
Bipolar	Not bipolar	0	2	2	4
	Bipolar	5	16	9	30
Unipolar	Not bipolar	0	10	6	16
	Bipolar	5	10	9	24
Neither	Not bipolar	8	26	68	102
	Bipolar	29	30	37	96
Total		47	94	131	272

Measures

Family Assessment Device (FAD) The FAD is a parent report questionnaire measuring transaction patterns that can distinguish healthy from unhealthy families (Epstein et al. 1983). A 27-item short form of the FAD shows good reliability and validity for young children and adolescents (Byles et al. 1988). The FAD has been used widely with families with children as young as 4 years (e.g., Byles et al. 1988; Sanford et al. 2006; Zwaanswijk et al. 2005). The primary *caregiver* rated each statement based on how well it described their own family, using a four-point Likert-type scale, ranging from a 1 (*Strongly Agree*) to 4 (*Strongly Disagree)*. Ten of the items are reverse keyed, so that higher total scores (after reversal of items that are keyed negatively) always mean less adaptive family functioning. In the present sample, the total score showed α=0.91 in the younger (ages 5 to 10) and 0.93 in the older (ages 11 to 17 years) subsamples; the general functioning scale showed α=0.84 and 0.87, the problem solving scale had α=0.75 and 0.79, and the communication scale had α=0.73 and 0.81, respectively.

Conflict Behavior Questionnaire (CBQ) The short form of the Conflict Behavior Questionnaire measured family conflict (Prinz et al. 1979; Robin and Foster 1989). The short form correlates 0.96 with the longer version (Robin and Foster 1989), with adequate internal consistency and discriminant validity (Prinz et al. 1979). The CBQ is a measure of perceived communication-conflict between parents and children and taps both dissatisfaction with family member's behavior and evaluations of the interactions between family members. The CBQ has been widely used with families with young children as well as adolescents (e.g. Forehand and Jones 2003; Kim and Brody 2005; Kotchick et al. 2005). Sample items include "My child is easy to get along with," "We almost never seem to agree" and "My child and I compromise during arguments." The *caregiver* most familiar with the target child indicated whether or not each behavior occurs in their family (yes or no). Six of the items are reverse keyed in calculating the total score, and higher scores indicate greater severity. The CBQ showed α= 0.92 (ages 5 to 10) and 0.94 (ages 11 to 17) in the present sample.

Child Diagnoses: Semi-structured Diagnostic Interview Using the Schedule of Affective Disorders and Schizophrenia for Children All participants and their families com-

pleted a semi-structured diagnostic interview by a highly trained research assistant, using the Schedule for Affective Disorders and Schizophrenia for School-Age Children—Present and Lifetime (KSADS-PL) or Epidemiological (KSADS-E). The same interviewer worked with both parent and child sequentially, resolving discrepancies using best clinical judgment (see Findling et al. 2001). Diagnoses of bipolar I, bipolar II, cyclothymia, and bipolar not otherwise specified (NOS) were made in strict accordance with diagnostic criteria published in the DSM-IV (American Psychiatric Association 2001). NOS cases typically had a sufficient number and severity of symptoms, but were insufficient in duration of spontaneous abnormal mood states to warrant a strict DSM-IV diagnosis of mania (Leibenluft et al. 2003).

Research assistants (n=17, ranging from BA in psychology to PhD or MD) were trained to criterion by having them rate along while observing five KSADS interviews by an experienced rater. New raters then led five KSADS interviews with an experienced rater and achieved an overall kappa >0.85 at the symptom severity level and 1.0 agreement about the presence or absence of diagnoses on each in order to graduate from training. Acceptable inter-rater reliability was maintained by having joint rating sessions at every tenth interview or monthly, whichever happened first.

Comorbidity was addressed via a hierarchy for grouping diagnoses (e.g., Youngstrom et al. 2001; Youngstrom et al. 2004), such that children with a bipolar spectrum diagnosis were always assigned to the bipolar category, regardless of comorbidity; children with unipolar mood were assigned to the depression category. In a similar manner, a disruptive behavior disorders (DBD)/attention deficit hyperactivity disorder (ADHD) category was formed of cases with externalizing diagnoses but no comorbid mood diagnoses—because if a youth met criteria for both an externalizing and a mood diagnosis, they were already assigned to one of the prior mood categories. Finally, 31 youths presented with no axis I diagnosis. These were recruited specifically to form a non-impaired comparison group, although they were retained in the sample even if they happened to score high on measures of symptoms or impairment due to challenges in daily living.

Child Mood State The same interviewer conducting the KSADS also used parent report, youth report, and clinical observations during the interview to complete the Young Mania Rating Scale (YMRS; Young et al. 1978) and the Child Depression Rating Scale-Revised (CDRS-R; Poznanski et al. 1984). These measures were chosen as they are the assessments most commonly employed in clinical trials research with children and adolescents suffering from mood disorders and they provide additional more fine-tuned measurement of the mood symptoms that youth are experiencing. The YMRS contains seven items rated from 0 to 4, and four items rated from 0 to 8, with high scores indicating worse mania (α=0.90 in present sample). The YMRS demonstrates good reliability between raters and has good discriminant and concurrent validity in children and adolescents in the age ranges used in this study (Fristad et al. 1992). The CDRS-R contains 12 items rated 1 to 7 and five items rated 1 to 5, with higher scores indicating more severe depression (α=0.92 in this sample). The CDRS demonstrates good reliability between raters and across time and has good criterion and concurrent validity (Poznanski et al. 1984).

Parental Mental Health Diagnoses After the initial KSADS diagnostic assessment interview, the parents were assessed for psychiatric diagnoses. Primary caregivers were directly interviewed using Schedule for Affective Disorders and Schizophrenia (SADS-LB; Endicott and Spitzer 1978), conducted by the same highly-trained pool of raters as performed the KSADS. Thus, all raters had completed extensive training and supervision on using semi-structured interviews, as detailed above. When possible, the other parent was also directly interviewed with the SADS-LB. Caregivers provided indirect report about other family members using the Family History Research Diagnostic Criteria (FH-RDC; Andreasen et al. 1977) to describe the history of the other relatives. Clinical diagnoses were also considered if they were made at the affiliated adult mood disorders program and families consented. More than 90% of mothers and more than half of fathers completed direct SADS interviews. Parent diagnoses were reviewed using similar consensus procedures as used with the child KSADS diagnoses. Review by a physician at consensus meetings resulted in changes of diagnosis in fewer than 5% of cases.

Procedure

The parent or guardian provided written consent, and all youths provided written assent to participation. All participants and their families completed the intake assessment. Family history of mental illness was gathered either during the initial evaluation or at a subsequent interview as needed to avoid undue burden on the family. Raters were neither systematically informed of child diagnoses, nor were strict precautions taken to preclude awareness of child diagnoses (Youngstrom and Kogos Youngstrom 2005). Most of the family history interviews were completed prior to the consensus meeting where final child diagnoses were assigned, but not all family history was completely blind to child diagnosis.

Results

Preliminary Analyses

Preliminary analyses checked the appropriateness of distributions for subsequent analyses. Univariate descriptive statistics indicated that variables were functionally not different from normal distributions (absolute values of observed skewness and kurtosis all <1.0, with no extreme outliers on box plots). Because of the large age range of participants, we examined whether the reliability or average scores on the CBQ or FAD scales were associated with child age and gender independent of child diagnosis. For group comparisons of age effects, age was divided into ten years and younger versus eleven years and older, based on age eleven as being when many self-report instruments begin to be used, and also based on the transition to puberty often occurring around this age (Petersen et al. 1988). No significant associations were found between child age and the CBQ or any of the FAD scales. No significant associations were found between the CBQ and child gender, and only one significant association was found between child gender and the FAD: Girls' families had significantly worse problem solving than boys' families $F(1, 271)=4.75$, $p<0.03$. Internal consistency reliability coefficients remained similar even when stratifying by youth age (as detailed in the "Measures").

Is Parental Mood Disorder Associated with Family Functioning or Conflict? (Hypothesis 1)

We examined the distribution of bipolar diagnoses in offspring, stratified by the history of mood disorder in both biological parents. Table 1 indicates the breakdown of unipolar and bipolar disorder in both parents. Slightly more than half of the children had a current bipolar spectrum diagnosis, as assessed by the KSADS-PL, whereas 17% of mothers and 12.5% of fathers had a history of bipolar disorder. Thirty percent of children had at least one parent with a bipolar diagnosis and 2% had both parents with bipolar diagnoses. Seventy-nine percent of children with at least one parent with a history of bipolar spectrum disorder had a current diagnosis of a bipolar spectrum disorder, and 100% of children with both parents with a history of bipolar spectrum disorder had a current bipolar spectrum disorder diagnosis, potentially indicating very strong direct effects.

ANOVA examined the first hypothesis, that parental mood disorder would be associated with poorer family functioning and increased conflict. Post-hoc comparisons used the Games-Howell procedure, which is more robust to heterogeneity of variance than the Tukey HSD procedure and provides better type I error control than the Newman–Keuls and other commonly use procedures, but provides better power than the Bonferroni adjustment (Kirk 1995). Point-biserial correlations provided a measure of the effect size of parental unipolar and bipolar diagnoses on family functioning and conflict. Table 2 presents the results of a one-way ANOVA examining differences in family measures as a function of family history of mood disorder. Both parents having a history of mood disorder was associated with significantly more overall impaired family functioning, as measured on all subscales of the FAD, compared to families without any history of mood disorder. Additionally, families where *both* parents had a history of mood disorder had significantly worse general family functioning

Table 2 ANOVAs for family functioning and conflict measures as a function of family risk for mood disorder

Child family risk	FAD total score		FAD general functioning scale		FAD problem solving scale		FAD communication scale		CBQ	
	M	(SD)	*M*	(SD)	*M*	(SD)	*M*	(SD)	*M*	(SD)
No parent with mood disorder	55.1_a	9.4	24.0_a	4.7	12.8_a	2.4	18.3_a	3.2	10.9	6.4
One parent with unipolar disorder	57.7	12.3	25.1_a	5.9	13.5	2.7	19.1	4.8	11.6	5.8
One parent with bipolar disorder	57.2_a	12.3	25.1_a	6.0	13.0_a	3.0	19.1	4.4	10.9	5.9
Both parents with mood disorder	63.2_b	9.5	28.5_b	4.9	14.5_b	2.9	20.3_b	3.2	13.0	5.8
	$F(3, 268)=6.27***$		$F(3,268)=7.74***$		$F(31,268)=4.55**$		$F(31,268)=3.06*$		$F(31,268)=1.53$	

Within columns, means with different subscripts are significantly different from each other only, not the rest of the column, at $p\leq0.05$. *FAD* Family Assessment Device, *CBQ* Conflict Behavior Questionnaire.
$*p\leq0.05$, $**p\leq0.01$, $***p\leq0.001$

compared to families with just one parent with a mood disorder. Family problem solving was also significantly worse when *both* parents had a mood disorder compared to having just one parent with a bipolar spectrum disorder. No differences were found in families' degree of conflict as a function of family history of mood disorder. In other words, conflict levels were similar across families who did and who did not have any type of mood disorder. The pattern of findings suggests that the cumulative load of parental mood disorder (number of affected parents) may have more impact on family functioning than does the specific type of mood disorder. Both parental unipolar depression and bipolar diagnoses showed significant correlations with poorer family functioning (Table 3). Thus, the quantity was more important than the quality of the mood disorders.

Does Poor Family Functioning Predict Increased Conflict? (Hypothesis 2)

Correlation also quantified the relationship between family functioning and conflict. Table 3 presents the correlations between measures of family functioning and conflict. All FAD scales correlated with CBQ conflict, with *r*s ranging from 0.27 to 0.47, thus supporting the hypothesis. These findings are unlikely to be due entirely to "source variance," as parent diagnoses were not directly correlated with parent-reported conflict.

Are Child Bipolar Diagnoses Associated with Differences in Family Functioning and Conflict?

Table 3 presents the correlations for all the variables included in the path analyses. Parental mood disorders were significantly related to impaired family functioning, as assessed by both the total score and the general family functioning scales on the FAD. Parental unipolar, but not bipolar, disorder was further related to impairment in problem solving. Greater conflict in the family, as assessed by the CBQ, was related to child bipolar spectrum disorders. The family functioning variables did not show significant correlations with bipolar diagnoses in the child. This appears to be due to the fact that the majority of the nonbipolar cases still had depression or DBD diagnoses, which also were associated with higher levels of problematic family functioning. Table 4 presents the means for the FAD scales and CBQ Total broken down by the child's diagnostic category. One-way ANOVAS were conducted to examine between group differences in family functioning and conflict by child diagnostic group. The "no diagnosis" group consistently showed better functioning than the other diagnostic groups. There were no significant differences in functioning between the bipolar, unipolar depression, and DBD/ADHD groups. If the sample were limited to include only bipolar cases versus youths with no diagnosis, then the bipolar cases would show worse family functioning. Overall, these findings are consistent with disrupted family function-

Table 3 Zero-order correlations between mood diagnoses and family functioning and conflict measures

	Child bipolar diagnosis[a]	Parent unipolar diagnosis[a]	Parent bipolar diagnosis[a]	FAD total score	FAD general functioning scale	FAD problem solving scale	FAD communication scale
Child bipolar disorder diagnosis	–						
Parent unipolar diagnosis	−0.01	–					
Parent bipolar diagnosis	0.42***	−0.09	–				
FAD total score	0.01	0.14*	0.15*	–			
FAD general functioning scale	0.04	0.14*	0.17**	0.95***	–		
FAD problem solving scale	0.00	0.17**	0.09	0.87***	0.78***	–	
FAD communication scale	−0.04	0.08	0.12	0.88***	0.73***	0.65***	–
CBQ	0.31***	0.09	0.03	0.42***	0.47***	0.37***	0.27***

FAD Family Assessment Device, *CBQ* Conflict Behavior Questionnaire
[a] Dummy coded, 1 = presence and 0 = absence of relevant diagnosis
*$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$

Table 4 Means for family functioning and conflict measures by Child DSM-IV diagnosis

Diagnosis	*N* (%)	FAD total score *M* (SD)	FAD general functioning scale *M (SD)*	FAD problem solving scale *M (SD)*	FAD communication scale *M (SD)*	CBQ *M (SD)*
Child bipolar spectrum diagnosis	150 (55%)					
Bipolar I	100 (37%)	57.9 (9.9)	25.8 (5.1)	13.2 (2.5)	18.9 (3.6)	13.8_a (4.5)
Bipolar II, cyclothymia, BP NOS	50 (18%)	57.0 (11.9)	24.8 (5.7)	13.3 (3.2)	18.9 (4.2)	11.8_a (5.5)
Child non-bipolar diagnosis	122 (45%)					
Unipolar Depression	63 (23%)	60.3_a (11.8)	26.0 (6.4)	13.8_a (3.0)	20.5_a (4.3)	11.8_a (6.0)
Disruptive behavior disorder and ADHD	28 (10%)	57.6 (7.2)	25.6 (3.7)	13.7_a (1.8)	18.3_b (2.9)	11.6_a (4.7)
No axis I diagnosis	31 (11%)	51.7_b (10.9)	22.7 (5.5)	11.7_b (2.8)	17.3_b (3.4)	2.4_b (4.2)
		$F(4,267)=3.27$*	$F(4,267)=3.37$*	$F(4,267)=3.30$*	$F(4,267)=4.14$**	$F(4,267)=30.61$***

Within columns, means with different subscripts are significantly different from each other only, not the rest of the column, at $p \leq 0.05$. *FAD* Family Assessment Device, *CBQ* Conflict Behavior Questionnaire
*$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$

ing being associated with child pathology in general, but not specifically linked to bipolar spectrum disorders.

Do Family Functioning and Conflict Mediate Links Between Mood Disorder in Parent and Child? (Hypothesis 3)

Path analysis was guided by the theoretically derived model that parental mood disorders would increase the risk of the child having bipolar disorder through both direct effects (including the transmission of a genetic diathesis; Goodwin and Jamison 1990) as well as indirectly by means of increasing family dysfunction. The models were estimated using AMOS 5.0 (Arbuckle and Wothke 1999), using maximum likelihood (ML) estimation. After entering a completely saturated model, the non-significant paths were dropped and the model was rerun to attain the best fitting model. The model including the Global FAD score was considered the primary model, and then separate exploratory models were tested for each of the FAD scales to explore whether there were differences in the role of communication or problem solving (both of which are targets of family focused interventions; Miklowitz et al. 2003a).

The hypothesized model provided an excellent fit with the data, χ^2 (2, N=272)=2.38, p=0.30, AGFI=0.97, CFI=1.0, RMSEA=0.026. A fitted model in which nonsignificant paths were omitted (dropping direct effects from parental mood diagnoses to conflict) was used as the basis for subsequent analyses[1]. This was done to decrease the type I error risk by reducing the number of paths tested in the later models exploring age and sex effects. Figure 1 presents the resulting final model. Substituting the other family functioning scales in the model resulted in equally

[1] Because AMOS does not allow for modeling of latent thresholds for bivariate outcome variables, analyses were also run in M-Plus 4.0, using both WLSMV, ML, and MLR estimators, to explicitly model the thresholds for categorical outcomes. These analyses did not result in any changes in the paths in terms of sign or significance; nor did the models appear to change in terms of fit. We also estimated the polyserial correlations using the "polyserial" procedure in R version 2.4.0 to examine whether modeling them instead of point-biserial estimates would change findings. The polyserial and point-biserial correlations were all well within one standard error of each other. The correlation between youth bipolar spectrum status and CBQ Total score was 0.42 for point-biserial and 0.38 for polyserial estimation (standard error=0.06). For youth bipolar status and FAD total score, both correlations were 0.01 (with a standard error of 0.08). Given the similarity in findings, the greater range of model comparison statistics available in AMOS versus M-Plus, and the greater congruence between the AMOS model and the current thinking about bipolar disorder as being a spectrum, not a categorical, phenomenon, we chose to present the AMOS results in the main body of the paper. Results of the M-Plus analyses are available upon request from the corresponding author.

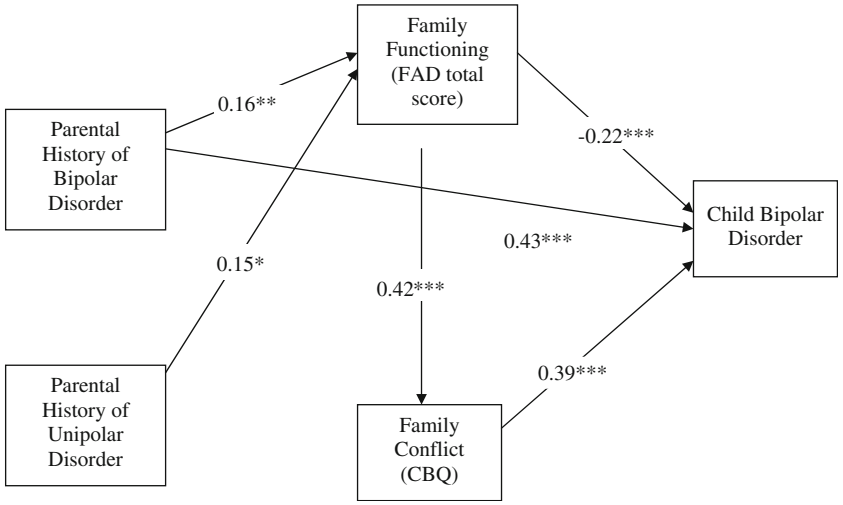

Fig. 1 Family functioning and conflict as potential mediators of parental mood disorders predicting child bipolar disorder. *p<0.05, **p<0.01, ***p<0.001, two-tailed

good fits. Both parental bipolar and unipolar disorders were indirectly related to child bipolar spectrum disorders through family processes. Both parental mood disorders were related to increased family functioning problems which, in turn, was associated with greater family conflict, and it was the increased family conflict that was positively associated with child diagnosis of bipolar spectrum disorders.

To determine the significance of mediation (Hypothesis 3), the indirect effect was calculated and tested for significance, using Sobel's (1982) test. This analysis sequence allowed us to examine the direct association between parental bipolar disorders and child bipolar disorders and allowed quantification of the extent to which family processes mediated this association. Tests of the product of path coefficients, such as the Sobel test, have better power and more accurate type I error rates than the popular "causal steps" approach described by Baron and Kenny (1986; see MacKinnon et al. 2002). Furthermore, tests of the product of coefficients are more appropriate in situations where path coefficients might have opposite signs, resulting in a suppression effect where mediating paths appear to cancel out the correlation between a more distal variable and the outcome of interest (MacKinnon et al. 2000).

The effects of parental mood disorder on family conflict were indirect through disruptions in family functioning. There were significant indirect effects between parental bipolar and unipolar disorder and family conflict through family functioning, based on the Sobel test (z=2.55, p<0.05 and z=2.46, p<0.05, respectively). Thus, compromised problem solving and communication were significant pathways between parental mood disorders and family conflict.

There was a significant indirect pathway in the link between family functioning and child bipolar spectrum disorders through family conflict (z=4.92, p<0.001), based on the Sobel test, but there was also a substantial direct effect; and the model including the direct effects yielded a significantly better fit than the model without the direct links, χ^2_{diff} (1, N=272)=15.19, p<0.001. Thus, family functioning added significantly to the prediction of child bipolar spectrum disorders and provided an indirect pathway in the relations between parental bipolar disorder and child bipolar disorders. Furthermore, family functioning had both direct and indirect effects in its prediction of child bipolar disorders. However, even though all of the path segments for the indirect effects achieved statistical significance, the cumulative indirect effects mediated by family functioning and conflict were small (−0.04 mediated via family functioning, and 0.03 via functioning and conflict) compared to the direct effect of parental bipolar disorders on youth bipolar disorders (β=0.43; see Fig. 1).

The direct link between impaired family functioning and child bipolar disorders was negative when also controlling for conflict levels such that more impaired family functioning was associated with children having other diagnoses besides bipolar spectrum. The zero-order correlations between the FAD scales and child bipolar disorders were all non-significant with rs ranging from 0.00 to 0.04 (see Table 3). Although families with pediatric bipolar disorders showed poorer family functioning than did families of youths with no axis I diagnoses, families with other diagnoses (i.e., youth unipolar depression, ADHD, or DBDs) showed equal or even worse levels of family functioning. The correlations in Table 3, and also the path analyses, all collapse these other youth diagnoses into the "nonbipolar" comparison group, testing what associations

might be unique to bipolar disorders. Present findings are consistent with prior literature finding equal (Zahn-Waxler et al. 1988) or stronger (Adrian and Hammen 1993) links between family processes and unipolar depression or other child diagnoses versus bipolar disorders. The significant negative pathway only appears when controlling for family conflict, which is highest in the context of pediatric bipolar disorders.

These findings suggest that after controlling for the level of conflict, additional effects of impaired communication or problem solving are more likely to be associated with other child diagnoses besides bipolar disorder. Consistent with this possibility, the pathway between family functioning and youth unipolar depression (instead of bipolar) was significant and positive (b=0.21, p<0.01). Similarly, if youth mood symptoms were used as the criterion, then after partialling out conflict levels and parental bipolar diagnosis, family functioning showed a positive relationship to CDRS-R depression scores (b=0.14, p<0.01) and a negative path to YMRS mania scores (b=−0.17, p<0.01). One explanation might be that dysfunctional family processes other than conflict make a stronger contribution to youth depression.

Overall, parent mood diagnoses accounted for 18% of variance in youth bipolar diagnoses, and family functioning and conflict combined to explain 13% of the variance (both sets significant p<0.0005). Because of the suppression effect in the regression equations, the variance estimates for the unique contributions of each set of predictors wound up being similar to the total variance estimates, and the part correlations increased slightly compared to the zero order correlations for conflict and for parent mood diagnoses.

Child Effect and Bidirectional Effects on Conflict An alternative hypothesis might be that the youth's bipolar disorder leads to greater conflict than otherwise would be expected based on the family's functioning, consistent with a "child effects model" (Lytton 1990). Two alternate models tested this possibility: A "child effects" model where the path was reversed so that youth bipolar disorders predicted increased conflict, and a "bidirectional" model where family conflict and youth bipolar disorders mutually amplified each other (a "nonrecursive" model). The "child effects" model fit the data poorly—χ^2 (2, N=272)=11.52, p<0.0005, and an ECVI of 0.138 versus.098 for the model in Fig. 1 (lower numbers indicating better fit). The bidirectional model did not provide improvement in fit over Fig. 1, χ^2_{diff} (1, N=272)=0.24, p>0.50; it also was less likely to cross-validate, with an ECVI of.104 versus 0.098. When paths in both directions were included, the effect of conflict on youth bipolar disorder remained large (b=0.44), whereas the effect of youth bipolar disorder on conflict was nonsignificant (b=−0.06).

Family Functioning and Comorbid Youth Diagnoses (Secondary Analyses)

Exploratory analyses evaluated the possibility that the high rate of comorbidity also might complicate results. Regression analyses addressing this question used dummy codes for youth diagnostic categories to predict family functioning variables after controlling for parent mood diagnoses (the second set of exploratory analyses). Dummy codes examined the unique effects of each youth diagnosis while simultaneously accounting for comorbid diagnoses, and also for the diverse mix of diagnoses in the sample. More than two thirds of the youths with bipolar diagnoses also met criteria for ADHD, and more than a third also met criteria for oppositional defiant disorder (ODD). Post hoc regression analyses used family functioning as the dependent variable, and dummy codes indicating the presence of bipolar disorder, ADHD, or ODD/conduct disorder as predictors, after controlling for parent mood status. Bipolar spectrum disorders made no unique contributions to family functioning after controlling for other youth diagnoses. Instead, child ODD was related to family functioning and conflict, after controlling for parent diagnoses and for other child diagnoses. Regression analyses indicated that child ODD was significantly related to the FAD general functioning scale, b=1.72, p=0.053 and the CBQ, b=2.74, p<0.01. Youth bipolar disorders remained significantly associated with CBQ scores even when controlling for all the other variables listed above, b=3.21, p<0.0005.

Moderating Effects of Child Age and Sex (Secondary Analyses, Sets Three and Four)

To explicitly test whether child age and sex moderated the relations among bipolar spectrum disorders in parents and children and family functioning and conflict (the third and fourth sets of secondary analyses), multiple-group comparisons were conducted for the path analyses, based on the best fitting model in the full sample. To test whether age moderated relationships hypothesized in our model we divided the sample into two groups: Younger children (ages 5–10) and older youths (ages 11–17). This resulted in 128 children in the younger group (85 with a bipolar spectrum diagnosis and 43 without) and 144 youths in the older group (65 with a bipolar spectrum diagnosis and 79 without). See Appendix for 2× 2×2 cross tabulations of the number of males and females in each age group by bipolar diagnosis. Based on the model presented in Fig. 1 (using the FAD total score), we found that the age-specific model fit better, χ^2_{diff} (6)=16.47, p<0.01. The effect on family functioning of both parental unipolar and parental bipolar diagnoses changed significantly depending upon the age of the youth. Parental diagnosis of bipolar disorder

predicted poorer family functioning for younger children (β=0.33, p<0.001), but not older children (β=0.02, p>0.05). Conversely, parental diagnosis of unipolar disorder affected family functioning for older youths (β=0.24, p<0.01), but not younger ones (β=0.04, p>0.05).

The final set of exploratory analyses examined possible interactions with child sex. There were 114 girls and 158 boys (see Appendix). The model fit equally well for girls and boys: χ^2_{diff} (6)=2.58, p>0.05, indicating that no sex differences were found in the model. Based on the sample size of N=272, with 43 participants in the smallest group tested for moderation effects (young children without a bipolar disorder), post hoc estimated power would be 0.85 to detect interaction effects with an f^2 of 0.032 or larger (comparable to a part correlation of 0.16 or larger), with alpha set at 0.05, two tailed (Erdfelder et al. 1996). Therefore, power was adequate for detecting both age or sex moderation effects.

Discussion

To date there has been little systematic study of relations between parental mood disorder, family functioning and conflict, and children's risk for disorder. This study examined the roles of family functioning and family conflict in the relations between parental diagnosis of bipolar and unipolar disorder and children's diagnosis of a bipolar spectrum disorder. Results indicated that there was a significant indirect pathway from parental bipolar and unipolar disorder to family conflict via impaired family functioning. Increased family conflict in turn was predictive of child bipolar disorder. This suggests that conflict is not entirely a direct result of parents having a mood disorder, but rather that it develops out of a negative family climate, including deficits in problem solving and communication. These data are consistent with previous work indicating that parental psychological problems are often associated with similar problems in children through the mechanisms of family processes (Cummings et al. 2000), particularly parental depression (Du Rocher Schudlich and Cummings 2003). Exploratory analyses indicated that poor problem solving and poor communication skills were both sources of risk for developing pediatric bipolar disorder through their links with increased family conflict, supporting their value as potential targets for treatment (Fristad and Goldberg-Arnold 2004; Miklowitz et al. 2003a). However, the indirect effects mediated by family processes were small compared to the remaining direct effect of parental bipolar diagnoses on youth bipolar diagnoses. Furthermore, "child effects" or bidirectional models where youth bipolar disorder predicted increased family conflict provided no improvement or even worse fit to the present data.

Family functioning appears to be a nonspecific correlate of child psychopathology. Family functioning was equally or more impaired in families where youths had unipolar depression, ADHD, or DBDs versus bipolar disorders. Consequently, family functioning was uncorrelated with bipolar versus nonbipolar youths (collapsing all other diagnostic categories together). The appearance of a negative pathway between family functioning and pediatric bipolar disorders after controlling for conflict levels was unexpected, and it partly reflects the high levels of disrupted family functioning associated with nonbipolar diagnoses such as unipolar depression and disruptive behavior disorders (Table 4).

At the same time, all diagnostic groups showed greater impairment than families whose children had no diagnosis. Disrupted family processes resulting from parental mood disorder may be a nonspecific mechanism leading to a variety of poor outcomes for youths, not just conveying increased risk for bipolar disorder (Chang et al. 2001; Hodgins et al. 2002; Radke-Yarrow 1998). In particular, children of parents with bipolar disorder may have higher rates of depression as well, which affects our comparisons of children with bipolar disorder to unaffected youth control groups (as opposed to youths with depression or externalizing problems). In the present data, family functioning showed a significant positive path to youth depressive symptoms (including depression in youths with bipolar diagnoses), whereas family functioning showed a negative path to manic symptoms after controlling for conflict levels, suggesting that family processes such as disrupted problem solving or poor communication might be more strongly linked to depression than mania (Miller et al. 1986).

Youth age moderated the effects of parental bipolar disorders on impaired family functioning. For adolescents, there was not a significant link from parental bipolar disorder to family functioning; whereas the path from parental diagnosis of unipolar disorder to family functioning was significant for older, but not younger, youths. Prior work with adolescents and young adults has found parental unipolar depression to be more disruptive to family functioning than parental bipolar diagnoses (Hammen et al. 1990). Present findings thus replicate prior work and suggest that there may be something particularly noxious about parental depressed mood in terms of impact on adolescents. Conversely, findings might indicate that parental mania is more disruptive to family functioning when children are younger. Exploratory analyses failed to find sex differences in the association between parental mood disorders, family functioning and conflict, and pediatric bipolar disorder.

Limitations of this study include that parent mental health diagnoses were sometimes based on indirect report

for fathers, and parental diagnoses were not systematically blinded to the diagnoses of the children. These concerns are tempered by the fact that diagnoses were not systematically informed by child diagnoses, and structured research interviews were used to derive almost all diagnoses. As with most covariance analyses, due to the correlational nature of the data and the likelihood of bi-directional effects, the direction of causality is ambiguous. Prospective, longitudinal data following a high-risk sample of parents-to-be (i.e. one or more parents with a mood disorder) from the prenatal period through child's adolescence and adulthood are necessary to assess the causal and interactive nature of the relationships between family functioning, conflict and bipolar disorder in parents and children. Given the interactive nature between parent and child depression and family conflict (Cummings et al. 2000), it is likely that bipolar spectrum disorders would operate similarly. Children who are at high risk for bipolar spectrum disorders due to one or more of their parents being diagnosed with a mood disorder may be exposed to heightened levels of conflict prior to developing symptoms and perhaps because of the child's predisposition, there may be an interaction between child attributes and parent attributes that exacerbates both the conflict and the child's disorder. For example, a recent study by Meyer et al. (2006) found that early exposure to maternal negativity increased risk for frontal lobe dysfunction in children which increased children's risk for bipolar disorder. There may also be age moderated effects in which conflict affects children's symptoms differently, with older children being more sensitive to more subtle aspects of conflict than younger children, but at the same time having developed more sophisticated coping strategies.

An additional limitation is the use of self-report measures for assessing family functioning and conflict in this study, rather than observational measures. Compared to behavioral observations, self-report measures are subject to memory and response biases, limit the specificity of dimensions of family responses that can be obtained, and prohibit assessment of nonverbal or other non-public responding (e.g. internal feelings or thoughts of others). Furthermore, more powerful measures of family functioning might increase the size of the correlations involving family process, and thus increase the size of the indirect pathways. This would lower the size of the direct effect of parent diagnosis on child diagnosis, because more of the covariance could be attributed to mediational processes. Another limitation is the exploratory nature of many of the analyses. Additionally, the observed suppressor effect involving family functioning and conflict with regard to youth bipolar status is likely to depend on whether the nonbipolar comparison group is limited to healthy controls (who will show better average family functioning) versus including high rates of other diagnoses also linked to poor family functioning. Family functioning's relations with global functioning and with other child problems, such as disruptive disorders, were in predicted directions, supporting the hypothesis that family functioning is an important indirect effect to consider, albeit not unique to bipolar disorder.

Finally, other factors not examined in this study may be important in explaining the link between parent and child bipolar disorder. For example, parenting, marital relations and children's coping mechanisms have all been found to be important mediators in explaining links between parental depression and child adjustment problems (Cummings et al. 2000; Du Rocher Schudlich 2004). The present study did not include direct measures of important constructs such as expressed emotion or parent-child attachment (e.g., Hammen et al. 1990). These more proximal family variables may also be explanatory factors in families with bipolar disorder for both adults (Hooley and Hiller 2001) as well as youths (Geller et al. 2003; Miklowitz et al. 2003b).

Despite these limitations, these findings lay the groundwork for future testing of causal pathways linking parental bipolar disorder, family functioning and conflict, and children's bipolar disorder and hold important implications for clinicians treating families with bipolar disorder. Although family factors do not fully account for the links between parent and child bipolar disorder, they contribute to risk of a broad range of additional problems. Family processes are also potential targets for intervention, offering potential mechanisms by which interventions could reduce the risk of mood episodes in children. In fact, interventions that target problem-solving and communication skills for parents and children have been found to be successful in terms of symptom reduction in children (Feeny et al. 2006; Fristad 1999; Fristad et al. 2002, 2003; Miklowitz et al. 2003a). Given the impaired family functioning and consequent family conflict in families with parental bipolar disorder, improving the coping skills of children in these families may also be beneficial in reducing their risk for future disorder.

Acknowledgement This work was supported in part by a Stanley Medical Research Institute clinical research center grant as well as NIMH R01 MH066647.

Disclosures Robert L. Findling receives or has received research support, acted as a consultant and/or served on a speaker's bureau for Abbott, AstraZeneca, Bristol-Myers Squibb, Celltech-Medeva, Cypress Biosciences, Forest, GlaxoSmithKline, Johnson & Johnson, Lilly, New River, Novartis, Otsuka, Pfizer, Sanofi-Aventis, Sepracore, Shire, Solvay, Supernus Pharmaceuticals, and Wyeth.

Appendix

Table 5 Child age by sex by bipolar diagnosis

Child sex	Bipolar diagnosis status	Child age group		
		Younger	Older	Total
Male	No Bipolar	20	40	60
	Bipolar	59	39	98
Total		79	79	158
Female	No Bipolar	23	39	62
	Bipolar	26	26	52
Total		49	65	114

References

American Psychiatric Association (2001). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Adrian, C., & Hammen, C. (1993). Stress exposure and stress generation in children of depressed mothers. *Journal of Consulting and Clinical Psychology*, *61*, 354–359.

Anderson, C. A., & Hammen, C. L. (1993). Psychosocial outcomes of children of unipolar depressed, bipolar, medically ill, and normal women: A longitudinal study. *Journal of Consulting and Clinical Psychology*, *61*, 448–454.

Andreasen, N. C., Endicott, J., Spitzer, R. L., & Winokur, G. (1977). The family history method using diagnostic criteria. Reliability and validity. *Archives of General Psychiatry*, *34*, 1229–1235.

Arbuckle, J. L., & Wothke, W. (1999). *Amos 4.0 users guide*. Chicago: SmallWaters Corporation.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182.

Berument, S. K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: diagnostic validity. *British Journal of Psychiatry*, *175*, 444–451.

Biederman, J., Kwon, A., Wozniak, J., Mick, E., Markowitz, S., Fazio, V., et al. (2004). Absence of gender differences in pediatric bipolar disorder: Findings from a large sample of referred youth. *Journal of Affective Disorders*, *83*, 207–214.

Bossuyt, P. M., Reitsma, J. B., Bruns, D. E., Gatsonis, C. A., Glasziou, P. P., Irwig, L. M., et al. (2003). Towards complete and accurate reporting of studies of diagnostic accuracy: the STARD initiative. *British Medical Journal*, *326*, 41–44.

Byles, J., Byrne, C., Boyle, M. H., & Offord, D. R. (1988). Ontario Child Health Study: Reliability and validity of the general functioning subscale of the McMaster Family Assessment Device. *Family Process*, *27*, 97–104.

Caplan, H., Cogill, S., Alexandra, H., Robson, K., Katz, R., & Kumar, R. (1989). Maternal depression and the emotional development of the child. *British Journal of Psychiatry*, *154*, 818–823.

Chang, K. D., Blasey, C., Ketter, T. A., & Steiner, H. (2001). Family environment of children and adolescents with bipolar parents. *Bipolar Disorders*, *3*, 73–78.

Conrad, M., & Hammen, C. (1989). Role of maternal depression in perceptions of child maladjustment. *Journal of Consulting and Clinical Psychology*, *57*, 663–667.

Cooke, R. G., Young, L. T., Mohri, L., Blake, P., & Joffe, R. T. (1999). Family-of-origin characteristics in bipolar disorder: A controlled study. *Canadian Journal of Psychiatry*, *44*, 379–381.

Cummings, E. M., & Davies, P. T. (1994). Maternal depression and child development. *Journal of Child Psychology and Psychiatry*, *35*, 73–112.

Cummings, E. M., Davies, P. T., & Campbell, S. B. (2000). *Developmental psychopathology and family process: Theory, research, and clinical implications*. New York: Guilford.

Danielson, C. K., Feeny, N. C., Findling, R. L., & Youngstrom, E. A. (2004). Psychosocial treatment of bipolar disorders in adolescents: A proposed cognitive-behavioral intervention. *Cognitive & Behavioral Practice*, *11*, 283–297.

Davis, B., Sheeber, L., Hops, H., & Tildesley, E. (2000). Adolescent responses to depressive parental behaviors in problems-solving interactions: Implications for depressive symptoms. *Journal of Abnormal Child Psychology*, *28*, 451–465.

DelBello, M. P., & Geller, B. (2001). Review of studies of child and adolescent offspring of bipolar parents. *Bipolar Disorders*, *3*, 325–334.

Downey, G., & Coyne, J. C. (1990). Children of depressed parents: An integrative review. *Psychological Bulletin*, *108*, 50–76.

Du Rocher Schudlich, T. D. (2004). Parental dysphoria, marital conflict, and parenting:Relations with children's emotional security and adjustment. Unpublished doctoral dissertation.

Du Rocher Schudlich, T. D., & Cummings, E. M. (2003). Parental dysphoria and children's internalizing symptoms: Marital conflict styles as mediators of risk. *Child Development*, *74*, 1663–1681.

Du Rocher Schudlich, T. D., Papp, L. M., & Cummings, E. M. (2004). Relations of husbands' and wives' dysphoria to marital conflict resolution strategies. *Journal of Family Psychology*, *18*, 171–183.

Dunn, L. M., & Dunn, L. M. (1997). *Examiner's manual for the peabody picture vocabulary test – third edition*. Circle Pines, MN: American Guidance Service.

Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview: The schedule for affective disorders and schizophrenia. *Archives of General Psychiatry*, *35*, 837–844.

Epstein, N. B., Baldwin, L. M., & Bishop, D. S. (1983). The McMaster Family Assessment Device. *Journal of Marital and Family Therapy*, *9*, 171–180.

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPower: A general power analysis program. *Behavior Research Methods, Instruments & Computers*, *28*, 1–11.

Feeny, N. C., Danielson, C., Schwartz, L., Youngstrom, E. A., & Findling, R. L. (2006). CBT for bipolar disorders in adolescents: A pilot study. *Bipolar Disorders*, *8*, 508–515.

Fergusson, D., Horwood, L. J., & Lynskey, M. (1995). Maternal depressive symptoms and depressive symptoms in adolescents. *Journal of Child Psychology and Psychiatry*, *36*, 1161–1178.

Findling, R. L., Gracious, B. L., McNamara, N. K., Youngstrom, E. A., Demeter, C., & Calabrese, J. R. (2001). Rapid, continuous cycling and psychiatric co-morbidity in pediatric bipolar I disorder. *Bipolar Disorders*, *3*, 202–210.

Forehand, R., & Jones, D. J. (2003). Neighborhood violence and coparent conflict: Interactive influence on child psychosocial adjustment. *Journal of Abnormal Child Psychology*, *31*, 591–604.

Forehand, R., & McCombs, A. (1988). Unraveling the antecedent-consequence conditions in maternal depression and adolescent functioning. *Behaviour Research and Therapy*, *26*, 399–405.

Fristad, M. A. (1999). Psychoeducation: A model intervention for families with mood disordered children. *The Family Psychologist*, *15*, 14–18.

Fristad, M. A., Gavazzi, S. M., & Mackinaw-Koons, B. (2003). Family psychoeducation: An adjunctive intervention for children with bipolar disorder. *Biological Psychiatry*, *53*, 1000–1008.

Fristad, M. A., & Goldberg-Arnold, J. S. (2004). *Raising a moody child: How to cope with depression and bipolar disorder*. New York: Guilford.

Fristad, M. A., Goldberg-Arnold, J. S., & Gavazzi, S. M. (2002). Multifamily psychoeducation groups (MFPG) for families of children with bipolar disorder. *Bipolar Disorders*, *4*, 254–262.

Fristad, M. A., Weller, E. B., & Weller, R. A. (1992). The mania rating scale: Can it be used in children? A preliminary report. *Journal of the American Academy of Child & Adolescent Psychiatry*, *31*, 252–257.

Ge, X., Conger, R. D., Lorenz, F. O., Shanahan, M., Elder, G., et al. (1995). Mutual influences in parent and adolescent psychological distress. *Developmental Psychology*, *31*, 406–419.

Geller, B., Craney, J. L., Bolhofner, K., DelBello, M. P., Axelson, D., Luby, J., et al. (2003). Phenomenology and longitudinal course of children with a prepubertal and early adolescent bipolar disorder phenotype. In B. Geller, & M. P. DelBello (Eds.) *Bipolar disorder in childhood and early adolescence* (pp. 25–50). New York: Guilford.

Geller, B., & Luby, J. (1997). Child and adolescent bipolar disorder: A review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 1168–1176.

Geller, B., Tillman, R., Craney, J. L., & Bolhofner, K. (2004). Four-year prospective outcome and natural history of mania in children with a prepubertal and early adolescent bipolar disorder phenotype. *Archives of General Psychiatry*, *61*, 459–467.

Goodwin, F. K., & Jamison, K. R. (1990). *Manic-depressive illness*. New York: Oxford University Press.

Hammen, C. (1988). Self-cognitions, stressful events, and the prediction of depression in children of depressed mothers. *Journal of Abnormal Child Psychology*, *16*, 347–360.

Hammen, C. (1991). *Depression runs in families: The social context of risk and resilience in children of depressed mothers*. New York: Springer.

Hammen, C. (2002). Context of stress in families of children with depressed parents. In S. H. Goodman, & I. H. Gotlib (Eds.) *Children of depressed parents* (pp. 175–202). Washington, DC: American Psychological Association.

Hammen, C., Adrian, C., Gordon, D., Burge, D., Jaenicke, C., & Hiroto, D. (1987). Children of depressed mothers: Maternal strain and symptom predictors of dysfunction. *Journal of Abnormal Psychology*, *96*, 190–198.

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., Burge, D., Burney, E., & Adrian, C. (1990). Longitudinal study of diagnoses in children of women with unipolar and bipolar affective disorder. *Archives of General Psychiatry*, *47*, 1112–1117.

Hellander, M., Sisson, D. P., & Fristad, M. (2003). Internet support for parents of children with early-onset bipolar disorder. In B. Geller, & M. P. DelBello (Eds.) *Bipolar disorder in childhood and early adolescence* (pp. 314–329). New York: Guilford.

Hodgins, S., Faucher, B., Zarac, A., & Ellenbogen, M. (2002). Children of parents with bipolar disorder. A population at high risk for major affective disorders. *Child & Adolescent Psychiatric Clinics of North America*, *11*, 533–553.

Hooley, J. M., & Hiller, J. B. (2001). Family relationships and major mental disorder: Risk factors and preventive strategies. In B. R. Sarason, & S. Duck (Eds.) *Personal relationships: Implications for clinical and community psychology* (pp. 61–87). New York: Wiley.

Judd, L. L., Akiskal, H. S., Schettler, P. J., Endicott, J., Leon, A. C., Solomon, D., et al. (2005). Psychosocial disability in the course of bipolar I and II disorders: A prospective, comparative, longitudinal study. *Archives of General Psychiatry*, *62*, 1322–1330.

Keitner, G. I., Baldwin, L. M., Epstein, N. B., & Bishop, D. S. (1985). Family functioning in patients with affective disorders. *International Journal of Family Psychiatry*, *4*, 405–437.

Kim, S., & Brody, G. H. (2005). Longitudinal pathways to psychological adjustment among black youth living in single-parent households. *Journal of Family Psychology*, *19*, 305–313.

Kirk, R. E. (1995). *Experimental design: Procedures for the behavioral sciences* (3rd ed.). New York: Brooks/Cole Publishing.

Kotchick, B. A., Dorsey, S., & Heller, L. (2005). Predictors of parenting among African American single mothers: Personal and contextual factors. *Journal of Marriage and Family*, *67*, 448–460.

Kowatch, R. A., Youngstrom, E. A., Danielyan, A. K., & Findling, R. L. (2005). Meta-analysis of the phenomenology and clinical characteristics of mania in children and adolescents. *Bipolar Disorders*, *7*, 483–496.

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*, 623–631.

Leibenluft, E., Charney, D. S., Towbin, K. E., Bhangoo, R. K., & Pine, D. S. (2003). Defining clinical phenotypes of juvenile mania. *American Journal of Psychiatry*, *160*, 430–437.

Lytton, H. (1990). Child and parent effects in boys' conduct disorder: A reinterpretation. *Developmental Psychology*, *26*, 683–697.

MacKinnon, D. P., Krull, J. L., & Lockwood, C. M. (2000). Equivalence of the mediation, confounding and suppression effect. *Prevention Science*, *1*, 173–181.

MacKinnon, D. P., Lockwood, C. M., Hoffman, J. M., West, S. G., & Sheets, V. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods*, *7*, 83–104.

McGuffin, P., Rijsdijk, F., Andrew, M., Sham, P., Katz, R., & Cardno, A. (2003). The heritability of bipolar affective disorder and the genetic relationship to unipolar depression. *Archives of General Psychiatry*, *60*, 497–502.

Meyer, S. E., Carlson, G. E., Wiggs, C. E., Ronsaville, D. S., Martinex, P. E., Klimes-Dougan, B., Gold, P. W., & Radke-Yarrow, M. (2006). A prospective high-risk study of the association among maternal negativity, apparent frontal lobe dysfunction, and the development of bipolar disorder. *Development and Psychopathology*, *18*, 573–289.

Miklowitz, D. J. (2004). The role of family systems in severe and recurrent psychiatric disorders: a developmental psychopathology view. *Development and Psychopathology*, *16*, 667–688.

Miklowitz, D. J., George, E. L., Richards, J. A., Simoneau, T. L., & Suddath, R. L. (2003a). A randomized study of family focused psychoeducation and pharmacotherapy in the outpatient management of bipolar disorder. *Archives of General Psychiatry*, *60*, 904–912.

Miklowitz, D. J., Richards, J. A., George, E. L., Frank, E., Suddath, R. L., Powell, K. B., et al. (2003b). Integrated family and individual therapy for bipolar disorder: Results of a treatment development study. *The Journal of Clinical Psychiatry*, *64*, 182–191.

Miklowitz, D. J., Wisniewski, S. R., Miyahara, S., Otto, M. W., & Sachs, G. S. (2005). Perceived criticism from family members as a predictor of the one-year course of bipolar disorder. *Psychiatry Research*, *136*, 101–111.

Miller, I. W., Kabacoff, R. I., Keitner, G. I., Epstein, N. B., & Bishop, D. S. (1986). Family functioning in families of psychiatric patients. *Comprehensive Psychiatry*, *27*, 302–312.

Murray, L., Sinclair, D., Cooper, P., Ducournau, P., & Turner, P. (1999). The socioemotional development of 5 year old children of postnatally depressed mothers. *Journal of Child Psychology and Psychiatry*, *40*, 1259–1271.

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. Children and their development: Knowledge base, research agenda, and social policy application. *American Psychologist*, *44*(Special issue), 329–335.

Perlis, R., Miyahara, S., Marangell, L. B., Wisniewski, S. R., Ostacher, M., DelBello, M. P., et al. (2004). Long-term implications of early onset in bipolar disorder: Data from the first 1000 participants in the systematic treatment enhancement program for bipolar disorder (STEP-BD). *Biological Psychiatry*, *55*, 875–881.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth & Adolescence*, *17*, 117–133.

Poznanski, E. O., Miller, E., Salguero, C., & Kelsh, R. C. (1984). Preliminary studies of the reliability and validity of the Children's Depression Rating Scale. *Journal of the American Academy of Child Psychiatry*, *23*, 191–197.

Prinz, R. J., Foster, S. L., Kent, R. N., & O'Leary, K. D. (1979). Multivariate assessment of conflict in distressed and nondistressed mother-adolescent dyads. *Journal of Applied Behavior Analysis*, *12*, 691–700.

Radke-Yarrow, M. (1998). *Children of depressed mothers: From early childhood to maturity*. New York: Cambridge Press.

Rice, F., Harold, G., & Thapar, A. (2002). Negative life events as an account of age-related differences in the genetic aetiology of depression in childhood and adolescence. *Journal of Child Psychology and Psychiatry*, *44*, 977–987.

Robertson, H. A., Kuthcer, S. P., Bird, D., & Grasswick, L. (2001). Impact of early onset bipolar disorder on family functioning: Adolescents' perceptions of family dynamics, communication, and problems. *Journal of Affective Disorders*, *66*, 25–37.

Robin, A. L., & Foster, S. L. (1989). *Negotiating parent–adolescent conflict: A behavioral-family systems approach*. New York: Guilford.

Sanford, M., Boyle, M., McCleary, L., Miller, J., Steele, M., Duke, E., & Offord, D. (2006). A pilot study of adjunctive family psychoeducation in adolescent major depression: Feasibility and treatment effect. *Journal of the American Academy of Child & Adolescent Psychiatry*, *45*, 386–395.

Schwoeri, L., & Sholevar, G. P. (1994). The family transmission of depression. In G. P. Sholevar (Ed.) *The transmission of depression in families and children: Assessment and Intervention* (pp. 123–144). Northvale, NJ: Jason Aronson.

Singh, M. K., Delbello, M. P., Stanford, K. E., Soutullo, C., McDonough-Ryan, P., McElroy, S. L., & Strakowski, S. M. (2007). Psychopathology in children of bipolar parents. *Journal of Affective Disorders*, *102*, 131–136.

Snyder, J. R. (1998). Marital conflict and child adjustment: What about gender? *Developmental Review*, *18*, 390–420.

Sobel, M. E. (1982). Asymptotic confidence intervals for indirect effects in structural equation models.In *Sociological Methodology 1982* pp. 290–312. Washington, DC: American Sociological Association.

Young, R. C., Biggs, J. T., Ziegler, V. E., & Meyer, D. A. (1978). A rating scale for mania: Reliability, validity, and sensitivity. *British Journal of Psychiatry*, *133*, 429–435.

Youngstrom, E. A., & Kogos Youngstrom, J. (2005). Evidence based assessment of pediatric bipolar disorder, part 2: Incorporating information from behavior checklists. *Journal of the American Academy of Child & Adolescent Psychiatry*, *44*, 823–828.

Youngstrom, E. A., Findling, R. L., Danielson, C. K., & Calabrese, J. R. (2001). Discriminative validity of parent report of hypomanic and depressive symptoms on the General Behavior Inventory. *Psychological Assessment*, *13*, 267–276.

Youngstrom, E. A., Findling, R. L., Calabrese, J. R., Gracious, B. L., Demeter, C., Bedoya, B. D., et al. (2004). Comparing the diagnostic accuracy of six potential screening instruments for bipolar disorder in youths aged 5 to 17 years. *Journal of the American Academy of Child & Adolescent Psychiatry*, *43*, 847–858.

Zahn-Waxler, C., Mayfield, A., Radke-Yarrow, M., McKnew, D. H., Cytryn, L., & Davenport, Y. B. (1988). A follow-up investigation of offspring of parents with bipolar disorder. *American Journal of Psychiatry*, *145*, 506–509.

Zwaanswijk, M., van der Ende, J., Verhaak, P. F., Bensing, J., & Verhulst, F. (2005). Help-seeking for child psychopathology: Pathways to informal and professional services in The Netherlands. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*, 1292–1300.