



## Research paper

# Developmental evaluation of family functioning deficits in youths and young adults with childhood-onset bipolar disorder



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## ABSTRACT

**Background:** Childhood-onset bipolar disorder (BD) is a serious condition that affects the patient and family. While research has documented familial dysfunction in individuals with BD, no studies have compared developmental differences in family functioning in youths with BD vs. adults with prospectively verified childhood-onset BD.

**Methods:** The Family Assessment Device (FAD) was used to examine family functioning in participants with childhood-onset BD ( $n = 116$ ) vs. healthy controls (HCs) ( $n = 108$ ), ages 7–30 years, using multivariate analysis of covariance and multiple linear regression.

**Results:** Participants with BD had significantly worse family functioning in all domains (problem solving, communication, roles, affective responsiveness, affective involvement, behavior control, general functioning) compared to HCs, regardless of age, IQ, and socioeconomic status. Post-hoc analyses suggested no influence for mood state, global functioning, comorbidity, and most medications, despite youths with BD presenting with greater severity in these areas than adults. Post-hoc tests eliminating participants taking lithium ( $n = 17$ ) showed a significant diagnosis-by-age interaction: youths with BD had worse family problem solving and communication relative to HCs.

**Limitations:** Limitations include the cross-sectional design, clinical differences in youths vs. adults with BD, ambiguity in FAD instructions, participant-only report of family functioning, and lack of data on psychosocial treatments.

**Conclusions:** Familial dysfunction is common in childhood-onset BD and endures into adulthood. Early identification and treatment of both individual and family impairments is crucial. Further investigation into multi-level, family-based mechanisms underlying childhood-onset BD may clarify the role family factors play in the disorder, and offer avenues for the development of novel, family-focused therapeutic strategies.

## 1. Introduction

Childhood-onset bipolar disorder (BD) is a complex condition affecting 1–2% of youths (Van Meter et al., 2011). Compared to individuals with late adolescent- and adult-onset BD, youths with childhood-onset BD spend more time symptomatic with mixed depressive and manic presentations, rapid mood fluctuations, and subthreshold symptoms (Birmaher et al., 2009; Birmaher et al., 2014; Geller et al., 2008). These youths also have greater functional impairment (Perlis et al., 2009), poorer quality of life (Perlis et al., 2009), and higher risk for suicidality (Perlis et al., 2004). In addition, childhood-onset BD often persists into adulthood, leading to further impairment and negative outcomes (Axelson et al., 2011; Birmaher et al., 2009;

Birmaher et al., 2014; Geller et al., 2008; Leverich et al., 2007). Given the enduring nature of this disorder, there is a critical need for studies to directly evaluate developmental effects by aggregating data from children, adolescents, and adults in order to examine the phenomenology and mechanisms of BD across the lifespan, and thereby enhance diagnosis and treatment efforts.

Family functioning is one such process relevant to BD and important to understand from a developmental perspective, as findings could indicate optimal family involvement in treatment and age-specific intervention targets. In addition to the patient, families of individuals with childhood-onset BD are quite impaired. Compared to healthy controls (HCs) and youths with other psychiatric conditions, families of youths with BD display high levels of conflict, control, aggression, quarreling,

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forceful punishment, tension, stress, and negative expressed emotion; and low levels of warmth, affection, intimacy, cohesion, expressiveness, organization, and positive expressed emotion (Belardinelli et al., 2008; Keenan-Miller et al., 2012; Nader et al., 2013; Perez Algorta et al., 2017; Schenkel et al., 2008). Family dysfunction also predicts worse course of BD in youths, including: 1) low maternal warmth (Geller et al., 2008); 2) chronic stress in family, romantic, and peer relationships (Kim et al., 2007; Siegel et al., 2015); 3) frequency and severity of stressful life events (Kim et al., 2007); 4) low levels of cohesion and adaptability (Sullivan et al., 2012); and 5) high levels of conflict (Sullivan et al., 2012). This relationship is also bidirectional, with patients' symptoms/behaviors reciprocally influencing caregivers' burden/distress (Reinares et al., 2016b). Thus, psychosocial evidence-based treatments (EBTs) for childhood-onset BD incorporate family-based strategies including psychoeducation, communication, problem solving, and affect regulation to address these impairments (Fristad and MacPherson, 2014).

Familial caregivers (e.g., parents, spouses, close relatives) of adults with BD display comparable dysfunction, including low levels of cohesion, expressiveness, and organization; and high levels of conflict (Miklowitz, 2011; Miklowitz and Johnson, 2009; Reinares et al., 2016a; Solomon et al., 2008; Weinstock et al., 2006). In addition, high expressed emotion (Kim and Miklowitz, 2004; Yan et al., 2004) and familial negative affective style (O'Connell et al., 1991) predict recurrence in adults with BD. However, no research has examined the persistence of family dysfunction into adulthood among individuals with childhood-onset BD. One study demonstrated that adults with retrospectively obtained childhood-onset BD experienced sustained psychosocial/functional impairment during prospective observation on a measure that assessed work, relationships (including family), recreation, and life satisfaction (Perlis et al., 2009). Though, family functioning in particular was not assessed in this study, and determination of childhood-onset BD diagnoses may have been influenced by retrospective recall bias (Leboyer et al., 2005). Importantly, no studies have directly compared family functioning in youths with BD vs. adults with prospectively verified childhood-onset BD (youth participants with BD followed into adulthood).

Unfortunately, research is often artificially bifurcated by regulatory requirements or investigator expertise/training in pediatrics or adults, and few datasets have prospectively established childhood-onset BD (Birmaher et al., 2009; Geller et al., 2008). These limitations make it challenging to evaluate developmental differences in mechanisms and processes implicated in childhood-onset BD. In addition, no studies have specifically examined the developmental progression of familial dysfunction in this condition, despite its relevance to onset and course of the disorder (Geller et al., 2008; Kim et al., 2007; Reinares et al., 2016b; Siegel et al., 2015). Importantly, parent and family variables also influence psychosocial treatment outcomes in childhood-onset BD, serving as both moderators (Miklowitz et al., 2009; Sullivan et al., 2012; Weinstein et al., 2015) and mediators (MacPherson et al., 2016; Mendenhall et al., 2009). Thus, enhanced understanding of family processes in childhood-onset BD is crucial from both a phenomenological and intervention perspective.

To address gaps in the literature and better conceptualize familial dysfunction across development, the current study examined family functioning in youths with BD, adults with prospectively verified childhood-onset BD, and youth and adult HCs. Adults with BD were followed since childhood via their participation in the Brown University site of the Course and Outcome of Bipolar Youth (COBY) study to ensure that retrospective recall bias did not impact BD diagnoses (Birmaher et al., 2009; Leboyer et al., 2005). Hypotheses were based on research documenting a more severe course of illness and functional impairment in youths vs. adults with BD (Birmaher et al., 2009; Geller et al., 2008; Perlis et al., 2009; Perlis et al., 2004). In addition, youths likely had less time to seek treatment and develop strategies for managing symptoms/stressors than adults with childhood-onset BD,

given longer duration of illness in the latter, potentially contributing to exacerbated family dysfunction at younger ages. Thus, it was hypothesized that: 1) youths and young adults with childhood-onset BD would demonstrate impaired family functioning compared to HCs; and 2) youths with BD would display worse family functioning compared to adults with childhood-onset BD.

## 2. Method

### 2.1. Participants and procedures

Participants were enrolled in one of two studies approved by the Institutional Review Boards of Bradley Hospital and Brown University. Written informed parental consent and child assent were obtained for youths; written informed consent was obtained for adults. Subsequently, parents, youths, and adults completed assessments and measures cross-sectionally. The sample included 116 individuals with childhood-onset BD (70 youths, 46 adults) and 108 HCs (46 youths, 62 adults).

Inclusion criteria for participants with BD were: 1) ages 7–17 (youths) or 18–30 (adults); 2) English fluency; and 3) diagnosis of BD per the *DSM-IV-TR*. Youths with BD were recruited for a study that compared youths with BD vs. HCs, and were required to have BD-I ( $n = 68$ ) or BD-II ( $n = 2$ ). Adults with BD were originally enrolled as youths in the Brown University site of the COBY study, prior to enrolling in the current study. The COBY study required a diagnosis of BD-I ( $n = 28$ ), BD-II ( $n = 3$ ), or BD-Not Otherwise Specified (NOS) ( $n = 15$ ); the latter was defined as elation plus two associated symptoms or irritability plus three associated symptoms, change in functioning,  $\geq 4$  h within a 24 h period,  $\geq 4$  cumulative lifetime days (Birmaher et al., 2006). Thus, adults' diagnoses of childhood-onset BD were prospectively confirmed. Exclusion criteria were: 1) full scale IQ (FSIQ)  $< 70$  on the Wechsler Abbreviated Scale of Intelligence (WASI) (Wechsler, 2005); 2) autism spectrum disorder or primary psychosis; and 3) medical/neurological conditions potentially mimicking BD.

Inclusion criteria for HCs were: 1) ages 7–17 (youths) or 18–30 (adults); 2) English fluency; and 3) no current/lifetime psychiatric illness or substance abuse/dependence in participants or first-degree relatives. Exclusion criteria were: 1) FSIQ  $< 70$ ; 2) learning disorders or autism spectrum disorder; and 3) serious, non-psychiatric medical disorders potentially mimicking/confounding psychiatric illness.

### 2.2. Measures

#### 2.2.1. Family functioning

Current family functioning was assessed via youth/adult participant report on the Family Assessment Device (FAD) (Epstein et al., 1983), consisting of 60 items and seven subscales: 1) Problem Solving—family's ability to address problems adaptively; 2) Communication—style and clarity of verbal information sharing; 3) Roles—established behavior patterns to fulfill family functions; 4) Affective Responsiveness—family members' ability to experience appropriate affect across situations; 5) Affective Involvement—interest and value placed on family members' behaviors and concerns; 6) Behavior Control—the way family members maintain expectations for each other; and 7) General Functioning—overall summary of family processes. Items are averaged to produce a summary score for each subscale ranging from one to four; higher scores indicate poorer functioning, and scores above two suggest clinical severity. The FAD has demonstrated good reliability and validity across ages and populations (Miller et al., 1985; Pritchett et al., 2011; Staccini et al., 2015; Youngstrom et al., 2011).

#### 2.2.2. Demographic information

Parents reported on youths' age, race, current medications, and socioeconomic status (SES), categorized according to the Hollingshead Index (Hollingshead, 1975). Adult participants reported on these

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