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Expressed emotion, emotional distress, and individual and familial history of affective disorder among parents of adolescents with bipolar disorder

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Abstract

Parental expressed emotion (EE) attitudes are important prognostic indicators in the course of bipolar disorder (BD) in adolescents and adults. This study examined the hypothesis that parents' own susceptibility to affective disturbances contributes to their likelihood of high-EE attitudes. We examined past-week levels of emotional distress, lifetime affective diagnoses, and family histories of affective disorder among high- and low-EE parents of 86 adolescents with bipolar I or II disorder who were recovering from an episode of depression or (hypo) mania. High EE parents endorsed higher concurrent levels of depression, anxiety, and anger/hostility than low EE parents, and reported a greater familial history of depression and BD. No differences between high and low EE parents were found in concurrent levels of interpersonal sensitivity, lifetime rates of affective disorders, or familial loading of anxiety disorder. Parents' distress at the time of the EE assessment was the strongest correlate of EE. The results suggest that susceptibility to affective psychopathology may be an important contributor to the development of EE attitudes among parents of adolescents with BD.

Keywords

Expressed emotion; Pediatric bipolar disorder; Family history

1. Introduction

A growing literature suggests that family stress plays an important role in the course of bipolar disorder (BD). A common index of family stress is expressed emotion (EE), a measure of criticism, hostility, and/ or emotional overinvolvement in caregiving relatives (i.e., parents, spouse, siblings) of a concurrently ill psychiatric patient. EE is based on an interview with the relative during or shortly after the onset of a patient's illness episode. Several studies demonstrate that high EE in relatives is prospectively associated with risk of

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mood relapse among adults with BD (Kim and Miklowitz, 2004; Miklowitz et al., 1988; O'Connell et al., 1991; Priebe et al., 1989; Yan et al., 2004). Although fewer studies have examined youth with BD, low levels of maternal warmth are associated with more recurrences and shorter recovery periods in preadolescent and adolescent manic patients followed for up to 8 years (Geller et al., 2008; Geller et al., 2004) High parental EE is also associated with enhanced effects of family therapy in adolescents with bipolar spectrum disorders (Miklowitz et al., 2009; Miklowitz et al., 2013). Thus, EE is prognostically important for the early course and treatment of BD.

Understanding the origins of high- or low-EE attitudes in parents may clarify the role of environmental factors in the course of mood disorders and elucidate targets for intervention or prevention. Several perspectives on the origins of EE have been proposed. Some studies suggest that high EE attitudes in parents develop as a reaction to patient characteristics. A few studies have reported cross-sectional relations between parental EE and the severity of mood symptoms in youth, including depressive symptoms (Schwartz et al., 1990), externalizing symptoms (Tompson et al., 2010), and suicidal ideation (Ellis et al., 2014). However, no single patient variable has consistently explained the relation between EE and relapse across disorders, suggesting patient factors may be limited in their ability to explain the origins of EE attitudes (Hooley, 2007; Peris and Miklowitz, 2015).

An alternative perspective is that EE is at least in part a function of parent or caregiver characteristics. In support of this view, several personality features distinguish high versus low EE relatives. High EE relatives tend to be less tolerant of culturally aberrant or atypical behaviors than low EE relatives (Hooley and Hiller, 2000), and endorse a greater sense of agency regarding outcomes in their own lives compared to low EE parents (Hooley, 1998). High-EE relatives also tend to believe that their ill relative possesses more control over his or her own behaviors than do low-EE relatives (Hooley, 2007). Attributional biases such as these have been reported among high EE relatives of individuals with schizophrenia (Hooley, 2007), depression (Hooley and Campbell, 2002), and adult BD (Wendel et al., 2000). Thus, there is evidence that the origins of EE attitudes partially reside in the relative's personality or belief systems.

A cluster of parent/caregiver characteristics that has received less attention, particularly in families of patients with mood disorders, is the susceptibility of family caregivers to mood or anxiety disorders. A diathesis-stress conceptualization of EE suggests that relatives with increased vulnerability to experiencing affective symptoms – as measured, for example, by concurrent depression, anxiety, or lifetime histories of affective illness – may be more sensitive to the stress of having an offspring or spouse with an acute mood episode, and may become more critical, hostile, or overprotective toward him/her during a period of symptom exacerbation (Hooley and Gotlib, 2000). In support of this view, present and lifetime levels of anxiety and depression have been associated with higher EE in parents of youth with anxiety disorders, depression, and psychosis (Domínguez-Martínez et al., 2017; Hirshfeld et al., 1997; Tompson et al., 2010). Given that mood disorders are highly prevalent in the relatives of those with BD, it is plausible that a similar pattern exists among parents of youth with this disorder. The one study of EE and psychopathology in caregivers of adult patients

with BD found no relation, although approximately half of the participating relatives in this study were spouses with no genetic relation to the patient (Goldstein et al., 2002).

In addition to concurrent and lifetime levels of psychopathology, a family history of affective disorder has long been recognized as a risk factor for emotional disturbance. Parents of youth with BD who have more familial loading for affective illness (i.e., a diathesis) may be more likely to be high in EE than parents with less familial loading. No study has tested this hypothesis, although one study examined the family psychiatric histories of parents of young adults with schizophrenia (Subotnik et al., 2002). The authors found no overall differences in loading for mood disorder in the family histories of high versus low EE parents. However, heritability of mood disorders, of which emotional distress is a core feature, is especially high in the families of patients with BD (Lichtenstein et al., 2009; McGuffin et al., 2003). Thus, regardless of parents' own psychiatric status, it is possible that family history of affective illness is an important component of EE among parents of youth with BD, either independently or in conjunction with parents' individual emotional symptoms.

Drawing from diathesis-stress conceptualizations of EE, this study examined the role of family psychopathology in parental EE attitudes using a sample of adolescents with BD, all of whom entered shortly after the onset of an acute depressive, manic, mixed, or hypomanic episode. We hypothesized that compared to low EE parents, high EE parents would endorse (1) higher current levels of emotional distress (i.e., depression, anxiety, anger/hostility, or interpersonal sensitivity); (2) greater lifetime rates of affective disorders (i.e., major depressive disorder, mania/hypomania, anxiety disorder); and (3) a greater frequency of affective disorders in family pedigrees. Secondarily, we hypothesized that levels of emotional distress at the time of the EE interview would be the best of these predictors of parental EE status.

2. Method

2.1. Participants

Participants were 95 parents and their 86 offspring recruited as part of a three-site randomized trial of family-focused treatment plus pharmacotherapy for youth (ages 12–18) with bipolar I or II disorder. Eligible youth had experienced a manic, hypomanic, depressive, or mixed episode within the three months leading up to enrollment; (hypo) manic or depressive symptoms of at least moderate severity in the previous month; and a willingness to undergo pharmacotherapy for the 2-year study period (see below for sample demographics). At least one parent was willing to participate in therapy sessions (details in Miklowitz et al., 2014).

2.2. Measures

Youth diagnoses were determined by the Kiddie Schedule for Affective Disorders and Schizophrenia, Present and Lifetime Version (KSADS-PL; Kaufman et al., 1997) and a separate interview from a board-certified psychiatrist. Current severity of manic and depressive symptoms were rated using the 6- or 7-point K-SADS Mania Rating Scale and

Depression Rating Scale, respectively. Interviewers rated the worst week in the past month. Cross-site reliability (intraclass correlations) of the rating scales was 0.89 for the Depression and 0.81 for the Mania Rating Scales.

EE status (high or low) was coded from Five Minute Speech Sample (FMSS; Magaña et al., 1986) interviews of parents. For the FMSS, parents were asked to talk openly for five minutes about "what kind of person [the child] is and how the two of you get along together." Audiotaped responses were coded for the presence of criticism, hostility or emotional overinvolvement using standardized procedures (Magaña et al., 1986). Reliability (ICC) on EE status between 12 trained co-raters across 10 FMSS samples was 0.95.

Parents' concurrent levels of emotional distress (past-week levels of depression, anxiety, anger/hostility, interpersonal sensitivity) were assessed using the Symptom Checklist-90, Revised (SCL-90; Derogatis and Unger, 2010). Cronbach's alphas for the depression, anxiety, anger/ hostility, and interpersonal sensitivity subscales were in the good to excellent ranges (α's = 0.93, 0.84, 0.79, 0.82, respectively). The SCL-90 was administered at the same visit as the FMSS.

Lifetime presence of familial major depressive, bipolar, and anxiety disorders was gathered from parents using a semi-structured interview, the Family History Screen (FHS; Weissman et al., 2000). Each parent was rated dichotomously as either having or not having a personal history of each mood disorder. To improve the sensitivity of secondhand reports of relatives' symptoms, each parent's first-degree relative (i.e., the parent's siblings, own parents, and offspring ≥11 years old other than the patient) was rated on a 0–3 scale for each syndrome or disorder, where a 0 indicates no lifetime symptoms and a 3 indicates that lifetime DSM-IV criteria were met. Intermediate scores indicate subsyndromal levels of the disorder. To calculate lifetime familial loading of affective syndromes in first-degree relatives of parents, we summed the total FHS score for each disorder across each parent's first-ldegree relatives and divided by the total number of relatives. Severity loadings ranged from 0–3. For example, a parent with two first-degree relatives, one with fully syndromal major depression and one with no psychiatric history, would receive a severity loading of (3 + 0)/ 2 = 1.5.

2.3. Statistical analyses

For our primary hypotheses, we compared high and low EE parents on their past-week levels of emotional distress (SCL-90) using t tests, and on their personal lifetime (FHS) psychiatric diagnoses using χ^2 tests. We compared high and low EE parents on severity loadings for family history of affective disorders among their first-degree relatives (e.g., the parent's siblings) using t tests.

The parent's vulnerability to psychopathology is of theoretical interest to conceptualizations of EE. To that end, we computed summary scores for each of the three parental dimensions (total current emotional distress, lifetime affective diagnosis, family history of affective disorder) by summing the subscales within each dimension that were significantly related to EE status. For example, all SCL-90 subscales that significantly differed between high- and low EE parents were summed to create an SCL-90 summary score. We then entered these summary variables into a series of logistic regression analyses to determine their

comparative and joint contributions to the prediction of high- vs. lowEE status. Cox and Snell R^2 and regression coefficients were examined for each model to evaluate the incremental proportion of variance added by each predictive variable.

To evaluate whether parental EE status was related to the adolescents' current symptom severity levels, we compared high- and low EE parents on adolescents' Mania Rating Scale and Depression Rating Scale scores collected near to the time of the FMSS assessment of EE, using *t*-tests. For these analyses, where two parents corresponded to a single child, we included only the mother's data as correlates of adolescents' mania or depression symptoms. Given our interest in parental susceptibility to emotional distress, other aspects of child symptoms (e.g., psychosis) were not examined.

3. Results

3.1. Sample characteristics

Parents in this study were 78 mothers and 17 fathers, with a mean age of 42.89 (SD = 6.23). Participating youth ($N = 86$) were on average 15.5 years old (SD = 1.41); 54.6% ($n = 47$) were female, with an average socioeconomic position of middle to upper-middle class (Hollingshead $M = 44.9$, SD = 13.99). There were 47 (55%) with a diagnosis of bipolar I disorder and 39 (45%) with a diagnosis of bipolar II disorder. The majority of youth were White ($n = 75$, 87.2%); 7 (8.1%) were Black, 3 (3.4%) were Multiracial, and 1 (1.2%) was Native American. In regards to ethnicity, 5 (5.8%) were Hispanic. The high and low EE parents did not differ in age or on any of the youth demographic variables.

3.2. Relation of parental symptoms, parental diagnostic history, and family history of mood disorders to parental EE classifications

As shown in Table 1, high EE parents endorsed more past-week symptoms of depression ($t(94) = 3.31$, $p = 0.001$), anxiety ($t(94) = 3.73$, $p < 0.001$), and anger/hostility ($t(94) = 3.03$, $p = 0.003$) on the SCL-90 than low EE parents. No differences were observed in levels of interpersonal sensitivity ($t(94) = 1.48$, $p = 0.14$). High and low EE parents did not significantly differ in the probability of having a lifetime diagnosis of major depressive disorder ($\chi^2(1) = 2.66$, $p = 0.10$), bipolar disorder ($\chi^2(1) = 1.03$, $p = 0.31$), or anxiety disorder ($\chi^2(1) = 1.62$, $p = 0.20$) on the FHS. In their family pedigrees, high EE parents had higher familial loadings of depressive ($t(70) = 2.12$, $p = 0.03$), and manic/hypomanic syndromes ($t(70) = 2.01$, $p = 0.04$) than low EE parents, but not anxiety syndromes ($t(70) = 1.86$, $p = 0.07$).

Next, we examined the independent and additive relations between the parental SCL-90 and family psychiatric history summary variables and EE status, using logistic regression models. Parents' own diagnostic history, their family history of anxiety syndromes, and concurrent levels of interpersonal sensitivity were all unrelated to EE, and therefore these variables were excluded from the logistic models. Given our hypothesis that current emotional distress would be most closely related to EE status, we first regressed EE on the new SCL-90 variable indicating total current levels of depression, anxiety, and anger/hostility. In model 2, we regressed EE on the new family history variable indicating total

history of depressive and manic/hypomanic symptoms among parents' first-degree relatives. In model 3, we considered the joint and individual contributions of total current emotional distress and family history of mood syndromes by regressing EE on both of these aggregate variables simultaneously.

The full results of the models are displayed in Table 2. As expected from the earlier t tests, both total current emotional distress (model 1) and family history of depressive and manic/hypomanic syndromes (model 2) significantly predicted EE status when entered into individual regression models. In the third model, which included both variables together, family history of depressive and manic/hypomanic syndromes was no longer significantly related to EE, and the effect of current emotional distress on EE was attenuated, but still significant ($\chi^2(1) = 3.99$, $p = 0.046$).

3.3. Sensitivity analyses for non-independent observations

For a total of 9 adolescents, both parents participated in this study. Thus, there is potential for the data from these participants to be non-independent. For example, parental EE classification of one parent may be dependent on the classification of his/her spouse. A set of sensitivity analyses indicated that dropping the father's data in these 9 cases did not change the results of any of the group comparisons or logistic regression models reported above. For example, parents' current symptoms of depression, anxiety, and anger/hostility continued to be associated with EE status (all comparisons, $p < 0.005$) when only one parent per family was considered

3.4. Adolescent symptoms and EE classification

Adolescent mood symptom severity (worst week of symptoms in the past month) was unrelated to parental EE classification. High- and low EE parents did not differ in the current severity of their offspring's Depressive Rating Scale (high EE: $M = 25.9$ (10.7); low EE: $M = 24.8$ (10.0); $t(84) = 0.49$, $p = 0.63$) or Mania Rating Scale scores (high EE: $M = 31.1$ (11.5); low EE: $M = 26.8$ (10.3); $t(84) = 1.80$, $p = 0.08$) from the K-SADS.

4. Discussion

This study examined the relation of individual and family affective variables and EE status among parents of adolescents recovering from an acute episode of BD. High EE was associated with several indicators of susceptibility to affective psychopathology in parents, including more past-week symptoms of depression, anxiety, and anger/hostility, and a higher familial loading for depression and BD. Concurrent distress was more strongly associated with levels of EE than was family history of mood disorders among parents. Overall, these findings suggest that among parents of adolescents with BD, a susceptibility to affective disturbance is associated with the expression of critical comments, hostile remarks, or emotionally overinvolved attitudes when talking about their relationship with the adolescent.

No single variable has consistently explained the relation between caregiver EE attitudes and relapse across patients with mood or psychotic disorders (Hooley, 2007; Peris and Miklowitz, 2015). As previously noted, however, several patient and caregiver characteristics have been identified as correlates of high or low EE attitudes in caregivers. This is the first

study to show that parents' emotional distress and family history of depressive and bipolar disorder relate to EE levels among parents of adolescents with BD I or II. Although BD is highly heritable (Lichtenstein et al., 2009; McGuffin et al., 2003), our findings suggest that EE is probably not the result of familial heritability. Parents' EE status was not associated with their own diagnostic history or the current severity of BD in offspring, and was no longer related to their family history of mood disorder when controlling for concurrent levels of parental emotional distress. Thus, when considered together these findings suggest a limited genetic contribution to EE attitudes. It is important to note, however, that participants in this study were selected based on the presence of BD in the child, and thus the sample is enriched for familial mood disorder compared to the general population. Determining the extent of any absolute genetic influence on EE attitudes may require healthy control participants who were not selected for the presence of BD.

Because our findings are cross-sectional, causality cannot be inferred regarding the relation between EE and family psychopathology. Nonetheless, our findings are consistent with a diathesis-stress conceptualization of EE, and have implications for the understanding of the origins of EE attitudes. EE is most commonly thought of as a dyadic construct, representing the nature of the relationship between the patient and the relative (Hooley, 2007). From this perspective, one potential mechanism by which parents' emotional distress may be elicited is through the stress of caregiving (Awad and Voruganti, 2008; Perlick et al., 2007; Van der Voort et al., 2007). EE in this study was assessed during a period of symptom exacerbation in adolescents, a period likely to elicit stress and negative emotional reactions from relatives. Although we found no relation of current symptom severity in adolescents (worst week in prior month) to parental EE status, it is possible that the stress generated by parenting an offspring with active symptoms of BD provoked parents' vulnerability to high EE attitudes, either in the form of anger and criticism or anxiety and overprotectiveness (Miklowitz, 2004).

An additional consideration is the effect of internal processes within caregivers that may be independent of their relationship with the child. Personality attributes such as an internal locus of control (Hooley, 1998; Weisman et al., 2000) and intolerance for cultural non-conformity (Hooley and Hiller, 2000), which are believed to be relatively stable personal features, are more often seen in high versus low EE caregivers. Thus, internal personality or cognitive traits may play a role in provoking the emotional distress associated with EE attitudes.

The relation between emotional distress and parental EE has clinical/therapeutic implications. If emotional distress in fact underlies high-EE attitudes, reducing EE in relatives may require alleviating caregiver distress near the time of the patient's illness episode. A recent study of adults with BD found that an intervention for parent caregivers improved symptoms of depression among both the parents and the patients; further, patients' symptomatic improvements were mediated by improvements in parents' depression scores, even though patients did not participate in the intervention (Perlick et al., 2018). Thus, attention to the level of distress shown by parents during treatment may help to reduce levels of EE or their impact on the course of the patient's disorder.

Family interventions for adolescent BD represent a promising strategy for reducing distress in high EE environments (Peris and Miklowitz, 2015). Specifically, family-focused therapy and multifamily group psychoeducation have both been shown to improve symptoms in children and adolescents with mood disorders (e.g., Miklowitz, 2010; Fristad and MacPherson, 2014). These programs aim to reduce caregiver distress and improve family interactions through providing information about the nature and causes of mood disorders in children and skills training to communicate and solve problems more effectively.

Some limitations of our study should be noted. First, the cross-sectional design prevents inference of causality between emotional distress, family history of mood syndromes, and EE. Second, the FHS may be limited in its ability to detect psychiatric histories of parents' relatives, given that these relatives were not present for the interview and scores reflect the presence of psychopathology at any time in the lifespan. We also did not have reliability estimates for this instrument. Direct interviews of parents' first-degree relatives using structured diagnostic interviews, use of healthy control groups for clinical or population-based studies, and/or molecular genetic analyses with a range of psychiatric populations may further clarify whether EE is influenced by familial or genetic factors. Third, the majority of youth in the present sample were White and of middle to upper-middle class socioeconomic backgrounds. As cultural and contextual factors are likely to influence relatives' EE attitudes (e.g., Gurak and Weisman de Mamani, 2017; Weisman et al., 2003), the role of individual and familial levels of affective psychopathology should be examined across diverse sociodemographic populations.

A fourth study limitation was the measurement of EE using the FMSS. The FMSS, which is much shorter than the standard Camberwell Family Interview, has been found in some studies to under-identify high EE caregivers (Hooley and Parker, 2006). Replication of the current study using the Camberwell interview and coding procedures would increase confidence in the findings.

The results of our study provide evidence for the role of affective vulnerabilities as correlates of EE in parents of adolescents with BD. Together, these results suggest that affective vulnerability in caregivers is an important area of investigation for studies of the origins of EE attitudes as well as interventions to reduce EE within families of patients with mood disorders.

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Table 1

Relations of affective psychopathology among parents and their first-degree relatives to parental expressed emotion (EE).

	High EE (*n* = 49)	**Low EE (*n* = 46)**		
Group differences in parents' current affective symptoms (SCL-90)				
	Mean (SD)		*t*	*p*
Depression	16.90 (10.75)	10.31 (8.42)	3.31	0.001
Anxiety	8.69 (6.00)	4.67 (4.41)	3.73	< 0.001
Anger/hostility	5.27 (3.65)	3.22 (2.87)	3.03	0.003
Interpersonal sensitivity	6.81 (4.10)	5.32 (5.54)	1.48	0.14
Group differences in parents' lifetime affective diagnoses (FHS)				
	Percent diagnosed		χ^2	*p*
MDD	48.6	29.4	2.66	0.10
Mania/hypomania	25.7	15.6	1.03	0.31
Anxiety disorder	34.3	20.6	1.62	0.20
Group differences in parents' family history of affective symptoms (FHS)				
	Mean severity ratio (SD)		*t*	*p*
MDD	0.91 (0.82)	0.52 (0.67)	2.12	0.03
Mania/hypomania	0.57 (0.53)	0.33 (0.47)	2.01	0.04
Anxiety disorder	0.74 (0.73)	0.41 (0.77)	1.86	0.07

Note. For SCL-90 analyses, *df* = 94 except for anxiety and interpersonal sensitivity, for which Levine's test estimated unequal population variances; *df* for these analyses therefore = 88.03 and 81.12, respectively. Some participants were missing family history data. For these analyses, there were 34 participants with high EE parents and 37 with low EE parents. Dimensional mania/hypo- mania variables were square root transformed to address skew. SCL- 90 = symptom checklist-90, revised; FHS = Family History Screen; MDD = major depressive disorder.

Table 2

Logistic regression models predicting parental expressed emotion status from current parental affective symptoms and parental family history of mood disorder.

Variable	*b* (SE)	Wald χ^2 (1)	*P*	Odds ratio	Cox & Snell R^2
Model 1					
SCL-90 total emotional distress	0.03 (*0.01*)	6.94	0.008	1.03	0.08
Model 2					
Family history total mood score	0.44 (*0.22*)	4.11	0.043	1.55	0.06
Model 3					0.12
SCL-90 total emotional distress	0.02 (*0.01*)	3.99	0.046	1.02	-
Family history total mood score	0.35 (*0.22*)	2.54	0.111	1.42	-

Note. SCL-90 Total emotional distress is the sum of SCL-90 depression, anxiety, and anger/hostility scales. Family history total mood score is the sum of the Family History Screen depression and mania/hypomania parental relative scales. SCL-90 = symptom checklist-90, revised.