

## Original Article

# Family environment of children and adolescents with bipolar parents

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**Objectives:** The effect of family environment on the development of bipolar disorder (BD) in children is not known. We sought to characterize families with children at high risk for developing BD in order to better understand the contributions of family environment to the development of childhood BD.

**Methods:** We collected demographic data and parental ratings on the Family Environment Scale (FES) for 56 children (aged 6–18 years) from 36 families with at least one biological parent with BD. The cohort had previously been psychiatrically diagnosed according to semi-structured interviews.

**Results:** Statistical comparisons with normative data indicated that parents' ratings were significantly lower on the FES Cohesion and Organization scales and were significantly higher on the FES Conflict scale. Multivariate analyses of variance indicated that families with both parents having a mood disorder had no significantly different FES scores than families with only one parent with a mood disorder (BD). Diagnostic data indicated that while 54% of the children in the sample had an Axis I disorder and 14% had BD, FES scores did not differ significantly for subjects with or without an Axis I disorder, or with or without BD.

**Conclusions:** Families with a bipolar parent differ from the average family in having less cohesion and organization, and more conflict. Despite this difference, it does not appear that the environment alone of families with a bipolar parent determines the outcome of psychopathology in the children, or that the psychopathology of the children determines the family environment.

**Kiki D Chang<sup>a</sup>,  
Christine Blasey<sup>b</sup>, Terence  
A Ketter<sup>a</sup> and Hans Steiner<sup>a</sup>**

<sup>a</sup> Department of Psychiatry and Behavioral Sciences, <sup>b</sup> Division of Child and Adolescent Psychiatry, Stanford University School of Medicine, Stanford, CA, USA

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Corresponding author: Kiki D Chang, MD, Stanford University School of Medicine, Division of Child and Adolescent Psychiatry, 401 Quarry Road, Stanford, CA 94305-5540, USA. Tel: +1 650 725 0956; fax: +1 650 723 5531; e-mail: kchang88@stanford.edu; http://www.stanford.edu/group/bipolar.clinic/index.html

Children of parents with bipolar disorder (BD) may be at four times greater risk for developing mood disorders compared to children of parents without psychiatric disorders (1). The familial transmission of BD has been well established by pedigree analyses and twin studies (2). As the concordance rate in identical twins does not approach 100%, it has been proposed that BD develops in a child with genetic predisposition in response to external stressors (3). One formative entity in a child's development is the family environment, which may provide both protective factors, as well as act as an external stressor.

There is substantial evidence that having a psy-

chiatrically ill parent will increase a child's chances of having psychopathology (4–7). Aside from conferring genetic risk, the presence of a bipolar parent who intermittently may become psychotic, dysfunctional, neglectful, or absent must be considered a powerful influence on a child's development. The expressed emotion literature in schizophrenia also shows the important contribution familial influences have on disease progression and regression (8–10), and it is possible that a similar process will be found to be important in BD. However, it would be necessary to first describe the general characteristics of families with a bipolar parent with a broader measure.

Families having a member with BD have been postulated in uncontrolled studies to have abnormal characteristics that may have helped to cause the BD in the affected individual. Psychodynamic theorizations have been made without adequate study to prove these theories (11). A recent study using the Family Environment Scale (FES) (12) found no differences between FES scores of families with and without a bipolar member (13). Negative family interactions, including higher expressed emotion, have reported to be predictive of *relapse* in bipolar adolescents and adults (14), but no studies have been conducted in families with a bipolar parent to ascertain the family environment effect on psychopathology of offspring. Previously, we reported on a cohort of child and adolescent bipolar offspring, in which we found 52% to already have a psychiatric disorder by DSM-IV criteria (15). Here, we report on the family environments of these offspring and the relationship of their family environments on their current distinct psychopathology.

## Methods

### Design

Fifty-six subjects were recruited from parents who were patients at the Stanford University Bipolar Disorders Clinic, from local support groups for bipolar adults, and from the surrounding community. Subjects were enrolled from 36 different families, between June 1997 and August 1998, were between 6 and 18 years old, and had at least one biological parent with bipolar I or II disorder. Attempts were made to include all offspring from each bipolar parent. One child was unable to participate in the evaluation due to mental retardation and was excluded from the study.

### Assessment and diagnosis

After written informed consent was obtained, parents were interviewed by a board-certified psychiatrist and diagnosed according to the Diagnostic Statistical Manual, IV (DSM-IV) (16) criteria. Offspring of bipolar parents were evaluated by the affective disorders module of the Washington University Schedule for Affective Disorders and Schizophrenia in Children and Adolescents (WASH-U-K-SADS) (17) and the K-SADS-PL (18). Subjects were evaluated either by a child psychiatrist or a trained research assistant, who were both non-blinded to parental status. Interrater 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. (17). Diagnostic decisions were ultimately made by a child psychiatrist based on personal interview, discussion with the research assistant, and on written notes of parental and subject responses to individual WASH-U-K-SADS questions. Diagnoses were made according to DSM-IV criteria. Each patient was assigned a Global Assessment of Functioning (GAF) score by the interviewer to quantify overall social and academic functioning.

Parents were asked to complete the FES, a 90-item, true/false questionnaire evaluating the family in ten different categories: cohesion (C), expressiveness (EX), conflict (CON), independence (IND), achievement orientation (AO), intellectual-cultural orientation (ICO), active-recreational orientation (ARO), moral-religious emphasis (MRE), organization (ORG), and control (CTL). In two-parent families, both parents were asked to fill out one FES form together as best as possible. Each child within one family received the same FES score. Parents were asked to base responses on general family environment, not pertaining to only that particular day or week.

Normative FES data have been collected and reported on by Moos and Moos (19). The normative sample consists of 1432 families from all over the USA, of diverse makeup (single parent, multigenerational, different ages), and varying ethnicity. The prevalence of psychiatric disorders in this group is not known, as it was meant to be a normative, not healthy control, sample. The FES has been found to have good internal consistency (average Cronbach Alpha = 0.71) and good test-retest reliability (19). Furthermore, the FES has been shown to have construct and discriminant validity (20–22).

### Cohort

Fifty-four percent of the children had an Axis I disorder. Fourteen percent had BD (defined as bipolar I, II, or cyclothymic disorder). We divided the subjects into two categories based on theoretical risk for developing BD: the unilineal group, or high risk group, consisted of children and adolescents with only one parent with a mood disorder, that being BD. The bilineal group, the very high risk category, consisted of offspring of one bipolar parent and the other parent with either major depressive disorder or BD. There were 11 families (18 children) in the bilineal group and 25 families (38 children) in the unilineal group. Demographic information collected included age, gender, parental occupation and level of education, house-

hold income, ethnic status, number of siblings, and parental age of illness onset. Socioeconomic status (SES) was assessed using Hollingshead's Two Factor Index of Social Position, from the gathered demographic information.

Statistical methods

Data analysis was performed in two stages. First, mean FES scores were statistically compared to the normative FES data using single sample Z-score test statistics. Correlational analyses were used to measure relationships between demographic variables and FES scale scores. Within-group analyses were conducted using three multivariate analyses of variance (MANOVA) models in order to compare the FES scores of subgroups of families: bilineal versus unilineal risk, families with at least one BD child versus no children with BD, and families with one or more children diagnosed with an Axis I disorder versus no Axis I disorder. All data met the appropriate assumptions of multivariate normality, linearity, and homogeneity of variance. An alpha of 0.05 was used as the threshold for statistical significance for the three omnibus F-tests.

Results

Demographics

The demographic data for the entire cohort and for individuals within cohort groups are presented in Table 1. There were no significant differences in SES or age among the subgroups. Subjects having

an Axis I disorder were more predominantly male than subjects not having an Axis I disorder. No other differences in gender were found among the subgroups.

Correlational analyses

Spearman's rho correlation coefficients indicated that the average age of children, number of children diagnosed with BD, and number of children diagnosed with any Axis I disorder were not significantly related to FES scale scores. SES, as measured by the Hollingshead, was not significantly correlated with any FES scales. The number of children was unrelated to FES scores, with the exception of the conflict scale: families with more children had higher reported levels of conflict ( $r = 0.35, p = 0.036$ ).

Comparison of sample FES scores with FES normative data

The Bipolar Offspring sample ( $n = 36$ ) obtained average scores that were significantly different from normative data on 8 of the 10 FES scales. The most robust differences ( $p \leq 0.001$ ) were indicated by the bipolar offspring's increased mean conflict scores ( $Z = + 5.81, p = 0.000$ ), lower cohesion ( $Z = - 4.69, p = 0.000$ ), and lower organization scores ( $Z = - 4.20, p = 0.000$ ). Further, sample mean scores were significantly lower ( $p \leq 0.01$ ) on the independence ( $Z = - 3.14, p = 0.002$ ), and achievement orientation scales ( $Z = - 2.78,$

Table 1. Demographic characteristics of bipolar offspring

	Entire sample (n = 56)	Bipolar disorder (n = 9)	No Bipolar disorder (n = 47)	Axis I disorder (n = 29)	No Axis I disorder (n = 27)	Bilineal (n = 18)	Unilineal (n = 38)
<b>Age</b> —years Mean (SD)	10.4 (3.1)	10.6 (2.6)	10.4 (3.2)	10.9 (2.8)	9.9 (3.4)	10.2 (3.5)	10.6 (2.9)
<b>Gender</b> n (%)							
Female	26 (46)	2 (22)	24 (51)	10 (34)	16 (59)	6 (33)	20 (53)
Male	30 (54)	7 (78)	23 (49)	19 (66)	11 (41)	12 (67)	18 (47)
<b>Ethnicity</b> n (%)							
Asian	5 (9)	0	5 (11)	2 (7)	3 (11)	0	5 (13)
African-American	1 (2)	0	1 (2)	0	1 (4)	1 (6)	0
Caucasian	50 (89)	9 (100)	41 (87)	27 (93)	23 (85)	17 (94)	33 (87)
<b>SES</b> Hollingshead (%)							
I	9 (16)	1 (11)	8 (17)	5 (17)	4 (15)	0	9 (24)
II	12 (21)	3 (33)	9 (19)	1 (24)	5 (18)	5 (28)	7 (18)
III	28 (50)	5 (56)	23 (49)	14 (48)	14 (52)	7 (39)	21 (55)
IV	3 (5)	0	3 (6)	0	3 (11)	2 (11)	1 (3)
V	0	0	0	0	0	0	0
Unknown	4 (7)	0	0	3 (10)	1 (4)	4 (22)	0

SES = Socioeconomic status.

Table 2. Comparisons of mean FES scores of bipolar families (n = 36) to population means

	Bipolar families (n = 36) (SD)	Population means (SD)	Single sample Z score	p
Cohesion	5.58 (2.51)	6.73 (1.47)	-4.69	<0.001
Expressiveness	5.44 (1.58)	5.54 (1.61)	-0.37	0.71
Conflict	5.03 (2.37)	3.18 (1.91)	5.81	<0.001
Independence	6.00 (1.64)	6.66 (1.26)	-3.14	0.002
Achievement orientation	4.72 (2.01)	5.47 (1.62)	-2.78	0.005
IC orientation	6.19 (1.90)	5.56 (1.82)	2.08	0.04
AR orientation	4.92 (2.35)	5.33 (1.96)	-1.26	0.20
Moral-religious emphasis	5.36 (2.46)	4.75 (2.03)	1.8	0.07
Organization	4.14 (2.26)	5.47 (1.90)	-4.20	<0.001
Control	5.03 (1.73)	4.26 (1.84)	2.51	0.01

IC = intellectual-cultural; AR = active-recreational.

$p = 0.005$ ), and higher on scores of control ( $Z = +2.51$ ,  $p = 0.01$ ). The sample's mean score on the intellectual-cultural orientation scale was also higher than normal ( $Z = +2.08$ ,  $p = 0.04$ ). While the bipolar offspring obtained higher than normal scores on moral-religious emphasis, the difference did not reach statistical significance ( $Z = +1.8$ ,  $p = 0.07$ ). On the expressiveness and active-recreational orientation scales, the bipolar offspring did not score differently from the normative populations ( $Z = -0.37$ ,  $p = 0.71$ ;  $Z = -1.26$ ,  $p = 0.20$ , respectively). Means, standard deviations, and statistical comparisons are provided in Table 2.

Within-sample comparison of FES scores: families of children with an Axis I diagnosis versus no Axis I diagnosis

A one-way MANOVA model, with a single factor of group (i.e. presence/absence of children with an Axis I diagnosis), was conducted for the 10 FES scales. The Wilks lambda indicated no significant main effect for presence of an Axis I disorder (Wilks = 0.92, Omnibus  $F = 0.34$ ,  $p = 0.93$ ). Thus, families with offspring diagnosed with an Axis I disorder ( $n = 23$ ) did not differ in mean FES scores from families without offspring diagnosed with an Axis I disorder ( $n = 13$ ).

Within-sample comparison of FES scores: bilineal versus unilineal risk for BD

A similar MANOVA model was conducted to see if bipolar offspring who had a unilineal risk for BD ( $n = 25$ ) had a unique profile of variation in FES scores compared to those with a bilineal risk ( $n = 11$ ). For this multivariate model, no significant effect was observed for the group factor (Wilks = 0.821, Omnibus  $F = 0.54$ ,  $p = 0.84$ ).

Within-sample comparison of FES scores: offspring with BD versus no BD

A third MANOVA model was conducted to see if families with bipolar offspring who had a diagnosis of BD ( $n = 9$ ) had a unique profile of variation in FES scores compared to families with no children diagnosed with BD ( $n = 27$ ). For this model, there was no detectable main effect for group (Wilks = 0.95, Omnibus  $F = 0.20$ ,  $p = 0.98$ ).

## Discussion

This is the first study to examine the relationship of general family environment to child psychopathology in children at high risk for developing BD. We have found families with a bipolar parent to report significant differences in their family environments compared to population means. Families having a bipolar parent may differ from the average family in having less cohesion and organization, and more conflict and control. Bipolar families also reported less independence and achievement orientation, but more intellectual-cultural orientation than population means. Families with the other parent having a mood disorder, as well, may have further problems with decreased fostering of independence. However, families of bipolar offspring already with psychiatric disorders did not differ from families of currently diagnoses-free bipolar offspring.

These findings may not seem surprising in the context of the chaotic and debilitating nature of bipolar illness, but families with a bipolar parent have not previously been described in this manner. Cooke et al. (13), recently reported no differences found on the FES between bipolar adults and healthy controls. However, the subjects were retrospectively reporting on their family of origin, remembering the environment when they were growing up as children and adolescents. Thus, they

did not describe their current family environment as parents.

Researchers have noted similar findings in families with members having other psychiatric illnesses. Families with a depressed parent have been reported as less supportive, with low independence and social integration and less well organized (23, 24). Children in these families who had higher support had fewer health problems themselves (24, 25). Families with a schizophrenic member have been found to have less cohesion and more conflict than control families (26). Mental illness in a parent may, therefore, increase the likelihood of less cohesive and supportive family environments.

It has been proposed that family environments may strongly affect those family members who have BD. Miklowitz et al. (14) reported that bipolar adults with families with high expressed emotion and negative affective style were more likely to relapse sooner after hospital discharge than those with families with low expressed emotion and a positive affective style. It might be extrapolated that those family members at high risk for BD might develop BD partly in response to a certain type of family. However, we did not find a specific type of family environment that was directly associated with a bipolar outcome for the children in this study.

It should be noted, however, that in a previous study, we reported that the diagnoses-free children in our cohort differed from those with psychiatric diagnoses in having temperaments that were highly adaptive and stable (15). At the same time, we previously reported on familial influences on defense and coping, which may ultimately prove to be important mediators of psychopathology as well (27). Thus, we are postulating that, ultimately, we will find significant interactions between individual characteristics, strengths, weaknesses and vulnerabilities, and environmental challenges and stressors that will account for more of the variance of the emergence of psychopathology in this cohort at high risk for psychopathology because of putative genetic factors.

Our findings may suggest a lack of association of a specific negative family environment and development of psychopathology in these children. However, this result may also be due to the possibility that the negative family characteristics reported overall by the bipolar parents were predominantly due to their individual perception and functioning. We do not know how the children themselves would report their family environments, as we are waiting for them attain a certain age before being able to report. On the other hand,

the FES is a relatively general reporter of family characteristics, and we may have to add more sophisticated assessments of familial interactions and shared and non-shared characteristics to ascertain the precise role of familial factors in the onset of disorder.

It is interesting to note that although our cohort of bipolar families reported less achievement orientation in their homes, they reported greater intellectual-cultural orientation. This finding was the only difference that might be construed as 'positive' towards child development. Researchers have suggested a link between BD and creativity, with many musicians, artists, and writers having been historically or currently diagnosed with manic-depression (28–30). This finding of bipolar families with higher intellectual-cultural orientation may be due to this phenomenon, but may also be an artifact of the fairly high SES of our cohort and their access to cultural activities in the greater San Francisco area.

Limitations of this study include use of a self-report questionnaire to determine family environment rather than an observation-based rating. The information was collected from the parents only; therefore, the children's impressions of the family environment were not included. We do not know if having a mood disorder in the parent may have biased the report in some manner, and there are data from our previous study with eating disorders and depression that this might be so (31). Parents' and children's psychiatric states may have affected the parental comorbid disorders which were not included in the analyses; these comorbidities may have had impact on the family environment as well. Our cohort tended towards a higher SES; it is not known what effect this may have had on the data collected. There may also have been some selection bias, in that this was a voluntary study, recruiting from the community and offering free psychiatric evaluations. Thus, more families with greater psychopathology in the offspring may have entered the study. Conversely, families with serious impairments making it difficult to make appointments or seek medical care may not have sought to join the study. Also, in our overall characterization of our cohort, we relied on normative data instead of a control comparison group that may have been better matched. Our relatively small sample of families with a bipolar child ( $n = 9$ ) may also limit our statistical power and preclude finding anything but very large differences in family environments when comparing them to families without bipolar children. Finally, as this is a single time-point study of still developing children, diagnostic groups may change with longitudinal follow-up.

Further studies on family structure of children at risk for BD need to be conducted to evaluate the potentially crucial role of the family in development of serious mood disorders in children.

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