Expressed Emotion and Levels of Family Psychopathology in Parents of Bipolar Adolescents

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Abstract

Objective. The objective for this study is to better understand the etiology of expressed emotion (EE) attitudes in parents by evaluating differences in psychopathology and family history among high- and low-EE parents of youth with bipolar disorder (BD). The primary hypotheses were: (1) high-EE parents are more likely than low-EE parents to have a personal history of psychiatric disorders, and (2) high-EE parents are more likely than low-EE parents to have a family history of major affective disorders. Method. Levels of family psychopathology were assessed based on semi-structured interviews using the Family History Sheet to evaluate personal and familial psychiatric history. Included was a total of 63 parents of 56 bipolar adolescents who reported an acute mood episode within the past 3 months and were enrolled in family therapy. Parents were presented with a series of cards listing symptoms associated with various psychiatric conditions and were asked to indicate how many of the symptoms described themselves or first-degree relatives at any point in life. Parental EE attitudes were assessed using the Five Minute Speech Sample. Offspring of high- and low-EE parents were compared on scores obtained using the Kiddie Schedule for Affective Disorders and Schizophrenia, Mania and Depression Rating Scales. Results. There were no differences among high- and low-EE parents in their personal history of psychopathology, nor were there differences in levels of family loading of affective disorders. Secondary analyses revealed that high-EE/critical and critical/emotionally overinvolved (EOI) parents had a significantly greater personal history of anxiety than low-EE parents and parents who were EOI only. There were no differences between bipolar patients in high-EE and low-EE families in symptom severity. Discussion. Sensitivity and specificity of the family history interview may be compromised due to the difficulty of assessing the psychiatric histories of multiple subjects based on second-hand accounts. The findings should thus be viewed with caution. However, EE attitudes do not appear to be related to parental psychopathology or family history of affective disorders. However, anxiety may make parents particularly vulnerable to expressions of criticism during the open-ended FMSS. The EE/psychopathology relationship should be studied longitudinally using the FMSS and more rigorous genetic measures than those employed in the present study.

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INTRODUCTION

Bipolar disorder (BD) is a chronic, recurrent affective disorder characterized by periods of mania or hypomania alternating with periods of depression and/or mixed episodes, which include both mania and depression [American Psychiatric Association, 2000; (APA)]. Child and adolescent onset BD commonly co-occurs with disruptive anxiety, substance abuse, psychosis, suicidality, and impaired social and academic functioning (Bellivier, Golmard, Rietschel, et. al., 2001). In adult populations, these characteristics are often associated with resistance to treatment and poor outcome (Miklowitz, Biukians, & Richards, 2006). BD and other mood disorders are hypothesized to develop as a result of multiple interactive factors, including family functioning, life events, and biological and psychological vulnerabilities (Miklowitz et al., 2006). The current study aimed to better understand the family environment of bipolar adolescents by exploring associations between parenting attitudes and levels of psychopathology among 3 groups: caregivers, patients, and caregivers' first-degree relatives.

What is Expressed Emotion?

Among bipolar adolescents, family environments that adapt to the adolescent's developmental needs can play a key role in recovery from manic and depressive episodes (Asarnow, Goldstein, Tompson, et al., 1993; Keitner, Ryan, Miller, et al., 1995; Miklowitz, et al., 2006) as well as in the prevention of relapse (Miklowitz et al., 2006). However, conflictual family environments can interfere with BD patients' recovery from

manic or depressive episodes and predict early relapse post-psychosocial treatment. The distinguishing variables between protective family environments and family environments that put adolescents at risk are thought to lie in boundary setting and the family's response to the patient's mood episodes. Protective families implement educated, structured responses, whereas high-risk families are emotionally reactive and demonstrate excessively flexible or rigid boundaries (Miklowitz et al., 2006). More broadly, life stress and stressful life events have been shown to be a risk factor for the onset of mood episodes among individuals with mood disorders (Johnson, 2005; Kim, Miklowitz, Biukians, et al., 2007). Within this context, family discord likely plays a role in the escalation or perpetuation of these stressful events or periods.

Of particular interest within this context are parental EE attitudes, which have received much attention in the research of a variety of medical conditions, particularly psychiatric disorders (Vaughn & Leff, 1976; Butzlaff & Hooley, 1998). EE is defined as an individual's hostile, critical, or emotionally overinvolved attitudes about the patient. EE has been rated in family members, usually a partner (Hooley & Teasedale, 1989) or caregiver (Hahlweg, Goldstein, Nuechterlein, et al., 1989; Miklowitz, Goldstein, Falloon, et al., 1984; Parker, Jonston, & Hayward, 1988), though has been measured in other family members as well (Miklowitz, Strachan, Goldstein, et al., 1986; Simoneau, Miklowitz, Richards, et al., 1999). It is traditionally rated using the Camberwell Family Interview (CFI), a 1.5 hour semistructured interview that focuses on relatives' reactions to patient behaviors and symptoms. The current study, however, employs the Five Minute Speech Sample (FMSS) evaluation (Magaña, Goldstein, Karno et al., 1986). During the FMSS, relatives are prompted to share thoughts or feelings about the patient,

uninterrupted by the interviewer. "High-EE" individuals are those who express significant critical, hostile, or emotionally overinvolved (EOI) attitudes toward the patient during the course of the FMSS. Specific rating criteria are described below.

The relationship between EE and symptom relapse has been studied extensively in various clinical populations. Initially explored in relatives of schizophrenic patients (Vaughn & Leff, 1976; Brown, Birley, & Wing, 1972), EE has been shown to have a robust effect across populations with mood disorders (Hooley & Teasedale, 1989, Miklowitz, et al., 2006), eating disorders (Szmukler & Eisler, 1985), Alzheimers-induced dementia (Vitaliano, Becker, Russo, et al., 1989), and diabetes (Koenigsberg, Klausner, Pellino, et al., 1993). In fact, in comparison to studies of schizophrenic patients and their families, the EE-relapse relationship has been shown to be even stronger among bipolar populations. In a trial of 23 young adult bipolar in-patients, those returning to an environment high in EE and negative affective style, defined as a tendency toward dysregulated emotional reactivity (Davidson, 2004), had a 94% rate of relapse within nine months, whereas those patients returning to a family environment which was not high in either had a 17% rate of relapse (Miklowitz et al., 1988).

What Causes Expressed Emotion Attitudes?

While research has explored the etiology of EE, its origins remain unclear. Both genetically- and environmentally-based models have been proposed, some of which highlight the contribution of the relative, and some of which highlight that of the patient in the development of EE attitudes. Unidirectional models are those that focus on the contribution of one individual, whereas bidirectional models emphasize the joint

contributions of multiple relatives. The following section outlines the factors hypothesized to influence EE attitudes.

A. Unidirectional Models

According to unidirectional models, a caregiver's EE status results from the significant influences of a single individual within the family, namely the patient or the caregiver himself. It has been suggested that EE status is one component of a broader phenotype, including temperamental traits, psychiatric status, beliefs regarding locus of control, or beliefs regarding the patient herself. Further, cross-sectional and longitudinal research has examined the degree to which characteristics of the patient, such as symptom severity, are associated with or predict a relative's EE status. In the following section, findings regarding unidirectional hypotheses are reviewed.

Influence of Relative. Most relevant to the present study, scholars have hyopthesized that high-EE attitudes are a reflection of symptomatology within the high-EE individual. Support for this hypothesis is mixed. In an effort to determine the degree to which maternal depression and EE status independently impact child symptomatology and functioning, Nelson and colleagues (2003) found that while the degree of maternal depression predicted high-EE criticism, high-EE was not solely an expression of maternal depression; rather, the two exerted their effects on child symptoms and functional impairment independently. Other studies have provided evidence against a significant correlation between parental psychopathology and EE attitudes (Goldstein, Miklowitz, & Richards, 2002), whereas others still have found that those parents whose high-EE

attitudes remained stable over time and measurement had the highest rates of personal psychopathology (Goldstein, Talovic, Nuechterlein, et al., 1992).

While cross-sectional designs have historically not revealed psychiatric differences in relatives grouped as high- and low-EE, Goldstein et. al. (2002) found that greater levels of lifetime Axis I diagnoses were associated with lower EOI scores among caregivers of bipolar adults. In combination with the Nelson study mentioned above, these results may suggest inherent distinctions between high-EE parents of different subtypes (i.e., critical or emotionally overinvolved).

Hooley and colleagues have suggested an alternative approach to the relative-influence perspective of EE attitudes (1995; Hooley & Gotlib, 2000). This approach highlights the cognitive attributions relatives make regarding the patient's behaviors, and overall has received more empirical support than the EE/psychopathology model. Hooley found that high-EE critical relatives are more likely than low-EE relatives to make attributions of controllability with respect to the patient's symptoms. In other words, high-EE relatives regard the patient's disruptive or distasteful behaviors as willful or intentional, whereas low-EE relatives tend to regard these behaviors as resulting uncontrollably from, for example, the bipolar illness. Currently, several studies support this model, yielding results from both schizophrenic and bipolar samples (Simoneau, Miklowitz, & Saleem, 1998; Hooley & Gotlib, 2000).

Influence of Patient. There is some evidence that symptoms of the patient unidirectionally affect EE attitudes of relatives. Hooley and Richters (1995) observed significantly more critical comments among parents coping with a schizophrenic patient

for 3-5 years than those parents coping with a schizophrenic patient for less than one year. Comparing several publications (e.g., Hogarty, et al., 1986; Tarrier, Barowclough, Vaughn, et al., 1988; Miklowitz, 2004), it seems that between 25 and 50% of EE attitudes among these populations tend to improve when patients' symptoms improve. In part, these findings support the hypothesis that EE attitudes develop as a function of symptomotology in the patient.

However, refuting evidence lies in other findings. Neither schizophrenic nor bipolar patients in high-EE families are found to be more ill to begin with when compared with patients in low-EE families (Vaughn & Leff, 1976; Miklowitz et al., 1988; Miklowitz, Axelson, & George et al., 2009), nor do the most severely critical, hostile, or emotionally overinvolved relatives have children with the most severe illness episodes (Miklowitz, 2004). Such findings suggest limitations in the patient-influence model of EE as this model would predict that levels of relatives' EE criticism would increase as levels of patient severity increase. Overall, data regarding this unidirectional model of EE are inconclusive (Miklowitz, 2004).

B. Bidirectional Models

The patient- and relative-influence models assume that relatives' EE attitudes result from a single individual in the family. These unidirectional models are therefore limited in their scope of describing the family system and the individuals therein. A more inclusive model would consider the influences of both relative and patient in the development of EE attitudes and the combined effect in a family coping with an ill member. In the following section, two separate and empirically supported bidirectional

influences are described. Then, a comprehensive developmental psychopathology view of EE attitudes as presented by Miklowitz (2004) is discussed.

Influence of Interactions Between Relative and Patient. Disordered communication has long been observed in families of the psychiatrically ill (Fromm-Reichman, 1948; Bateson, Jackson, Haley, et. al., 1956; Lee, 1975). Recent studies have shown that high-EE families have been found to be most distinguishable from low-EE families by a "three-volley sequence" of negative expressivity, involving a negative comment by one family member, a negative response by the patient, and a third negative comment by the first member. This "reciprocal negativity" has been observed during or after problem-solving discussions across several psychiatric disorders (Hooley, 1986, 1990; Hahlweg et al., 1989, Strachan, Feingold, Golstein, et al., 1989; Simoneau et al., 1998; Simoneau et al., 1999).

The reciprocal nature of these interactions suggests that multiple individuals contribute to the tumultuous family environments of bipolar patients. However, such observations do not point to family conflict as a cause or effect of high-EE attitudes among caregivers. It remains possible that these interactions provoke the criticism, hostility, and emotional overinvolvement that typify high-EE attitudes. However, rather than causing high-EE, it is more likely that negative familial interactions *characterize* the family environment of high-EE caregivers. Salient characteristics among both patient and caregiver (e.g., distress intolerance, negative affectivity) may interact to produce a high-stress family environment that has been observed in the form of reciprocal negative interactions.

Influence of Shared Biology Between Relative and Patient. Despite diagnosable psychopathology, some parents may be apt to react to a child's cognitive or temperamental deficits with frustration, anxiety, or guilt, as a result of their own neurobiology (Miklowitz et al., 2006). This biological makeup may act as a shared vulnerability factor between a parent and his or her bipolar child. Indeed, several molecular correlates have been identified among populations with affective disorders (Manji, Quiroz, Payne, et al., 2003). Classic and well-supported theories on the pathophysiology of BD have focused on amine neurotransmitter systems, including transport, depletion, and/or hyperactive models within the serotonergic, dopaminergic, noradrenergic, and cholinergic systems. Such systems are widespread in the limbic brain, which has long been understood to be important for regulation of basic mammalian functions such as sleep, appetite, arousal, and mood. Consistent with the amine neurotransmitter approach, common symptoms of BD include disruptions in several of these physiological and behavioral domains.

However, recent evidence suggests that the intracellular signal transduction pathways catalyzed by the binding of these neurotransmitters to post-synaptic receptor sites may be significant to the extent that therapeutics targeting these post-receptor sites may be necessary in improving pharmacotherapy for BD patients (Manji et al., 2003). Examples of these intracellular processes include the protein kinase C (PKC) and cyclic AMP (cAMP) signaling pathways. The family of G proteins include a peripheral cell membrane protein, which, when active, triggers the cAMP signaling cascade. This protein is implicated in basic mammalian functions such as those described previously,

making them targets for cellular and molecular research in BD (Manji, 1992). Abnormalities in G protein function as well as in PKC activity have been found in the central nervous systems of individuals with affective disorders (Freidman et al., 1993; Young, Li, Kish, et al., 1993; Hahn & Freidman, 1999; Wang & Freedman, 1996).

Given that mood disorders have a clear and strong genetic component, it is possible that among families with a bipolar adolescent and an unaffected parent, the parent and child may, to a varying degree, share diverse cellular abnormalities within the central nervous system. The effects of mood, appetite, and sleep disturbances, joined with several other risk factors, as discussed below, may increase the risk for two individuals with moderate to severe disturbances in these domains to experience a psychosocially imbalanced dynamic. Biological abnormalities therefore serve as a candidate contributor to the ways in which individuals (i.e., parents of bipolar adolescents) may be predisposed to react affectively to their child's functional impairment (e.g., with criticism). A development of these vulnerabilities in combination with various environmental stressors, including the burden of a patient's acute mood episode, may therefore predispose these families to negative interactional patterns. Among high-EE parents, a greater personal and family history of psychopathology, particularly of mood disorders, may be a reflection of greater cellular abnormalities within the central nervous system, as compared to low-EE parents.

C. The Developmental Psychopathology Model

Data regarding aforementioned postulates suggest that EE is at least in part an interactional process between relative and patient. Could high-EE attitudes begin with a

predisposition to emotional and behavioral dysregulation in both parent and child, but be exacerbated by the patient's psychosocial dysfunctions and, subsequently, negative familial interactions? A developmental psychopathology perspective on early-onset BD and EE seems to be the most comprehensive approach to understanding these phenomena. This perspective endorses the biopsychosocial model (Engle, 1977), which views psychiatric illness within an interpersonal context and assumes that biological, cognitive, social, and cultural factors contribute to the expression of the illness as well as to the psychology and behavior of members within the family. It holds that patient and relative react to the psychological health and behavior of one another based on the combined effects of these factors, and that each individual in turn affects the others' functioning. The developmental psychopathology approach as proposed by Miklowitz (2004) incorporates this theory into a model that views high-EE attitudes as a result of a lifetime of development of biological and environmental factors. Unlike the parent-influence models described above, this perspective views EE attitudes not as a character defect of the relative but as an indicator of the family climate as a whole. Finally, this model provides a *pathway* that accounts for many interactive factors that may contribute to the relationship between a patient's symptoms, a relative's symptoms, and that relative's EE attitude.

As an example of such a pathway: an individual with a genetic predisposition to emotional dysregulation has a child with whom he or she shares a common neurobiological abnormality, such as a hyperactive amygdala. However, the biochemical abnormality is greater in the child, and he or she is temperamentally difficult. The resulting early dynamic between parent and child poses immediate interpersonal

challenges between the two. It is at this point in the pathway where the bidirectional feedback between parent and child begins, creating a dyad fraught with conflict. The parent reacts to the child's difficult temperament with frustration, anger, guilt, or anxiety, and becomes critical or overprotective of the child. Though the parent's intention may be to help the child through these criticisms and protective behaviors, the child may interpret the parent's reactions as suggesting that the child is incapable, incompetent, or unsatisfactory. As self-schemas develop around these beliefs during the first several years of life, the child's behavior may become more withdrawn, irritable, or affectively reactive, therefore becoming a target of more concern and criticism from the mildly or moderately dysregulated parent, or from other individuals (e.g., teachers, family friends). Levels of family stress, to which the parent may be vulnerable, increase dramatically as an index mood episode eventually takes place, and the negative feedback loop thus intensifies.

It is possible that high-EE attitudes develop through various pathways. The aforementioned hypothetical dyad describes one of many sequences of biopsychosocial processes that may result in high-EE parental attitudes. The vulnerability-stress approach to understanding psychiatric disorders holds that these disorders result from a biological predisposition to the disorder that is expressed when met with some environmental stress. These diatheses and environmental pressures may exist in varying degrees. For example, one individual may have a strong genetic predisposition to emotional dysregulation (as observed by a family pedigree heavily loaded with mood disorder), such that a lesser degree of environmental stress (e.g., negative life-events, poor temperamental fit with child or partner) is needed to trigger anxiety, frustration, and the behavioral

manifestations of these emotions, such as criticism or emotional overinvolvement. In other words, the individual is highly vulnerable to life stress. A second individual, however, may have a less loaded predisposition to emotional dysregulation (e.g., fewer abnormalities in G protein expression within the CNS) but experiences a high level of environmental stress throughout the course of or at any given point in his or her life. For this second individual, a greater degree of environmental stress is needed to trigger the expression of a high-EE attitude.

Cicchetti and colleagues (1993; 1997) have suggested that other protective- or risk factors play a role in the development of EE attitudes as well. These include both endogenous and exogenous influences, and may take the form of personality and temperamental traits, such as locus of control, levels of self-consciousness, anxiety, self-discipline, and tolerance for unpleasant circumstances; or "transient buffers or losses" such as sudden losses or gains in social or occupational status. These biological and environmental factors may act as precursors, present in various magnitudes between individuals, which interact through the same developmental pathway and result in the same or similar high-EE attitudes among those individuals. Miklowitz (2004) has proposed that children for whom risk factors are present or for whom protective factors are absent are more likely to be negatively affected by a high-EE caregiver. Likewise, care-giving relatives who exemplify these conditions are likely more vulnerable to the effects of a mood episode of a child or spouse.

Expressed Emotion Across Generations

As previously discussed, research on the relationship between EE and personal symptomatology has received considerable attention. However, little research has been done with the aim of exploring psychiatric history of first degree relatives of high-EE individuals. Often replicated has been the observation of intergenerational transmission of psychopathology (Hammen, Shih, & Brennan, 2004; Kane & Garber, 2004; D'Onofrio, Slutske, Turkheimer, et al., 2007), personality traits (Soenens, Elliot, Goossens, et al., 2005), and trends in interpersonal relationships (Doumas, Margolin, & John, 1994; D'Onfrio, Turkheimer, Emery, et al, 2007). EE attitudes seem to be composed of some combination of these three psychological and behavioral domains. Evaluating the evidence of inheritance in these domains is therefore crucial for developing hypotheses about how, if at all, EE attitudes may be transmitted across generations. Recent studies have explored the phenomenon of the aforementioned intergenerational transmission across three generations, with mixed results (Warner, Weissman, & Mufson, 1999; Olino, Pettit, Klein, et al., 2007; Pettit, Olino, Roberts, et al., 2008). While grandparental psychopathology has been largely predictive of child symptoms among these studies, the mediating role of parental psychopathology is unresolved. Given this body of literature, one might suspect that if a relationship is to exist between EE attitudes and psychiatric history of first degree relatives, the genetic transmission of vulnerabilities to psychopathology would predict high-EE attitudes.

Among schizophrenic patients, Subotnik and colleagues (2002) found a significant correlation between parents' EE attitude and their family history of psychiatric illness. The results, however, were unexpected. A family history of mood disorder was associated with low-EE, whereas a family history of schizophrenia was not significantly

associated with either high- or low-EE. The authors speculated that prior exposure to a family member who has experienced and recovered from a major depressive episode may inspire tolerant and hopeful attitudes toward major psychiatric disorders. Schizophenia, however, is more likely to create a sustained family burden, and relatives are more likely to struggle to relate to the schizophrenic patient, factors that together may not lead as commonly to the development of tolerant attitudes toward major psychiatric disorders. The unexpected findings combined with the exposure postulation warrant further research regarding the family history of high- and low-EE caregivers.

In summary, EE status is an established predictor of symptom relapse post-psychosocial treatment in various psychiatric populations. While high- and low-EE families and individuals have been distinguished based on factors such as interactive and attributional styles, the origins of EE attitudes remain unclear. The core assumption of this study was that caregivers' critical, hostile, or emotionally overinvolved attitudes toward a bipolar child reflect the caregiver's diathesis to psychopathology (e.g., significant stress intolerance, emotional dysregulation). To address this issue, high- and low-EE parents were compared on their levels of individual psychopathology, rates of psychopathology among their first-degree relatives, and severity of psychopathology among their bipolar children.

If the study's core assumption is accurate, one would expect several outcomes following these comparisons. First, the same diatheses that are expressed as high-EE attitudes should be observable by other measures. *We hypothesized that high-EE parents of bipolar teenagers would have a greater personal history of psychiatric symptoms than*

low-EE parents (hypothesis #1). The current assumption would also expect that critical-hostile expressions among caregivers would reflect separate vulnerabilities from expressions of emotional overinvolvement, and therefore may differ in associated psychiatric diagnoses. This issue was addressed by comparing rates of psychopathology among parents who were classified as critical EE subtypes to those classified as noncritical.

Second, one would expect that those psychiatric diatheses hypothesized to contribute to high-EE would be found in biological relatives of high-EE caregivers. In exploring family history, this study focused on mood disorders, given the disorders' common symptoms (e.g., irritability, episodic nature), strong genetic component, and attention received in previous studies (Subotnik, Goldstein, Nuechterlein, et al., 2002). *We hypothesized that high-EE parents would be more likely to have a first-degree relative with major mood disorder than low-EE parents* (hypothesis #2). Similar to hypothesis #1, hypothesis #2 was further explored after reclassifying parents based on the presence of EE cricitism.

Third, a diathesis-stress approach to human behavior assumes that the greater the degree of biological predisposition, the more likely the phenotype in question will be expressed when met with environmental stress (Engle, 1977; Hooley & Gotlib, 2000; Miklowitz, 2004; Cicchetti, 2010). *Thus*, *we hypothesized that the greatest predictor of high-EE would be the joint contribution of the parent's own symptoms and his or her own family history of mood disorder* (hypothesis #3). The notion that EE can in part be understood by identifying a broader parental phenotype yielded our final hypothesis, that *the bipolar children of high- and low-EE parents would not differ in severity of manic*

and depressive symptoms (hypothesis #4).

METHODS

Inclusion Criteria And Sample Characteristics

Participants were part of an ongoing five-year, three-site randomized trial evaluating the efficacy of psychoeducational family-focused treatment for bipolar adolescents (FFT-A) plus optimal pharmacotherapy. Among this population, FFT-A has been shown to improve overall symptoms, reduce relapse rates, and increase medication adherance (Miklowitz, Axelson, Birmaher, et al., 2008). Subjects of the current study included bipolar adolescents and at least one parent for each adolescent. Participants were recruited from several referral-based sources, including community mental health centers and University of Colorado clinics.

To enroll in the treatment study from which the current sample was drawn, adolescent participants must have (1) met criteria for either bipolar I disorder or bipolar II disorder with a manic, hypomanic, or mixed episode in the past 3 months, or a depressed episode in the past 3 months with a prior history of a manic, hypomanic, or mixed episode; (2) had at least 1 week in the past 3 months which received a Psychiatric Status Rating of at least a 4 on a 0 to 6 scale on the Adolescent LIFE interview; (3) had at least 1 week in the past 3 months which received a score of at least 12 on the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS) Depression Rating Scale or at least a 13 on the KSADS Mania Rating Scale; (4) been willing to participate in family therapy and willing to take to medication as recommended by study psychiatrists; (5) lived with

at least one caregiver who was willing to attend therapy sessions. All participants were able and willing to give written informed consent or assent to participate upon intake.

No independent inclusion criteria existed for parents of the adolescents. However, for the present study, only data from biological relatives of the adolescent were considered; data from adoptive families were excluded. Subjects were drawn from each of the 3 sites participating in the current trial of FFT-A (Univ. of Colorado, Univ. of Cincinnati, Univ. of Pittsburgh). The subjects were a total of n = 147 families; adolescents were n = 80 females, n = 67 males; n = 125 Caucasian, n = 4 Asian, n = 15 Black or African American, n = 3 Native American; n = 12 of these adolescents were Hispanic or Latino. Of these families, 18 were adopted, and 73 had missing, uncollected, or unusable data (e.g., FMSS data for a stepfather only), resulting in 56 adolescents. Of the 56 adolescents, 7 had 2 biological parents with usable data, resulting in 63 caregivers.

Of the parent sample, there were 7 pairs of parents (14 individuals) for whom EE data were available, each corresponding to a single child, resulting in 7 fewer children than parents in the sample. Of these 7 children, 1 had 2 high-EE parents, 4 had 2 low-EE parents, and 2 had 1 high- and 1 low-EE parent. Overall, 28 children were paired with high-EE parents, and 30 with low-EE parents.

Measures and Assessment

Expressed Emotion. EE was assessed using the Five Minute Speech Sample (FMSS; Magaña et al., 1986). Caregivers are asked to "talk for five minutes about your child, tell us what kind of person s/he is and how the two of you get along together". The recorded interviews are assessed by "blind" evaluators for critical comments, hostility, or

emotional overinvolvement (EOI). High-EE subjects are subsequently categorized as critical or EOI subtype.

To meet criteria for high-EE critical, subjects must either (a) make one or more critical comments (resentments, dislikes, or disapproval), (b) receive a negative relationship rating (based on comments made about the relationship between parent and child that express a complete thought), or (c) make a negative initial statement about the child (a criticism, negative relationship statement, or negative attribution of character or behavior) during the FMSS.

To meet criteria for high-EE EOI, a subject must either (a) report self sacrificing or overprotective behavior (providing a pleasurable circumstance for the child at the caregiver's expense, diverting uncomfortable circumstances for the child's sake and at the caregiver's expense), (b) display excessive emotion (breaking down in tears), or (c) express excessive praise or detail about the past (5 or more positive comments about the child's character or abilities) during the FMSS.

Within the context of the larger treatment study, the FMSS interview is administered at intake, at follow up appointments completed in month 4.5, and again in month 9. However, the current study used only the initial EE assessment, given FFT-A targets EE attitudes with the aim to decrease high-EE characteristics, and the proposed study aimed to evaluate parental characteristics independent of treatment.

Parental and Family Psychiatric History. The current study used the Family History Sheet (FHS; Weissman, Wickramaratne, Adams et al., 2000) to assess current and past psychiatric history of the caregivers and their first-degree relatives. For each

patient, the semi-structured interview is conducted with at least one caregiver who is involved in FFT. Caregivers are provided with cards listing symptoms of several psychiatric disorders, and are asked to identify any symptoms that have been present in the lifetime of an identified first- or second-degree biological relative of the patient. Assessments of symptoms are made based on the DSM-IV-TR criteria.

The FHS measures the following syndromes and disorders: major depressive episode, dysthymia, mania, hypomania, schizophrenia, psychosis, anxiety, conduct disorder, substance abuse, substance dependence, and suicidality. A 0-3 severity dimension is used for each variable, and symptoms are noted to be recurrent if so determined in the course of the interview. Overall lifetime psychiatric severity was calculated by adding caregivers' total score for each variable.

Child Bipolar Symptoms. The severity of adolescents' manic and depressive symptoms was assessed using the KSADS Mania Rating Scale (MRS; Axelson et al., 2003) and Depression Rating Scale (DRS; Chambers et al., 1985). The measures allow extensive coverage of affective symptoms and a range of responses, as most questions are rated on a 1-6 severity dimension. Manic and depressive symptoms are assessed for the worst week in the past month. Each semi-structured interview measure was conducted separately with the adolescent and caregiver, and discrepancies in reports were resolved by making a consensus rating. Offspring symptom and parental EE data were collected on the same day, during study intake.

Data Analyses. Hypotheses 1, 2, and 4 were explored beyond the high/low categorization of EE after initial hypothesis testing by rerunning analyses based on EE subtype. However, because the sample lacked sufficient statistical power when categorized by each subtype (critical, critical/EOI, EOI, "borderline" critical or EOI, low-EE; primarily accounted for by the small number of EOI subjects and large number of "borderline" subjects), parents were categorized on a basis of high and low criticism. Thus, EOI subjects were grouped with low-EE subjects to comprise the low criticism group, and high-EE critical, high-EE/critical/EOI, high-EE/critical/borderline EOI and high-EE/EOI/borderline critical parents comprised the high criticism group. This grouping attempted to isolate the criticism variable.

Hypothesis #1: *High-EE parents of bipolar teenagers will have a greater personal history of psychiatric symptoms than low-EE parents.* Hypothesis #1 was tested using a *t*-Test, with parental FHS scores as dependent variables, and EE status as the independent variable. High- and low-EE parents were compared on average overall psychiatric score, which was computed by adding the sum of scores across each disorder. Secondary analyses were conducted on each specific syndrome or disorder screened in the FHS. This hypothesis was then explored by re-grouping the sample of caregivers based critical/noncritical EE types. Upon re-grouping, the above analyses (*t*-test and chi square) were reconducted.

Hypothesis #2: *High-EE parents will be more likely to have a first-degree relative with major mood disorder than low-EE parents.* Hypothesis #2 was also tested using a *t*-test, with parental EE as the independent variable and family mood disorder history score as the dependent variable. Relatives who scored a 3 (meets full DSM-IV-TR criteria for a

diagnosis) on major depressive episode, dysthymia, mania, or hypomania variables on the FHS were categorized as positive in this analysis. A caregiver's family mood disorder history score was calculated by dividing the number of the individual's biological first-degree relatives (excluding children) who scored a 3 on any of the previously mentioned variables by that individual's total number of biological first-degree relatives (again excluding children). The hypothesis was tested further by computing a chi-square analysis, in which high- and low-EE parents were identified as having either a presence or absence of mood disorder in any biological first degree relative (excluding children). This test used a 2 X 2 (EE X mood disorder) design. As with hypothesis #1, hypothesis #2 was then tested by regrouping parents based on critical or noncritical EE subtypes and rerunning the above analyses (*t*-test and chi-square).

Hypothesis #3: *The greatest predictor of high-EE will be the joint contribution of the parent's own symptoms and his or her own family history of mood disorder.* A Logistic Regression Analysis was used to examine the hypothesized interaction between parents' individual psychopathology and their family psychiatric history (independent variables) in predicting EE (dependent variable).

Hypothesis #4: *The bipolar children of high- and low-EE parents will not differ in severity of manic and depressive symptoms.* A *t*-test was conducted to test hypothesis #4, with child MRS, DRS, and combined scores as dependent variables, and parental EE status as the independent variable. To examine any differences in offspring symptom severity between high and low critcal parents, caregivers were then regrouped based on the high and low criticism criteria described above, and a *t*-test was conducted.

RESULTS

Prior to testing our hypotheses, a single factor Analysis of Variance (ANOVA) was conducted to determine if overall parental psychopathology scores differed across the three universities in which the larger study was conducted. No differences were found between the three sites [F (2, 60) = 1.1, p = .34], indicating equal distribution of rated parental psychopathology. Next, a single factor ANOVA was conducted to determine if family mood disorder history scores differed across sites. This analysis yielded no differences [F (2, 60) = .54, p = .54], also indicating equal frequencies of mood disorder. Finally, a single factor ANOVA was conducted to determine if offspring KSADS scores at study intake differed across the three sites, with no differences found [*MRS*: F (2, 55) = .85, p = .43; *DRS*: F (2, 55) = .57, p = .57; *sum*: F (2, 55) = .85, p = .43]. When data for these three variables were shown to be equal across study sites, we then sequentially tested our hypotheses.

Are high-EE parents more likely to have a lifetime psychiatric history than low-EE parents?

There were no significant differences between high- and low-EE parents in overall psychiatric score [(High-EE: M = 6.53, SD = 5.84); (Low-EE: M = 5.59, SD = 5.23); t (61) = -.96, p =.34]. Secondary analyses revealed no further differences between any syndrome or disorder screened on the FHS (for all, p >.05; see table 3). To explore the null findings, we attempted to isolate the criticism variable by assessing parental psychopathology among parents who expressed or did not express significant criticisms

during the FMSS. These analyses revealed no differences on overall psychiatric score [(Critical: $M = 6.92$, $SD = 5.8$); (Noncritical: $M = 5.7$, $SD = 5.3$); t (61) = -.86, $p = .39$]. However, the mean score for critical parents was significantly greater than for noncritical parents with respect to lifetime occurrence of clinical anxiety, as measured by the 0-3 FHS severity scale [(Critical: $M = 1.72$, $SD = 1.21$); (Noncritical: $M = 1.03$, $SD = 1.3$); t (61) = -2.12, $p = .04$. No other differences were found when average FHS scores were compared between high and low criticism parents.

Are high-EE parents more likely to have a family history of mood disorder than low-EE parents?

A *t*-test revealed that high- and low-EE parents did not differ in their family mood disorder history scores [(High-EE: $M = .28$, $SD = .32$); (Low-EE: $M = .17$, $SD = .17$); t (60) = -1.67, $p = .1$]. High-EE parents therefore did not have a greater ratio of affected to unaffected biological first-degree relatives than did low-EE parents, as determined by the family history interview. A chi-square analysis revealed no differences between high- and low-EE parents in their likelihood of having any first degree relative with a mood disorder [X^2 (1) = .19, $p = .1$]. No differences between mean family history scores were found when parents were grouped by critical and noncritical subtypes rather than overall EE status [(Critical: $M = .29$, $SD = .28$); Noncritical: $M = .2$, $SD = .24$); t (60) = .25], nor did a chi-square analysis indicate differences between these groups [X^2 (1) = .07, $p = .78$].

Table 4 summarizes the data relevant to hypothesis #2. The total number of caregivers' relatives who were evaluated during the family history interview with the

caregiver, the number of parents who had any family history of mood disorder, as well as the analyses conducted on these data, are presented.

Does the combined effect of caregivers' positive psychiatric history and a highly symptomatic child predict high-EE?

A logistic regression analysis revealed that neither parental psychopathology [Wald X^2 (1, N = 63) = .84, p = .36] nor family history of mood disorder [Wald X^2 (1, N = 63) = .4, p = .53] was a significant predictor of overall EE status. Likewise, the test of the overall model, which included these two variables, was not statistically significant [LR X^2 (1) = 1.88, p = .17]. In other words, the joint contributions of parental psychopathology and his or her family history of mood disorder did not predict EE status. Logistic regression output is summarized in table 5.

Do high-EE parents have more symptomatic offspring than low-EE parents?

The KSADS was used to measure the severity of manic and depressive symptoms of bipolar adolescents (n= 56). MRS, DRS, and combined scores of offspring of high- and low-EE parents were all similar [(*MRS* - High-EE: M= 29.71, SD = 11.16; Low-EE: M= 26, SD = 11.81; t {56} = -1.18, p = .24); (*DRS* - High-EE: M= 27.93, SD = 9.45; Low-EE: M= 25.53, SD = 9.87; t {56} = -.94, p =.35); (*sum* - High-EE: M= 57.64, SD = 14.95; Low-EE: M = 51.53; SD = 16.32; t {56} = -1.48, p = .14)], as were respective KSADS scores when parents were grouped by critical or noncritical EE subtypes [(*MRS* - High-EE: M = 30, SD = 10.03; Low-EE: M = 26.34, SD = 9.41; t {56} = -1.18, p = .24); (*DRS* - High-EE: M = 28.3, SD = 10.02; Low-EE: M = 25.63, SD = 9.04; t {56} = -1.03,

p = .3); (*sum* - High-EE: *M* = 58.3, *SD* = 15.96; Low-EE: *M* = 52.97, *SD* = 15.47; t {56} = -1.5, *p* = .14)]. The results indicate that the child's manic and depressive symptoms were no more severe for high-EE parents than they were for low-EE parents; further, that high-EE/critical or borderline critical parents did not have children whose bipolar symptoms differed in severity from those of high-EE/EOI and low-EE parents. Table 6 below presents these data.

DISCUSSION

The core assumption of this study was that caregivers' critical, hostile, or emotionally overinvolved attitudes toward a bipolar child reflect the parent's diathesis to psychopathology. To address this issue, high- and low-EE parents were compared on their levels of individual psychopathology, rates of psychopathology among their first-degree relatives, and severity of psychopathology among their bipolar children. No associations were found between EE and family history of major mood disorders or EE and children's manic or depressive symptoms. With the exception of high-EE/critical and critical-EOI parents showing a greater history of anxiety than high-EE/EOI and low-EE parents, no individual psychopathology differences between groups emerged. The general null results can be compared to findings of two previous studies that compared high- and low-EE parents on individual psychopathology (Goldstein et al., 1992; Goldstein et al., 2002), as well as findings from previous studies comparing high- and low-EE parents on the severity of child bipolar symptoms (Miklowitz et al., 1988; Miklowitz et al., 2009). The current results did not replicate an additional study by Subotnik et al. (2002), who

found that low-EE parents were more likely than high-EE parents to have a family history of major mood disorders.

Child Bipolar Symptoms

As anticipated, children's symptom severity did not differ between high- and low-EE parents in mania, depression, or the sum score of mania and depression. This finding is consistent with cross-sectional analyses conducted in previous studies of bipolar families (Miklowitz et al., 1988; Miklowitz et al., 2009). If EE attitudes develop as a function of child symptom severity, one would expect more severe bipolar symptoms among children of high-EE parents as compared to children of low-EE parents during or following an acute mood episode. This interpretation is applicable to the lack of differences found between bipolar offspring of critical and noncritical parents. While critical caregivers and home environments may differ in important ways from those characterized by emotional overinvolvement (e.g., Hooley and Gotlib, 2000), the relationship between these attitudes and the child's mood episodes are not revealed by only considering baseline assessments of these measures. In other words, the cross-sectional design used in the current study was unable to detect the ways in which parent and child characteristics likely influence each other over the onset and development of adolescent BD.

Support for this interpretation of the current results is provided by a study by Hooley and colleagues (1995), who found that parents living with a schizophrenic offspring for at least 4 years were more likely to be high-EE than those living with a schizophrenic offspring for less than 1 year. High-EE attitudes among relatives have also

been shown to convert to low-EE when patients improve (Hogarty, Anderson, Reiss, et al., 1986; Goldstein et al., 1992). While these longitudinal results may reflect the natural course of EE, it is possible they reveal the ways in which EE varies with patient symptoms.

Parental Psychopathology

High- and low-EE parents were compared on a variety of psychiatric disorders, as well as an overall psychiatric score, which was obtained by averaging the total sum of scores across each disorder. Including overall psychiatric score, no disorder reached significance. However, when parents were categorized based on critical and noncritical EE subtypes, high-EE/critical and high-EE/critical-EOI parents were found as a group to have a greater lifetime history of anxiety than high-EE/EOI and low-EE parents.

Discussion of general negative findings. The finding that high- and low-EE caregivers overall did not differ in rates of lifetime psychopathology largely replicates those of previous studies (Goldstein et al., 1992; Goldstein et al, 2002). The general null findings fail to support the hypothesis that high-EE attitudes reflect a caregiver's vulnerability to psychopathology and suggest the relevance of other possible contributors to EE: beliefs regarding the causes of the patient's behavior, recent interactions with the patient, or the duration of time having cared for the patient.

The current and previous cross-sectional findings that EE is largely unrelated to individual psychopathology nonetheless beg the question of why various differences have been found with respect to this variable. Indeed, Goldstein and colleagues (1992) found that high-EE caregivers who maintained their high-EE status 5-6 weeks after patients'

hospitalization had the most severe history of psychopathology. Goldstein and associates (2002) also found when measuring EE on a dimensional scale that lower EOI scores on the CFI predicted more Axis I DSM-III-R diagnoses in caregivers. Thus, while the Goldstein (1992) and Goldstein (2002) studies of EE and individual psychopathology at the cross-sectional level each yielded negative findings *overall*, intriguing correlations between EE and psychopathology were found when testing specific subgroups or dimensions of EE. Collectively, studies on EE and individual psychopathology may suggest that EE is heterogeneous.

Discussion of association between criticism and anxiety. This study found that criticism in parents was associated with greater levels of lifetime anxiety. Several contributing factors might underlie this finding. First, this study employed the FMSS. In the studies mentioned above, CFI-rated high-EE criticism was not found to be associated with any measure of individual psychopathology. The authors have suggested that individuals may respond differently to the open-ended nature of the FMSS compared to the more structured CFI (Goldstein et al., 1992; Goldstein et al., 2002) Possibly, parents who experience significant anxiety are more likely to spontaneously express critical comments about their child when met with the challenge of speaking freely about him or her for 5 minutes in the presence of study staff. Indeed, the EE/psychopathology finding of Goldstein et al. (1992) employed the FMSS at follow up, when differences between EE subgroups were found.

Beyond methodological considerations, the anxiety/criticism relationship observed in the current study raises questions about caregiver characteristics. Hooley et al. (1998) have demonstrated that caregivers of schizophrenic adults tend to attribute

patients' behavior to internal and controllable causes, and have a more internally based locus of control (LOC) than EOI or low-EE caregivers. Interestingly, Barlow (2002) has stated that at the core of anxiety disorders exists a considerable sense of uncontrollability when the anxious individual is confronted with potentially threatening experiences, and points to research suggesting that individuals with anxiety disorders in fact tend to endorse an *externally based* LOC (Nunn, 1998). It is possible that chronic anxiety, when met with a bipolar child's erratic behavior, may lead a caregiver to compensate for a sense of uncontrollability by being overly rigid, inflexible, or hostile toward the child.

The utility of EE criticism toward temperamentally difficult children has been suggested by Miklowitz (2004), who postulated that such expressions may be adaptive at some points in a family's life cycle, possibly serving as a motivator for behavioral change. However, the criticism/anxiety finding may reflect methodological limitations, and it is also important to note that the LOC finding has not been replicated in parents of bipolar adolescents. This pathway is therefore speculative in nature and its endorsement requires replication of the findings.

The isomorphic nature of EE has likely made further elucidation of the high-EE phenotype and interpretation of related pathological symptoms historically difficult. Indeed, Cicchetti has proposed the concept of "equifinality" as a major point of interest within the field of developmental psychopathology. Equifinality refers to the phenomenon whereby the same end state is reached from multiple initial conditions (Cicchetti & Rogosch, 1996). Observed levels of EE may be reflections of psychopathology, of an overall healthy psychological history, or of neither, depending on

the individual and the circumstances under which EE is measured. Nonetheless, the general negative findings with respect to hypothesis #1 suggest that EE and individual psychopathology are not related.

Family History of Mood Disorder

In this study, no differences were found in family history of mood disorders among high- and low-EE parents. To the author's knowledge, only one other study has tested the family history hypothesis of EE (Subotnik et al., 2002). While the Subotnik et al. findings were not consistent with the current study (low-EE caregivers were *more likely* than high-EE parents to have a first-degree relative with a major mood disorder), the implications for the current hypothesis remain unchanged: high-EE attitudes likely do not reflect a biological predisposition to affective dysregulation. Subotnik and colleagues suggested that environmental influences, such as prior exposure to a family member who has struggled with but overcome a mood disorder, may inspire hopeful attitudes in the care-giving relative.

It is possible that the present family history data did not capture subjects' true family loading of mood disorder due to the less intensive structure of the chosen interview (discussed in depth below). Similar to the EE/individual psychopathology findings, the isomorphism of EE may imply that not all caregivers of the same EE status will have comparable psychiatric histories, and replications of studies that have tested this hypothesis may therefore be difficult. If any connection exists between EE and psychiatric inheritance, it may be a weak relationship that is unlikely to result in consistent observations.

A logistic regression model did not reveal an effect of parental or family psychiatric history in predicting overall EE status. The combined effect of these variables also did not predict EE status or subtype. These results fail to support the study's core hypothesis and as previously stated may reflect inadequate measures of genetic/psychiatric variables. These findings may also further suggest the weak connection between predispositions to psychopathology and EE, as discussed above.

Expressed Emotion Beyond Psychopathology

Researchers have proposed sources of high-EE attitudes other than those tested in the present study. Most notable are the attributions and family interactions approaches. As mentioned previously, the attributions model suggests caregivers become critical or hostile when they believe that the patient can control his/her symptoms or behaviors, or that these symptoms or behaviors are characteristic of the patient rather than common experiences. There is substantial support for this hypothesis (Hooley, 1995; Hooley & Gotlib, 2000; Wendel, Miklowitz, Richards, et al., 2000). The family interactions approach to understanding EE seems to be the most systems-oriented model described thus far. Consistent with the biopsychosocial model, this approach holds that high-EE attitudes can be observed in families as a whole, rather than just one individual (e.g., the caregiver or the patient). Studies have shown increased rates of reciprocal negative interactions among high-EE families as compared to low-EE families (Simoneau, et al., 1998), suggesting that a phenotype approach to EE is overly reductionistic.

Limitations and Future Directions

This study had several limitations. First, assessments of parental and relative psychopathology were made during the same interview, over a period of approximately 45 minutes. Comparable studies used more extensive psychiatric evaluation measures over a longer interview, allowing for a more thorough diagnostic procedure and greater sensitivity and specificity. The Structured Clinical Interview for the DSM (SCID) used by Goldstein et al. (1992) and Goldstein et al., (2002) provides an example. The SCID evaluates a broader range of clinical disorders and specifies disorder subtypes; the measure also identifies Axis II disorders, which the FHS does not. Given the large number of individuals screened during the relatively short interview, the potential for type I and type II errors exists, perhaps not accurately reflecting the true psychiatric history of caregivers or their relatives.

Further, the FHS is limited for the purposes of the current study in that the timing and chronicity of documented symptoms are ambiguous. Any positive score on the measure may reflect a past diagnosis that has been treated and resolved, or a disorder that is current. The criticism/anxiety finding therefore does not suggest that EE criticism is influenced by co-occurring anxiety. Also of note is that the family history interviews were typically conducted between 6 and 18 months after intake, when the KSADS and FMSS data were collected. It is feasible that recent onsets within parents occurred between intake and family history interviews, and further, that EE status also changed during this time. In combination with the lack of reliability data among study raters for the measure, the limitations of the caregiver and family history measures suggest that the current data should be viewed with caution.

A second notable limitation lies in the use of the FMSS in evaluating EE. While variables such as time and patient or caregiver symptom remission cannot be ruled out, Goldstein (1992) found that the CFI and FMSS yielded a different EE status among several caregivers. Most caregivers who converted did so from low- to high-EE, though as discussed above, it is possible that the open-ended format of the FMSS solicits high-EE attitudes from a different subgroup of caregivers than the semi-structured interview format of the CFI. This effect may be particularly relevant to caregivers with anxiety disorders.

Third, the critical/noncritical design paired multiple subgroups together. Parents who were critical were paired with those who were critical and EOI, critical and borderline EOI, or EOI and borderline critical; EOI and low-EE parents were paired together. Though criticism has been thought to be a unique variable in the EE construct (e.g., Hooley and Gotlib, 2000), the diverse levels of criticism and overinvolvement in the critical group suggest that the criticism variable was not entirely isolated.

Fourth, the comparison of average child bipolar symptoms has several limitations. The primary limitation is that a measure of the EE/child symptoms association at a single point in time fails to capture any longitudinal change in the relationship. The previously mentioned study by Hooley et al. demonstrates that EE has state characteristics, suggesting the importance of measuring fluctuations in EE status in relation to patient symptoms, or perhaps in relation to caregiver symptoms. The current study provides no information regarding the length of the child's most recent episode, the number of years s/he has been ill, or the extent to which the child and parent's lives overlap (i.e., the child

and parent live together or do not live together). Future research should focus on the longitudinal course of EE, comparing families in treatment groups with those in controls.

A measure of manic and depressive symptoms does not capture the dimensions of psychopathology experienced by patients in the current study. Bipolar patients often have comorbid diagnoses of anxiety disorders, oppositional disorders, and ADHD. These disorders may substantially impact the way parents respond to their children's behavior and the attitudes they hold about them. Indeed, an incorporation of children's comorbid disorders into analyses may reveal that high-EE parents do in fact have more symptomatic children when comorbidities are considered.

That EE has only been measured in families who already have an ill member remains a limitation of EE research. The extent to which individuals within the general population hold high-EE attitudes toward a healthy relative may provide insight into the degree to which EE attitudes reflect characteristics of the individual himself. Rather than recruiting healthy controls for EE research, investigators may seek to better understand the family environments of the psychiatrically ill by making use of other relatives within the immediate family, such as a sibling of the patient or the spouse of the caregiver. For example, a high-EE caregiver may express low-EE attitudes toward a psychologically well sibling, suggesting the unique contribution of the patient's illness to EE. Examination of psychological health and the relationships between all immediate family members may make for a more complete picture.

In summary, the relationship between EE and individual and family history of psychopathology appears to be weak at best. However, it is important to consider that EE is likely a heterogeneous construct. This refers to the developmental trajectory of EE and

thus to both its etiology (including genetic and environmental effects) and course (including stability and severity of attitudes). What appear as counterintuitive findings with respect to EE and caregiver/family history of psychopathology may turn our attention to theoretical approaches and research designs that take into account the diversity of pathways leading to EE attitudes and their impact on patient symptomatology. The biopsychosocial and developmental psychopathology models of BD and the family climate provide a foundation for these pursuits. Studies that evaluate genetics, family functioning, member characteristics, and treatment response at different points within families' life cycles will contribute greatly to our understanding of how and why EE attitudes emerge. Further studies of the variable cognitive vulnerabilities (e.g., interpretation of social cues; McClure-Tone, 2010) among individuals with adult and adolescent BD may provide clues about patients' predispositions to be affected by high-EE attitudes, further elucidating the hypothesized bidirectional processes between caregiver and patient. The limitations and results of the current study highlight the need for interdisciplinary and longitudinal approaches to understanding the complex trajectory of families with a bipolar adolescent.

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APPENDIX

Table 1

Parental Demographics (*n* = 63)

Variable	Value
Age	
M	44.47
SD	6.09
Range	32-61
Sex	
Male	13 (20.63%)
Female	48 (79.37%)

Table 2

Patient Demographics (*n* = 56)*

Variable	Value (%)
Age	
M	14.72
SD	1.41
Sex	
Male	26 (46.4)
Female	30 (53.6)
Race	
Caucasian	50 (89.3)
African American	4 (7.1)
Native American	1 (1.8)

Asian/Pacific Islander	1 (1.8)
Biracial	0
Hispanic Ethnicity	4 (7.1)
SES	
M	46.84
SD	13.05
Living With	
Both Natural Parents	27 (48.2)
Joint Custody	2 (3.6)
1 Parent + Partner	11 (19.7)
Mother Only	14 (25)
Father Only	1 (1.8)
Grandparent	1 (1.8)
Diagnosis	
Bipolar I	26 (46.4)
Bipolar II	30 (53.6)

*SES was calculated using the Hollingshead index.

Table 3

Psychopathology of 63 parents as assessed using the FHS, based on overall EE status and critical/noncritical subtypes (df = 61)¥

Disorder	**EE Status**		**Critical/Noncritical**		**Test Statistics**			
	High (*n* = 29)	**Low (*n* = 34)**	**High* (*n* = 26)**	**Low** (*n* = 37)**	**EE *t*- Score**	**EE *p*- Value**	**Criticism *t*- *Score***	**Criticism *p*- Value**
Depression	1.79 (*1.37*)	1.42 (*1.32*)	1.88 (*1.33*)	1.39 (*1.34*)	-1.05	0.3	-1.43	0.16
Dysthymia	0.57 (*1.13*)	0.35 (*.77*)	0.52 (*1.1*)	0.41 (*.87*)	-0.9	0.37	-0.46	0.64
Mania	0.62 (*1.23*)	0.53 (*1.11*)	0.58 (*1.21*)	0.57 (*1.15*)	-0.3	0.77	-0.02	0.99
Hypomania	0.46 (*1.04*)	0.36 (*.82*)	0.32 (*.9*)	0.47 (*.94*)	-0.42	0.67	0.63	0.53
ADHD	0.93 (*1.36*)	0.5 (*.99*)	0.81 (*1.3*)	0.62 (*1.1*)	-1.45	0.15	-0.61	0.54
Conduct DO	0.18 (*.67*)	0.09 (*.51*)	0.2 (*.71*)	0.08 (*.49*)	-0.6	0.55	-0.78	0.44
Anxiety	1.54 (*1.3*)	1.12 (*1.3*)	1.72 (*1.21*)	1.03 (*1.3*)	-1.27	0.21	-2.12	0.04
Schizophrenia	0 (*n/a*)	0 (*n/a*)	0 (*n/a*)	0 (*n/a*)	*n/a*	*n/a*	*n/a*	*n/a*

Psychosis	0 (*n/a*)	0.15 (*.61*)	0 (*n/a*)	0.14 (*.59*)	1.28	0.21	1.15	0.25
Substance A	0.54 (*1.11*)	0.5 (*1.1*)	0.48 (*1.05*)	0.54 (*1.12*)	-0.13	0.9	0.21	0.83
Substance D	0.39 (*.96*)	0.38 (*.95*)	0.44 (*1.03*)	0.35 (*.92*)	-0.04	0.97	-0.36	0.72
Suicidality	0.86 (*1.3*)	0.74 (*1.3*)	0.92 (*1.35*)	0.7 (*1.24*)	-0.39	0.7	-0.67	0.51
Overall (Sum)	6.93 (*5.84*)	5.59 (*5.23*)	6.92 (*5.8*)	5.7 (*5.3*)	-0.96	0.34	-0.86	0.39

¥Mean FHS scores on a 0-3 severity scale, with standard deviations in parentheses

*This group included high-EE critical and critical/EOI subtypes

**This group included low-EE parents and high-EE, EOI subtype

Table 4

Family history data of 63 parents and their 313 first-degree relatives, based on overall EE status and critical/noncritical subtypes

Variable	**EE Status**		**Critical/Noncritical**	
	High (*n* = 29)	**Low (*n* = 34)**	**High (*n* = 26)***	**Low (*n* = 37)****
Total number of relatives	137	176	126	187
Mean number of relatives per family (*SD*)	4.72 (*2.53*)	5.33 (*2.67*)	5.04 (*2.71*)	5.05 (*2.56*)
Family history of mood DO				
Present	16	20	14	22
Absent	13	13	11	15
Family score (*SD*)	.28 (*.32*)	.17 (*.17*)	0.29 (*.28*)	0.2 (*.24*)
Test statistics				
X^2	X^2 = .19, df = 1, p = .67		X^2 = .07, df = 1, p = .78	
t	t = -1.67, df = 60, p = .1		t = -1.28, df = 60, p = .2	

*This group included high-EE critical and critical/EOI subtype

**This group included low-EE parents and those who were high-EE, EOI subtype

Table 5

Logistic regression output for the predictability of overall EE status using personal and family psychiatric history of 63 caregivers

Predictor	*B**	Wald X^2	*p*- Value
Parental psychopathology	0.99	0.84	0.36
Family mood disorder history	0.03	0.4	0.53
Model	-.57	1.88	.17

*LR coefficient

Table 6

Bipolar symptom severity of 56 adolescents using the KSADS, based on parents' overall EE status and critical/noncritical subtypes[¥]

Variable	EE Status		Critical/Noncritical		Test Statistics			
	High-EE (*n*= 28)	Low-EE (*n*= 30)	High Criticism* (*n*= 27)	Low Criticism** (*n*= 29)	EE *t*- Score	EE *p*- Value	Criticism *t*- Score	Criticism *p*- Value
MRS	29.03 (*11.16*)	26.59 (*11.81*)	23 (*10.03*)	35 (*9.41*)	-0.77	0.44	-1.18	0.24
DRS	28.14 (*9.45*)	26.19 (*9.87*)	28.3 (*10.02*)	25.63 (*9.4*)	-0.8	0.44	-1.03	0.3
Sum	57.64 (*14.95*)	51.53 (*16.32*)	58.3 (*15.96*)	51.97 (*15.47*)	-1.48	0.14	-1.5	0.13

[¥]Mean KSADS scores, with standard deviations in parentheses

*This group included high-EE critical and critical/EOI subtype

**This group included low-EE parents and those who were high-EE, EOI subtype