Social Functioning in Patients with Early-Onset Bipolar Disorder and Healthy Controls

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Social Functioning in Patients with Early-Onset Bipolar Disorder and Healthy Controls

by

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Department of Psychology and Neuroscience

2013
This thesis entitled:
Social Functioning in Patients with Early-Onset Bipolar Disorder and Healthy Controls
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The final copy of this thesis has been examined by the signatories, and we find that both the content and the form meet acceptable presentation standards of scholarly work in the above mentioned discipline.

IRB protocol # 11-0646
BACKGROUND: Early-onset bipolar disorder (BD), typically defined as illness onset occurring prior to late adolescence, is associated with a broad range of significant psychosocial impairments. The current study sought to characterize social functioning among patients who had previously participated in a randomized controlled trial of family focused treatment.

METHODS: Thirty BD subjects (mean age = 19.07; female = 62%) and 30 healthy adolescent controls (mean age = 17.50; female = 60%) completed interviews and a battery of questionnaires assessing social functioning and psychiatric symptoms. BD participants’ current data were compared to social functioning, affective symptom, and family functioning data collected during intake assessments for the treatment study.

RESULTS: BD participants reported poorer social functioning than controls across all study measures. Levels of depression predicted use of social skills; social functioning was otherwise independent of concurrent affective symptoms. Intake measures of social functioning did not predict current social impairment; teen- and parent-reported family cohesion at study intake significantly predicted current social skill use. Finally, depression at study intake, and not prior measures of social functioning, predicted current depression.

CONCLUSIONS: Those with early-onset BD lag behind their healthy peers in social functioning, suggesting the importance of interpersonal impairment as a treatment target with this population. Future longitudinal studies are needed to gain a more precise understanding of the relationship between social functioning and family variables.
“The human mind, no matter how highly trained, cannot grasp the universe. We are in the position of a little child, entering a huge library whose walls are covered to the ceiling with books in many different tongues. The child knows that someone must have written those books. It does not know who or how. It does not understand the languages in which they are written. The child notes a definite plan in the arrangement of the books, a mysterious order, which it does not comprehend, but only dimly suspects.”

Albert Einstein, 1930
I am incredibly grateful for the guidance of my graduate advisor, Dr. David Miklowitz, who has supported my professional development and increased my confidence - as both a clinician and researcher - with creativity, intelligence, and wit. He has inspired me to pursue my goals throughout graduate school; I am sincerely appreciative for his encouragement to find my muse and ‘go after it.’

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CHAPTER I

INTRODUCTION

Bipolar disorder (BD) is characterized by periods of elevated, energized mood and behavior, alternating or coexisting with periods of depression. Approximately 28% of adults with BD report the onset of symptoms before the age of 13, and approximately 66% prior to the age of 18 (Perlis et al., 2004). Early-onset BD, typically defined as illness onset prior to late adolescence, is often characterized by rapid cycling between moods, multiple comorbidities (for example, additional anxiety or behavioral diagnoses), and other treatment-resistant presentations of the illness (Geller, Craney, Bolhofner, Nickelsburg, Williams & Zimerman, 2002). Early-onset symptoms have been associated with worsened adult outcome (Perlis et al., 2004), and put youth at serious risk for a range of functional deficits (Miklowitz, Biuckians & Richards, 2006).

A growing literature suggests that youth with BD demonstrate particular difficulty with the establishment of close peer relationships (e.g., Geller et al., 2000; Goldstein et al., 2009; McClure et al., 2005; Youngstrom, Youngstrom & Starr, 2005; for summary, see Keenan-Miller & Miklowitz, 2011).

The successful development and maintenance of peer relationships during adolescence is a crucial milestone of healthy development, reflecting adaptive psychological adjustment and establishing a framework for continued personal growth (Sullivan, 1953). Research suggests that a successful transition from childhood to adolescence, which centers largely on a shifting of importance from family to peer relationships, results in the individuation of the adolescent (e.g., Brechwald & Prinstein, 2011; Helsen, Vollebergh & Meeus, 2000; Savin-Williams & Berndt,
acquisition of important social behaviors (Brechwald & Prinstein, 2011; Oberle, Schonert-Reichl & Thomson, 2010; Savin-Williams & Berndt, 1990), and the development of a broad social knowledge and insight (Brechwald & Prinstein, 2005; Oberle, Schonert-Reichl & Thomson, 2010; Prinstein & La Greca, 2004; Stevens & Prinstein, 2005). More recently, neurobiological research has supported the view that adolescence is a period of particular significance regarding the social development of the individual (e.g., Nelson, Leibenluft, McClure & Pine, 2005), similar to the language acquisition period experienced during infancy (Blakemore & Choudhury, 2006).

If social development through this period is stunted or incomplete, or healthy reciprocal peer relationships are not maintained, there may be significant consequences that extend into adulthood. Sullivan (1953) posited that these might include increased anxiety, a decreased sense of self-worth, and difficulty with reaching fully-developed maturity. Adolescents with BD should be at particularly high risk for impaired social functioning, as characteristics of early-onset BD – frequent symptoms of irritability and grandiosity, high rates of comorbidity, rapid cycling, and extended periods of subsyndromal symptoms (Geller et al., 2000; Sala, Axelsson & Birmaher, 2009; West & Pavuluri, 2009; Wilens et al., 2003; Wozniak, Biederman, Mundy, Mennin & Faraone, 1995) – are likely to interfere greatly with social development. While these symptoms could contribute directly to interpersonal difficulties, mood fluctuations are also likely to interfere with the development of a stable sense of self (Crowe, Inder, Joyce, Moor, Carter & Luty, 2008), adding additional challenge to relationship development. The current study was designed to enhance our understanding of social functioning in individuals with early-onset BD, as well as the factors that may contribute to these deficits.
Social Impairment in Early-Onset Bipolar Disorder

A growing research literature indicates that the majority of adolescents with BD do demonstrate significant deficits in academic and social functioning (Lewinsohn, Klein & Seeley, 1995; Hlastala, Kotler, McClellan & McCauley, 2010). The social impairment of early-onset BD has been characterized as significant and persistent (e.g. Goldstein, Miklowitz & Mullen, 2006; Goldstein et al., 2009; Geller, Warner, Williams & Zimerman, 1998; Geller et al., 2000; McClure et al., 2005; Youngstrom, Youngstrom & Starr, 2005). Poorer social functioning has been associated with greater experience of depressed symptoms (Altshuler et al., 2006; Kim, Miklowitz, Biuckians & Mullen, 2007; Weinstock & Miller, 2008), increased speed of symptom recurrence (Gitlin, Swendsen, Heller & Hammen, 1995), and higher rates of non-suicidal self-injury (Esposito-Smythers et al., 2010).

A number of studies have assessed relationships between social impairment and patient variables among those with early-onset BD, in both clinical and community samples. In an investigation of adolescent BD characteristics in a community sample, Lewinsohn, Klein and Seeley (2000) found that adolescents with a BD diagnosis or subthreshold BD symptoms demonstrated significantly greater psychosocial problems, completed fewer years of education, utilized mental health services more frequently, and had poorer global functioning than individuals without a psychiatric diagnosis. These deficits were significant, despite the fact that their sample of adolescents with fully-diagnostic or subthreshold BD was higher functioning (e.g. consistently attending high school) than those typically participating in BD research.

DelBello et al.’s (2007) longitudinal study examined the syndromatic, symptomatic, and functional recovery of 72 adolescents with BD I who were hospitalized as a result of their first manic or mixed episode. Follow up assessments were completed for a year following hospital
discharge. Only 39% of the sample achieved ‘functional recovery’, defined as a return to premorbid functional levels. Regarding specific domains of functioning, adolescents were more likely to recover in the areas of role performance, recreation, or sexual activities than in the area of interpersonal relationships. The authors report that adolescent functioning was independent of other study variables, including syndrome and symptom measures.

Goldstein et al. (2009) demonstrated an association between early-onset BD and mild to moderate psychosocial impairment in the areas of academics, interpersonal, and global functioning. Although the BD youth participating in this study (N=446, age 7-17 years) did report good recreational functioning, they endorsed significant levels of overall life dissatisfaction. Deficits were greatest during mood episodes, although impairment persisted during inter-episode periods as well. BD diagnosis (i.e., BD I, BD II, or BD NOS) did not influence level of social impairment. Functional impairment was greatest in those with comorbid diagnoses (ADHD, ODD, CD), psychotic symptoms, greater mood symptom severity, and for adolescent (as compared to child) patients, regardless of age of onset.

A number of studies have examined specific components of social functioning in early-onset BD populations. In a study by McClure et al. (2005), adolescents with BD demonstrated functional impairment in a number of tasks measuring aspects of teens’ social cognition. In assessments that required participants to flexibly generate appropriate responses to a variety of social situations, adolescents with BD performed significantly worse than age-, gender-, and IQ-matched controls. In addition, adolescents with BD made significantly more errors than controls in timed facial expression recognition tasks. Performance on social cognitive tasks correctly classified more than 85% of study participants as either BD or healthy controls. Interestingly, the
impaired performance of BD adolescents was unaffected by mood state (i.e. euthymic or symptomatic) or comorbidity status.

Goldstein et al. (2006) also isolated components of social functioning, separately examining social skills knowledge and social skills performance in early-onset BD. Contrasting with McClure et al.’s findings detailed above (and other similar findings, e.g. Scott, Stanton, Garland & Ferrier, 2000), minimally symptomatic adolescents with BD had levels of social skill knowledge comparable to that of healthy control subjects, suggesting that the acquisition, processing, and retention of social information was not impaired in the BD group. Although the groups were also similar in observable social skills performance during study interviews, adolescents with BD demonstrated significant skill impairment by self- and parent-report. The authors conclude that, as the self- and parent-report measures assessed a range of behavior not likely to occur during examiner interviews (e.g., teasing others, yelling), social deficits in BD adolescents may be specifically elicited in high-intensity or emotionally-charged situations requiring greater levels of emotion regulation.

Empathic understanding, or ‘theory of mind’ (TOM), may also be impaired in early-onset BD. Schenkel, Marlow-O’Connor, Moss, Sweeney & Pavuluri (2008) demonstrated impaired performance of youth with BD on TOM tasks measuring social inference and perspective-taking, particularly in emotionally-valenced situations (e.g., a negative storyline). Greater task deficits were associated with increased mania levels, earlier age of onset, and severity of youth diagnosis (i.e., BD I compared to BD II).

Finally, several studies have investigated neurobiological correlates of social functioning in early-onset BD. Structural cortical asymmetries are evident in youth with BP I, in areas such as the left ventrolateral prefrontal cortex, superior temporal gyrus, and middle and inferior
temporal gyri, which are distinct from structural abnormalities associated with early-onset schizophrenia (Gogtay et al., 2007). There is some suggestion that neuronal density may be lower in early-onset BD, specifically within the right dorsolateral prefrontal cortex (Chang, Adleman, Dienes, Barnea-Goraly, Reiss & Ketter, 2003).

Regarding functional neurobiological findings, a number of studies have examined activation patterns relating to facial affect recognition in early-onset BD (e.g., Pavuluri, O’Connor, Harral & Sweeney, 2007; Rich, Grimley, Schmajuk, Blair, Blair & Leibenluft, 2008; Rich, Fromm, Berghorst, Dickenstein, Brotman, Pine & Leibenluft, 2008; Schenkel, Pavuluri, Herbener, Harral & Sweeney, 2007). Research consistently suggests that adolescents with BD, as well as those with severe mood dysregulation (SMD; Leibenluft, Charney, Towbin, Bhangoo & Pine, 2003b), demonstrate difficulty with the recognition of facial expressions. These deficits are generally associated with abnormal functioning in the amygdala, posterior cingulate, dorsolateral prefrontal cortex, and ventrolateral prefrontal cortex – areas associated with the experience and regulation of emotion. There is some evidence that, if these facial affect recognition deficits (and, subsequently, neurobiological activation abnormalities) are not unique to early-onset BD, they are more pronounced as compared to youth with other psychiatric diagnoses (e.g., anxiety or ADHD; Guyer et al., 2007). For a further review of neurobiological findings, see McClure-Tone, 2009.

**Social Functioning Deficits in Teens with Bipolar Disorder versus Other Psychiatric Disorders**

Accumulating evidence suggests that these social deficits associated with early-onset BD are greater than those associated with other psychiatric illnesses. In a study of clinically-referred children under 12 years of age, Biederman and colleagues (2004) found that broadly-defined
psychosocial deficits experienced by children with BD were greater than those experienced by children diagnosed with ADHD. Similarly, Geller, Warner, Williams and Zimerman (1998) demonstrated greater levels of aggression, social withdrawal, and lower levels of social competence, in children with BD as compared to peers with ADHD. Furthermore, in Geller et al.’s comparison (2000) between children with BD or ADHD, teens with BD more frequently endorsed that they have few or no friends, experience regular teasing, and demonstrated impaired social skills.

Studies utilizing the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983; Achenbach, 1991; Achenbach et al, 1991) have suggested that a combination of difficulties in aggressive behavior, attention, delinquent behavior, and social problems are unique to early-onset BD, and that problems in these areas are more significant for adolescents with BD than difficulties experienced by children and adolescents with other psychiatric diagnoses such as ODD, ADHD, MDD, or PTSD (Youngstrom, Youngstrom & Starr, 2005). Although there is some evidence that the social functioning of adults with BD is superior to that of adult schizophrenic patients (Eckholdt & Lenzenweger, 1990; Bartels, Mueser & Miles, 1997), these comparisons have not been completed with adolescent samples.

**Relationship between Social Functioning and Patient Characteristics**

An extensive literature suggests the influence of gender on peer relationships in childhood and adolescence (see Rose & Rudolph, 2006 for a recent review). Overall, girls are more likely than boys to demonstrate prosocial behavior, engage in social conversations, self-disclose in friendships, and report emotional closeness and trust in peer relationships. These gender differences are noted as early as preschool, and become more pronounced throughout development into adolescence (Rose & Rudolph, 2006). These studies have been completed with
normative populations; the effect of gender on peer relationships for individuals with early-onset BD is less clear.

Goldstein (2006) reported superior clinician-scored social performance by females with BD during a brief interview, although gender differences among these participants were not reflected in levels of social skills knowledge or self-reported use of social behaviors. DelBello and colleagues (2007) reported that males were more likely than females to experience symptomatic recovery – although not functional recovery – following experience of a manic or mixed episode. In a study of adolescents and young adults at high risk for developing bipolar disorder (i.e., parents with a bipolar diagnosis), gender differences were seen such that high-risk male subjects were more quarrelsome and less agreeable than high-risk female participants; such gender differences were not noted in the low-risk (i.e., no parent with bipolar diagnosis) comparison group (Lennen, Rot, Ellenbogend & Young, 2009).

Many studies, however, do not show gender-based differences in BD adolescents’ peer relationships (e.g., Carlson, Bromet, Driessens, Mojtabai & Schwartz, 2002; Goldstein et al., 2009; Keenan-Miller et al., 2011). A large study by Biederman and colleagues (2004) did not find gender differences in measures of BD adolescents’ school functioning, GAF scores, or social adjustment, although peer-specific interaction was one of many components included in compared scores. Further complicating interpretation of the literature, many studies of social functioning with BD populations do not report specific examination of gender effects (e.g., Lewinsohn et al., 2000; McClure et al., 2005; McClure et al., 2003; Pavuluri et al., 2008; Rich et al., 2008b).

As detailed above, findings have also been mixed regarding the associations between adolescent social functioning and patient diagnostic characteristics, such as BD diagnosis (i.e.,
BD I, BD II, BD NOS) or comorbidity status. The literature also includes conflicting findings regarding the influence of a patient’s age of illness onset. Kutcher, Robertson and Bird (1998) suggested that adolescents’ academic and social functioning was within the normal range prior to their first bipolar episode. Quackenbush, Kutcher, Robertson, Boulos & Chapan (1996) found a significant deterioration in peer relationships following the onset of early-onset BD. Together, these findings may suggest that a later age of BD onset allows for a longer period of pre-morbid, healthy social development, and we would expect superior social functioning in those with later age of BD onset. This hypothesis has been supported by some (e.g., Schenkel et al., 2008; Weinstock & Miller, 2008), but not all (Goldstein et al., 2006; Goldstein et al., 2009), prior research.

It remains unclear how mood symptoms relate to social functioning in early-onset BD (Keenan-Miller & Miklowitz, 2011). Thus far, findings have been mixed, with some studies demonstrating independence between social functioning and mood symptoms (e.g., DelBello et al., 2007; McClure et al., 2005), while others suggest that social impairment is significantly decreased in the absence of bipolar symptoms (e.g., Altshuler et al., 2006; Goldstein et al., 2009 [manic symptoms only]; Schenkel et al., 2008). Most prior research has viewed social impairment as the result of – rather than a risk factor for – worsened mood symptoms in early-onset BD. As the majority of this research has been cross-sectional, however, study designs have prevented the ability to determine the direction of this relationship.

Recently, studies completed by Weinstock and Miller (2008, 2010) have suggested that psychosocial impairment may also be a predictor of bipolar symptoms – in particular, bipolar depression. In one study with adult patients with BD I (Weinstock & Miller, 2008), social functioning, as assessed by the UCLA Social Attainment Survey (UCLA-SAS; Goldstein, 1978),
and bipolar symptoms were measured at baseline and four-month follow up. Interestingly, symptom and social functioning variables were unrelated at baseline; additionally, symptom scores at baseline did not predict symptom levels at follow up. Instead, after controlling for baseline depression scores, social functioning predicted depressed symptom severity at follow up. More specifically, greater impairments in functioning in romantic relationships and involvement in organized activities were associated with increased depressive severity; functioning with peers was not predictive of depression. Manic symptoms were unrelated to social functioning.

In a second study (Weinstock & Miller, 2010), the authors tested the ability of several psychosocial variables, collected at study intake, to predict symptom severity at one-year follow up in a sample of adults with BD. Controlling for baseline symptom levels, social support uniquely predicted patients’ depression levels; social support remained a significant predictor when amount of maintenance treatment (e.g., additional family sessions, psychiatrist appointments) was included in statistical models. Baseline family functioning and social functioning (measured with the UCLA-SAS), in contrast, were unrelated to depression scores at follow up. Again, measures of social functioning were unrelated to manic symptoms. The authors posit that family interventions aimed at improving functioning in the home may address only one of a variety of important social variables for the BD patient.

In combination, studies authored by Weinstock and Miller suggest that BD symptoms and social impairment have a more reciprocal relationship than typically described in the literature. Namely, that while BD symptoms may negatively influence social functioning, social impairment in turn may also serve as a risk factor for continued BD symptoms. These findings, however, are yet to be replicated in early-onset BD samples.
Impairment Specific to Peer Relationships

While the literature consistently suggests that adolescents with BD experience notable social impairment, less is known about the characteristics of deficits specific to adolescent peer relationships. With few exceptions (e.g., Geller et al., 1998; Geller et al., 2000; Goldstein et al., 2006; Goldstein et al., 2009), prior research typically includes measures of overall social functioning, which combine assessments of multiple facets of the adolescent’s life (e.g., school performance, occupational functioning, participation in extracurricular activities, family relationships). Other studies use analogs for social functioning (e.g., spoken language assessments; McClure et al., 2005), or assessments of cognitive functioning that contribute to social functioning (e.g., facial affect recognition, Rich et al., 2008a). Neither broad nor indirect measures of social functioning allow for a detailed study of the quality of peer relationships in early-onset BD. In addition, control groups have not been utilized to directly compare social peer functioning between adolescents with and without bipolar disorder. Population norms are typically used, which designate social functioning as generally adaptive or impaired (e.g., Youngstrom, Youngstrom & Starr, 2005). The specific nature of peer social functioning deficits, as well as how particular these deficits may be to the early-onset BD diagnosis, remains unknown.

Social Impairment with Peers versus Family Functioning

Accumulating evidence suggests that family functioning may be correlated with symptom severity in early-onset BD. High levels of expressed emotion (EE; critical, hostile, or emotionally overinvolved attitudes) among caregivers, negative affective communication styles, and low levels of maternal warmth have all been associated with more severe mood disorder symptoms in youth (e.g., Asarnow, Tompson, Woo & Cantwell, 2001; Birmaher, Ryan &
Williamson, 1996; Dietz et al., 2008; Geller, Tillman, Bolhofner & Zimerman, 2008; Silk, Ziegler, Whalen, Dahl, Ryan, Dietz et al., 2009). Families of patients with BD report lower levels of family cohesion and adaptability and higher levels of conflict than families of healthy children or population norms (Belardinelli, Hatch, Olvera, Rene, Fonseca, Caetano et al., 2008; Robertson, Kutcher, Bird & Grasswick, 2001; Sullivan & Miklowitz, 2010). Several studies find that maladaptive levels of family functioning characterize youth who are genetically predisposed to BD (Chang, Blasey, Ketter & Steiner, 2001; Du Rocher Schudlich, Youngstrom, Calabrese & Findling, 2008; Romero, DelBello, Soutullo, Stanford & Strakowski, 2005).

Frequently, adolescent social functioning with family members and peers are treated as one and the same. These social groups, however, have differing importance to the developing adolescent, especially during late adolescence and early adulthood (e.g., Brechwald & Prinstein, 2011; Helsen et al., 2000; Savin-Williams & Berndt, 1990). In addition, the increased rates of familial psychopathology associated with early-onset BD are unlikely to influence peer relationships, but likely effect social functioning in the home environment (Keenan-Miller & Miklowitz, 2011). When studied as separate variables, evidence suggests that an adolescent’s level of social functioning can differ meaningfully between peer and family groups (Helsen et al., 2000; Robertson et al., 2001; Weinstock & Miller, 2010). As such, it would be useful for future research to consider adolescent social functioning with family members and peers separately.

In sum, these findings suggest the importance of social impairment with peers as a focus of further study. Although there is evidence that those with early-onset BD experience broad, uniquely severe psychosocial deficits, the specific characteristics of social impairment, especially regarding interactions with peers, remain largely unknown. Control group comparisons are
required to articulate the specific areas of social impairment experienced by patients with early-onset BD. Studies of early-onset BD social impairment have typically included child and adolescent populations; presently, research has not extended across developmental periods to include young adults with BD. Relatedly, the endurance of these deficits (i.e., whether social impairment with peers remains stable throughout adolescence and into early adulthood), as well as the factors that influence social functioning with peers (e.g., patient characteristics, affective symptom severity) also merit further investigation. Differentiation between functioning with peers and family is likely to be important in the study of social deficits in early-onset BD, given developmental milestones typically achieved during this timeframe. Finally, investigation of the power of social functioning variables to predict BD symptoms has not included adolescent or young adult populations. Cross-sectional and longitudinal studies are needed to effectively address these questions regarding social functioning in early-onset BD, and inform treatment strategies for this population.

**Current Study**

The aim of the current study was to characterize social functioning with peers among adolescents and young adults with early-onset BD, and examine variables that may relate to that functioning. Recruited BD participants had previously participated in a two-year randomized controlled trial of family-focused treatment for adolescents (FFT-A; Miklowitz et al., 2008); participants had completed either the 21-session FFT-A treatment protocol (focused on psychoeducation, communication skills training, and problem solving strategies) or a 3-session enhanced care (EC) condition (including psychoeducation and relapse prevention strategies). BD participants enrolled in this treatment study an average of 3.5 years prior to completion of the present study.
Specifically, we compared the social skills and peer relationships of adolescents and young adults with early-onset BD to those of healthy control subjects. The concurrent relationships between social functioning with peers and affective symptoms were also explored within these groups. For individuals with early-onset BD, we also compared measures of patients’ current social functioning to measures of social functioning, family functioning and affective symptoms collected during intake assessments for the original FFT-A treatment study. Finally, we examined the hypothesis that measures of social functioning collected at FFT-A study intake would predict current affective symptoms within our BD sample. Specific study aims are outlined below.

**Specific Aims**

**Specific Aim #1: Group Comparison of Social Functioning Variables**

We will make comparisons between the social functioning of 30 individuals with early-onset BD and the social functioning of 30 age- and gender-matched healthy control subjects.

**Hypothesis 1.** Relative to controls, participants with early-onset BD will report greater difficulties in specific social skill areas relevant to peer relationships: assertiveness, impulsivity, overconfidence, jealousy and withdrawal, and social appropriateness.

**Hypothesis 2.** Relative to controls, participants with BD will show more impairment in social functioning with peers, as reflected by decreased involvement in peer-related activities and a lower quantity of relationships (friendships and romantic relationships).

**Specific Aim #2: Concurrent Predictors of Social Functioning**

We will investigate concurrent relationships between participants’ social functioning with peers and affective symptoms.
**Hypothesis 3.** Participants’ symptoms of depression and mania will predict concurrent social functioning with peers, such that more severe symptom scores will predict greater levels of social impairment with peers.

**Specific Aim #3: Longitudinal Predictors of Social Functioning**

Within our sample of individuals with BD, we will examine the relationships between social functioning with peers during the current assessment and prior measures of social functioning, affective symptoms, and family functioning, as assessed during patients’ intake assessments for the randomized control trial of FFT-A (collected on average 3.5 years prior to the current study).

**Hypothesis 4.** Patients’ intake measures of social functioning with peers will predict current social functioning, such that greater levels of social impairment at intake will predict greater levels of current social impairment with peers.

**Hypothesis 5.** Patients’ intake measures of family functioning will predict current social functioning, such that greater levels of family functioning impairment at original treatment study intake will predict greater levels of current social impairment with peers.

**Specific Aim #4: Social Functioning at Intake as Predictor of Current Symptoms**

Within our sample of individuals with BD, we will investigate the relationships between measures of symptoms and social functioning, measured an average of 3.5 years earlier during intake assessments for the randomized control trial of FFT-A, and current affective symptoms. Specifically, we will test the hypothesis suggested by Weinstock and Miller (2008, 2010): intake
psychosocial functioning, when covaried with concurrently-collected levels of bipolar depression, will predict current levels of bipolar depression.

**Hypothesis 6.** Patients’ intake scores of social functioning, after covarying intake levels of depressive severity, will predict current depressive severity. Specifically, greater levels of social impairment at treatment study intake will predict increased severity of depression.
Participants

Subjects were recruited into either a patient or control subject group. Recruited patients with BD were prior participants in Dr. Miklowitz’s study of FFT for Bipolar Adolescents (FFT-A; R01-MH073871). In total, we recruited 30 adolescents and young adults from the 56 participants who were initially enrolled in the FFT-A treatment study (mean age =19.07 years; age range=15-23 years; 62% female; see Results section for additional information). Patients were in a variety of clinical states when assessed for the current study; participants with BD had completed treatment and follow-up assessments for the FFT-A treatment study prior to participation in the present study.

An additional 30 healthy subjects were recruited to serve as a healthy control group (mean age=17.50 years; age range=14-22 years; 60% female; see Results section for additional information). Participants were excluded from the control group if they met diagnostic criteria for any DSM-IV Axis I affective disorder (e.g., major depressive disorder, bipolar disorder, etc.; additional details below).

Procedures

Healthy control participants were recruited via email listserves (e.g., campus announcements sent to the University of Colorado (CU) faculty and staff), and advertisements posted throughout the CU department of Psychology and Neuroscience. Two potential control subjects endorsed having an affective psychiatric disorder prior to completing the study (e.g.,
major depressive disorder); they were not scheduled to complete a study assessment and were offered community referrals. Control participants who met diagnostic criteria for any DSM-IV disorder during completion of the study were offered a consultation with Ms. Sullivan as well as community referrals.

To recruit patient participants, we first sent a letter addressed to the adolescent or young adult with BD and his or her family. This letter informed them of the current research study, and indicated that a study researcher would directly call a parent within the family (if the adolescent was younger than 18 years) or the patient (if he/she was 18 years or older) in the near future. A separate page was included with the letter, allowing the parent(s) and/or patient to indicate (with a check mark) interest or lack of interest in the study. They were instructed to fill out this sheet and return it to the investigator in the provided stamped, addressed envelope. If the parent(s) and/or adolescent indicated no interest in the investigator’s study, then they were not contacted again for the present research.

If no response was received from the family, or a response was received that indicated their interest, the adolescent or young adult with BD was contacted by phone. On this call, the current study was discussed and the investigator gauged the potential participant’s interest in the project. If the patient (or, where appropriate, parent) was not interested in the study, then the researcher thanked them for their time and did not contact them further. 55 previous participants in the FFT-A study were contacted (1 participant’s family was not contacted because the patient was deceased). Ultimately, 34 patients expressed interest in participating in the current study, 10 declined, and 11 were unable to be contacted. Of the 34 patients who initially endorsed interest in the study, 30 completed the study; 4 patients later changed their mind or were unable to be contacted following their expression of interest.
When an adolescent patient or control subject expressed interest, he or she was encouraged to visit the Miklowitz Laboratory in the Center for Innovation and Creativity at the University of Colorado at Boulder. When parents and adolescents were unable to travel to the laboratory because of extreme financial or circumstantial hardship, then verbal permission was attained from the participant (and parent(s), when appropriate) to complete the interview over the phone. Prior to attaining consent, families were given a thorough explanation of study procedures, and they were able to ask any questions they may have had about the study process. The experimenter also made it clear that participants could withdraw from study participation at any time without penalty. Consent was attained from any patients over the age of 18. Adolescent patients under the age of 18 were asked to provide their assent; consent was attained from their parent(s). For assessments completed over the phone, appropriate consent and assent forms were be sent (mailed, faxed, or emailed) to the family. Once consents were signed and returned to the investigator, the assessment was scheduled and completed over the phone.

Assessments for patient and control groups included interview and questionnaire measures of social functioning, mood, and other symptoms. Interviews consisted of the K-SADS-PL, K-SADS MRS and DRS, and the UCLA-SAS. Questionnaire measures included the MESSY, SAS-SR, and a General Information form. These measures are detailed below. For assessments completed over the phone, participants had the option to complete questionnaires online. Subjects were compensated $20 for their participation.

Measures

Kiddie-Schedule for Affective Disorders –Present and Lifetime version. The K-SADS-PL (Kaufman et al., 1997) is a widely used semi-structured interview designed to assess the presence and severity of past and current psychopathology. Symptoms are rated on a 0-2 or
0-3 scale, with higher scores reflecting greater impairment and severity. The measure has shown high inter-rater reliability (.93-1.00), test-retest reliability (.63-1.00), and concurrent validity with diagnostic screeners (Kaufman et al., 1997). As early-onset BD patients previously completed this interview at intake into the original FFT-A treatment study, questions were only asked regarding the present timeframe. For participants in the control group, questions addressed both present and lifetime symptom occurrence.

**Kiddie-Schedule for Affective Disorders Depression and Mania Rating Scales.** The K-SADS Depression (DRS) and KSADS Mania (MRS) rating scales offer a thorough assessment of current depressive and manic symptoms on a 1-6 Likert scale of severity and impairment. These clinician-administered measures cover the most severe 1-2 weeks in the month prior to assessment. These interviews offer more extensive coverage of affective symptoms and greater range of response choices than the K-SADS-PL (Axelson et al., 2006). The DRS and MRS are administered to adolescents and parent(s) in Dr. Miklowitz’s FFT studies at baseline and multiple follow-ups. DSM-IV mood disorder diagnoses are derived from the MRS and DRS data. In prior work with this sample, MRS and DRS interrater reliability scores (51 ratings) were 0.97 and 0.89 (intraclass rs), respectively. For the current study, adolescent patients were asked only about their most significant mood experiences in the past month. Participants in the control group were asked about both their current (i.e. most significant within the past month) and lifetime experience with depressed and manic symptoms. For this study, Cronbach’s alpha for the DRS was 0.89 and for the MRS was 0.91.

**UCLA Social Attainment Survey.** The UCLA-SAS interview (Goldstein, 1978) contains 7 items evaluating peer and romantic relationships, as well as participation in activities and organizations. Each area is scored on a 1-5 scale, with higher scores indicating improved
social functioning. The measure correlates with other measures of social functioning (Phillips Scale of Premorbid Adjustment; Phillips, 1953). The interview has been used frequently in the social functioning assessment in patients with schizophrenia (e.g., Caton, Shrout, Dominguez, Eagle, Opler & Cournos, 1995; Miklowitz, Goldstein, Falloon & Doane, 1984; Niendam et al., 2006) and bipolar disorder (e.g., Frank et al., 2008; Miklowitz, Goldstein, Nuechterlein, Snyder & Mintz, 1988). In the current study, Cronbach’s alpha for the UCLA-SAS was 0.79.

In the current study, UCLA-SAS scores were transposed to reflect social impairment, as opposed to social strength, to be consistent with the scoring of the remaining study measures. To do this, subjects’ scores were subtracted from the possible total possible attainable score (e.g., for the total UCLA-SAS Impairment score, each subject’s score was subtracted from 35, as each of the 7 items is scored on a 1-5 scale). As such, greater scores on all symptom and social functioning measures reflect greater impairment (except where noted on individual subscales).

The Matson Evaluation of Social Skills with Youngsters. The MESSY (Matson, Rotatori & Helsel, 1983) is a self-report measure of social skills for children and adolescents up to 18 years of age. Five-point likert scales are used to assess the frequency of social behaviors in a number of domains, including assertiveness/aggression (e.g., ‘I threaten people or act like a bully’), impulsivity (e.g., ‘I speak when someone else is speaking’), overconfidence (e.g., ‘I think I know it all’), jealousy and withdrawal (e.g., ‘I feel angry or jealous when someone else does well’), and social appropriateness (e.g., ‘I look at people when I talk to them’). The MESSY demonstrates satisfactory psychometric properties, including good test-retest reliability and internal consistency, as well as agreement with other measures of social functioning (Matson, Heinze, Helsel, Kapperman & Rotatori, 1986). Finally, the MESSY correlates with observational measures of adolescent social functioning (Matson, Esveldt-Dawson & Kazdin, 1983). In the
current study, Cronbach’s alpha for the MESSY was 0.83.

**Social Adjustment Scale –Self Report, Short Form.** The SAS-SR (Weissman & Bothwell, 1976) is a 36-item self-report questionnaire assessing social functioning in adults; the current study utilized the Youth SAS-SR, a 22-item newly-developed version intended for use with children and adolescents. Assessed domains include functioning with school, family members, peer friendships, romantic relationships, and overall life satisfaction. Each item is rated on a 5-point scale, with greater scores reflecting increased impairment. Domain summary scores are the averaged participant rating of items within each subscale, with the denominator determined by the number of subscale items completed by the participant. The SAS-SR demonstrates adequate internal consistency, test-retest reliability, as well as convergent, discriminant, and concurrent validity. It has been used frequently to assess social functioning in adult populations with affective disorders (Talati et al., 2007; Weissman, Olfson, Gameroff, Feder & Fuentes, 2001). The Cronbach’s alpha for the friendship subscale of the Youth SAS-SR (i.e., Youth-SAS Friend score), used in the current study, was 0.72.

**The Child Behavior Checklist (previously collected).** The CBCL (Achenbach, 1991) consists of 118 parent-reported problem behavior items rated from 0 (“not at all typical of the child”) to 2 (“often typical of the child”) that can be added to determine “externalizing” and “internalizing” behavior problem scores as well as clinical syndrome scale scores. The Social Syndrome subscale measures social problems a child may be having with peer relationships (e.g. ‘gets teased a lot’). Higher scores on this measure reflect greater impairment. The Social Competence Scale measures social functioning in a number of areas, such as participation in organizations, how well the child gets along with others, and number of close friends. Lower scores on this measure reflect greater impairment. In the FFT-A randomized controlled trial,
parents rated the CBCL at intake, as well as multiple time points throughout the course of the study. CBCL data collected during intake assessments for the randomized controlled trial – specifically parent responses on the Social Problems and Social Competency subscales – were used for analyses.

The Family Adaptability and Cohesion Evaluation Scale, II (previously collected). The FACES-II (Olson, Russell & Sprenkle, 1983; Olson & Tiesel, 1991), a well-validated self-report measure, contains 30 questions rated on 5-point Likert-type scales. Items are divided between cohesion and adaptability scales, with lower scale scores indicating more impairment. Typical items from the cohesion subscale include “In our family, everyone goes his/her own way” and “Family members feel very close to each other”. Typical items from the adaptability subscale include “Our family tries new ways of dealing with problems” and “When problems arise, we compromise”. When summed, these scales indicate the degree to which a family is ‘balanced’. Previous research has suggested that these dimensions are most useful when analyzed separately, rather than combined as a summary of functioning (e.g. Place, Hulsmeier, Brownrigg & Soulsby, 2005). For the current study, both parent- and teen-reported scores were used. When multiple parent-reported responses were available for a given patient, an average score was used in the analyses detailed below. The Cronbach’s alpha for the FACES-II data used in the current study (for both parent – and teen- report) was 0.77.

The Conflict Behavior Questionnaire (previously collected). The CBQ (Prinz, Foster, Kent & O’Leary, 1979) is a self-report measure of interpersonal conflict within the family. Respondents answer “true/false” questions regarding their relationship with their offspring or parent (e.g. the mother about the adolescent or the child about the father). Typical items include, “My child is easy to get along with” or “My mother thinks my opinions don’t count.” Responses
are assigned a score of 0 or 1, dependent on whether or not the response reflects conflict; items are summed to generate a total conflict score (range: 0-20; higher scores indicate greater conflict). For the current study, both parent- and teen-reported scores were used. When multiple responses were available for a given patient, an average score was used in the analyses below. The Cronbach’s alpha for the child-report CBQ data used in the current study was 0.93; for parent-report CBQ data, Cronbach’s alpha was also 0.93.

General Information Form. A brief form asked participants to describe their current living situation, occupational and academic commitments, current medications, as well as their preference for gift card reimbursement.

Data Analysis

Initial analyses compared the patient and control groups on age, gender, living situation, employment and academic circumstances (i.e., is the adolescent attending high school full-time? Is he or she employed?), and medication usage. When significant differences existed, these variables were included as covariates in the following analyses:

Specific Aim 1: Group comparison of social functioning variables. For hypotheses 1 and 2, we predicted that adolescents and young adults with BD would demonstrate greater impairment than healthy adolescents and young adults on each measure of social functioning with peers. We conducted two-tailed t-tests comparing scores from participants with and without bipolar disorder on the relevant variable and/or subscale scores, outlined below:

Hypothesis 1. Aptitude with specific social skills that are relevant to peer relationships, namely: assertiveness, impulsivity, overconfidence, jealousy and withdrawal, and social appropriateness. These variables are calculated as separate subtotals from the MESSY questionnaire.
**Hypothesis 2.** Overall measures of the quantity and quality of relationships and engagement in activities with peers. These variables are measured by a combination of items from the UCLA-SAS. In addition to a Total Impairment score, deficits in same-sex peer relationships, leadership in same-sex peer relationships, opposite-sex peer relationships, romantic relationships and sexual relationships were combined into a measure of Relationship Impairment by the addition of items 1 through 5 from the UCLA-SAS; deficits in activity engagement with peers were measured by the combination of items 6 and 7 (‘outside activities’ and ‘participation in organizations’). Overall quality of peer friendships was also assessed with the Friendship subscale of the Youth-SAS-SR questionnaire.

**Specific Aim 2: Concurrent predictors of social functioning.** For hypothesis 3, planned linear regression analyses included data from patients with early-onset BD and healthy control subjects.

**Hypothesis 3.** We predicted that participants’ current DRS and MRS scores would predict concurrent social functioning – as measured by the Total MESSY Score, UCLA-SAS Impairment score, and Youth-SAS Friend score – such that greater symptom severity would predict greater social impairment.

**Specific Aim 3: Longitudinal predictors of social functioning.** For hypotheses 4 and 5, planned linear regression analyses included data only from patients with early-onset BD.

**Hypothesis 4.** We predicted that patients’ prior social functioning, as measured by the CBCL Social Syndrome and Social Competence subscales at intake into the original treatment study, would predict current social functioning with peers, as measured by the Total MESSY Score, UCLA-SAS Impairment score, and Youth-SAS Friend score.
Hypothesis 5. We predicted that patients’ prior family functioning, as measured by parent- and teen-reported FACES-II and CBQ questionnaires at intake into the original treatment study, would predict current social functioning with peers, as measured by the Total MESSY Score, UCLA-SAS Impairment score, and Youth-SAS Friend score.

Specific Aim 4: Prior social functioning as predictor of current affective symptoms. 
For hypothesis 6, planned linear regressions included data only from patients with early-onset BD.

Hypothesis 6. We predicted that patients’ intake social functioning with peers, as measured by the CBCL Social Syndrome and Social Competence Subscales, would predict adolescents’ current depressive symptom severity, as measured by the DRS. Controlling for intake depressive symptom severity, by inclusion of intake DRS scores in these models, we anticipated that greater impairment in social functioning would predict greater severity of current depressive symptoms.

For each of the analyses outlined above, the influence of symptom (e.g., BD I vs BD II) and demographic variables (e.g., gender, living situation) on social functioning was examined, where possible. The feasibility of these analyses was determined by the distribution of these variables within the recruited sample.

Statistical Analysis

While each of the statistical models outlined above make use of different variables, the initial model used in each analysis was essentially the same. In each case, analyses included the comparison of the following two models, with inclusion of additional covariates where appropriate.
Model A: \( Y_i = \beta_0 + \beta_1 X_i + \varepsilon_i \)

Model C: \( Y_i = \beta_0 + \varepsilon_i \)

The dependent variable \( Y \) is defined for each analysis as outlined above. In some models the independent variable ‘\( X \)’ is binary (e.g. patient versus healthy control group membership), while in others the independent variable is continuous (e.g. Social Syndrome subscale from the CBCL; patients’ self-reported FACES-II scores).

**Human Subjects and Confidentiality**

The University of Colorado’s Human Research Committee has continuously approved the treatment study from which participants were drawn. Separate IRB approval was obtained for this study protocol (IRB Protocol # 11-0646), including necessary consent and adolescent assent forms, recruitment materials, and data management procedures relevant to the current project.

**Risks to subjects.** The risks of the study were outlined in the initial letter, subsequently addressed during the first phone contact, and later detailed immediately prior to attaining consent. The major risk of this study was the potential for feeling uncomfortable when discussing mood and/or social difficulties. Mood assessment procedures had been used in Dr. Miklowitz’s and others’ similar studies without difficulty. Similarly, the measures of social functioning included with the current study had been used effectively with adolescent and adult populations in the past. Although the interviewer was prepared to address any distress or discomfort experienced throughout the course of the assessment, or discontinue the assessment if necessary, participants completed this study without reported or apparent distress. The investigator provided referrals to participants who met criteria for a DSM-IV disorder, or who requested local psychiatric resources.

**Confidentiality.** To ensure confidentiality, all research materials were (and continue to
be) held in the strictest confidence. Names did not appear on any of the questionnaires, and all interviews were coded by subject number. In the case of participants with bipolar disorder, their data were filed according to their existing family number, assigned during participation in the original FFT-A treatment study. Adolescents within the control group were assigned a unique number identifier. Study personnel in Dr. Miklowitz’s lab have sole access to the existing family numbers of BD participants. The investigator has exclusive knowledge of the coding system used with adolescents within the healthy control group. All assessment files have been stored in secure filing cabinets. All electronic files are password-protected, have been similarly coded by family number, and do not include names or other identifying information. Finally, all consent forms and identifying information have been filed separately from research materials to ensure confidentiality. The investigator will safely store these data indefinitely so they can be used for additional studies in the future. Any new study would also be reviewed by an Institutional Review Board.

**Power Analysis**

For group comparisons of bipolar and control participants (30 in each group), and using $\alpha = .05$, our design has 72% power to detect a medium effect size (Cohen’s $d = .5$) or 99% power to detect a large effect size (Cohen’s $d = .8$) in mean differences between groups. For within-group analyses (N= 30 bipolar participants) using $\alpha = .05$, our design has 42% power to detect a medium effect size (Cohen’s $d = .5$) or 93% power to detect a large effect size (Cohen’s $d = .8$).
CHAPTER 3

RESULTS

Missing Data

All participants completed all items on the Depression Rating Scale, Mania Rating Scale and K-SADS interviews. All participants completed the UCLA-SAS; 18 subjects responded to the item regarding sexual activity with “pass” or unratable responses (i.e., some participants requested to skip the question, while others did not provide sufficient detail to score this item). In these cases, the sexual activity item score was calculated by averaging the subject’s responses to the remaining six items on the interview.

Regarding questionnaire data, one control subject did not complete the Youth SAS–SR during her study appointment, and was unable to be reached to complete the form at a later time. One BD subject did not complete any online questionnaires following completion of the phone interview. In the case of missed items on the MESSY questionnaire, missing responses were prorated by calculating the average of the subject’s responses within the appropriate subscale. Missing item data were replaced in this manner for two control subjects (for each protocol, at least 87% of items were answered by the participant).

Prorating was not necessary with data for the Youth-SAS Friend score, as subscale scoring accounts for items missed by the participant (see measure description, above). A substantial portion of the study sample was not enrolled in school when they participated in the current study (32%; due to graduation or completion of the study during summer months), and school-related items were appropriately left uncompleted. As such, this domain was not included in present analyses. Similarly, a significant portion of the sample did not respond to the two
questions that comprise the Youth SAS-SR dating subscale (23%; due in part to questionnaire instructions limiting respondent age range), and these data were also not included in analyses.

Regarding social functioning data from entry into the FFT-A study, intake parent-rated CBCL scores were not available for eight of the 30 BD subjects enrolled in the current study. Subsequently, statistical power was reduced to 32% to detect a medium effect size (using $\alpha = .05$) or 83% power to detect a large effect size in analyses using this measure.

With respect to parent-reported family functioning data, three FACES-II questionnaires and four CBQ protocols not completed during intake. Statistical power was reduced to approximately 37% to detect a medium effect size (using $\alpha = .05$) or 89% power to detect a large effect size in analyses using these measures. Regarding teen-reported measures, six FACES-II questionnaires and six CBQ questionnaires were also not completed during intake into the original treatment study, and were therefore not available for the present analyses. As a result, statistical power was reduced to approximately 35% to detect a medium effect size (using $\alpha = .05$) or 87% power to detect a large effect size in analyses using these measures.

**Sample Characteristics**

A total of 61 subjects (31 control subjects; 30 with BD; see Table 1) participated in the study. One control subject was excluded from analysis following endorsement of depressive symptoms that met criteria for a major depressive episode. BD participants were significantly older than control subjects (Control: $M=17.50$ years; $SD=2.46$, range=14-22 years; BD: $M=19.07$ years, $SD=2.02$, range=15-23 years; $F(1,58)=7.28$, $p=.009$; Cohen’s $d=.70$). High school enrollment was significantly different between groups, such that control subjects were more likely to endorse being a full-time high school student (Control=67% vs. BD=27%), and BD subjects were more likely to report that they had graduated from high school (Control=33%
vs. BD=63%; $X^2(2, N=60)= 10.94, p=.004; \text{Cramer's } V=.43)$. Control subjects were more likely to have a part-time job (Control=43% vs. BD=30%), and BD subjects were more likely to endorse having a full-time job (Control=4% vs BD=27%; $X^2(2, N=60)= 6.48, p=.039; \text{Cramer's } V=.33$). Groups did not significantly differ on remaining gender, ethnicity, race, education, or living environment variables. Demographic data for study groups are presented in Table 1; statistical values reflect comparisons between subject groups.

Fifty-six adolescents with BD participated in the original FFT-A treatment study; the 30 BD subjects participating in the current study were compared to the 26 BD subjects from the original treatment study that did not participate in the current study. Participants and nonparticipants did not significantly differ on patient gender, ethnicity, race, socioeconomic status, bipolar diagnosis, severity of affective symptoms at intake for the original treatment study, CBCL Social Syndrome or Competence subscale scores, parent- or teen-reported measures of family functioning, or treatment group (i.e., participation in FFT-A versus the control Enhanced Care treatment condition). At the trend level, BD subjects in the current study completed a greater number of follow up assessments than patients who did not participate in the current study (Participant follow up weeks: $M=101.66, SD=23.56$; Non-participant follow up weeks: $M=83.58, SD=41.41$; $F(1,52)=3.98, p=.0514$, Cohen’s $d=0.54$).

**Symptom Data**

As expected, BD participants endorsed experience of greater levels of affective symptoms within the past month (Control DRS total: $M=3.00, SD=3.11$, range=0-14; BD DRS total: $M=13.13, SD=9.33$, range=0-34; $F(1,58)=31.82, p<.0001$, Cohen’s $d=1.46$; Control MRS total: $M=2.66, SD=2.56$, range=0-9; BD MRS total: $M=9.00, SD=9.58$, range=0-45; $F(1,58)=11.46, p=.001$, Cohen’s $d=0.90$). In addition, BD subjects were significantly more likely
Table 1: Comparison of Sample Demographic Data Between Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Controls M(SD)</th>
<th>Bipolar M(SD)</th>
<th>F(1,58)</th>
<th>p</th>
<th>Effect Size</th>
</tr>
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<tbody>
<tr>
<td>Age in Years</td>
<td>17.50 (2.46)</td>
<td>19.07 (2.02)</td>
<td>7.28</td>
<td>.009</td>
<td>d = .70</td>
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<table>
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<tr>
<th>Variable</th>
<th>Controls N (%)</th>
<th>Bipolar N (%)</th>
<th>X²(df)</th>
<th>p</th>
<th>Effect Size</th>
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<td>GENDER</td>
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</tr>
<tr>
<td>Male</td>
<td>12 (40%)</td>
<td>11 (38%)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>18 (60%)</td>
<td>19 (62%)</td>
<td>0.07(1)</td>
<td>.791</td>
<td>Φ = .03</td>
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<tr>
<td>RACE</td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Black/African American</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Asian</td>
<td>3(10%)</td>
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</tr>
<tr>
<td>Caucasian</td>
<td>26(87%)</td>
<td>29(97%)</td>
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</tr>
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<td>0</td>
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<tr>
<td>Other</td>
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<td>1(3%)</td>
<td>5.16(3)</td>
<td>.160</td>
<td>V = .29</td>
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</tr>
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<td>Hispanic</td>
<td>2 (7%)</td>
<td>5 (17%)</td>
<td>1.45(1)</td>
<td>.228</td>
<td>Φ = -.16</td>
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<td>Non-Hispanic</td>
<td>28 (93%)</td>
<td>25 (83%)</td>
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<td>HIGH SCHOOL</td>
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<tr>
<td>Full Time</td>
<td>20 (67%)</td>
<td>8 (27%)</td>
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<tr>
<td>Part Time</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quit</td>
<td>0</td>
<td>3 (10%)</td>
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<tr>
<td>Graduated</td>
<td>10 (33%)</td>
<td>19(63%)</td>
<td>10.94(2)</td>
<td>.004</td>
<td>V = .43</td>
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<td>COLLEGE</td>
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<tr>
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<td>20 (67%)</td>
<td>18 (60%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Part Time</td>
<td>2 (7%)</td>
<td>3 (10%)</td>
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<td></td>
</tr>
<tr>
<td>Full Time</td>
<td>8 (26%)</td>
<td>9 (30%)</td>
<td>0.36(2)</td>
<td>.834</td>
<td>V = .08</td>
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<td>JOB</td>
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<tr>
<td>No Job</td>
<td>16 (53%)</td>
<td>13 (43%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Part Time</td>
<td>13 (43%)</td>
<td>9 (30%)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Full Time</td>
<td>1 (4%)</td>
<td>8 (27%)</td>
<td>6.48(2)</td>
<td>.039</td>
<td>V = .33</td>
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<td>LIVING ENVIRONMENT</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With Parents/Guardian</td>
<td>20 (67%)</td>
<td>18 (60%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without Parents/Guardian</td>
<td>10 (33%)</td>
<td>12(40%)</td>
<td>0.29(1)</td>
<td>.592</td>
<td>Φ = .07</td>
</tr>
</tbody>
</table>
to be diagnosed with a comorbid psychiatric disorder, based on report of symptoms experienced within the past year ($X^2(1, N=60)=15.43$, $p<.0001$, Cramer’s $\Phi = .51$). These included behavior disorders (i.e., oppositional defiant disorder or conduct disorder), attention deficit and hyperactivity disorder, substance abuse or dependence disorders; BD patients were also more likely to experience psychotic symptoms ($p < .05$ for all). At the trend level ($p < .07$), BD participants were more likely to be diagnosed with an anxiety disorder (generalized anxiety disorder, panic disorder, social phobia, specific phobia, separation anxiety, or obsessive compulsive disorder). Affective and other symptom data for each group are presented in Table 2.

Particularly germane to the present study, six BD subjects and one control subject met criteria for social phobia. BD subjects with and without social phobia did not differ on affective or social functioning outcome measures ($p > .05$ for all). Results presented below were not significantly affected by the exclusion of these subjects.

### Table 2: Comparison of Affective and Other Symptom Data Between Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Controls M (SD)</th>
<th>Bipolar M(SD)</th>
<th>$F(1,58)$</th>
<th>$p$</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression Rating Scale Total</td>
<td>3.00 (3.11)</td>
<td>13.13 (9.33)</td>
<td>31.82</td>
<td>.001</td>
<td>$d=1.46$</td>
</tr>
<tr>
<td>Mania Rating Scale Total</td>
<td>2.66 (2.56)</td>
<td>9.00 (9.58)</td>
<td>12.22</td>
<td>.001</td>
<td>$d=.90$</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Controls N (%)</th>
<th>Bipolar N(%)</th>
<th>$X^2(1,58)$</th>
<th>$p$</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>COMORBID DIAGNOSES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any Psychiatric Diagnosis</td>
<td>5 (17%)</td>
<td>20(67%)</td>
<td>15.43</td>
<td>.000</td>
<td>$\Phi = .51$</td>
</tr>
<tr>
<td>Any Anxiety Disorder</td>
<td>4 (13%)</td>
<td>10 (33%)</td>
<td>3.35</td>
<td>.067</td>
<td>$\Phi = .26$</td>
</tr>
<tr>
<td>Any Behavioral Disorder</td>
<td>0</td>
<td>0</td>
<td>.</td>
<td>.</td>
<td>.</td>
</tr>
<tr>
<td>Any ADHD</td>
<td>1 (3%)</td>
<td>7 (23%)</td>
<td>5.19</td>
<td>.023</td>
<td>$\Phi = .29$</td>
</tr>
<tr>
<td>Any Substance Abuse</td>
<td>0</td>
<td>5 (17%)</td>
<td>5.45</td>
<td>.019</td>
<td>$\Phi = .30$</td>
</tr>
<tr>
<td>Any Substance Dependence</td>
<td>0</td>
<td>7 (23%)</td>
<td>7.92</td>
<td>.005</td>
<td>$\Phi = .36$</td>
</tr>
<tr>
<td>Any Psychotic Symptoms</td>
<td>0</td>
<td>5 (17%)</td>
<td>5.45</td>
<td>.019</td>
<td>$\Phi = .30$</td>
</tr>
</tbody>
</table>
Primary and Secondary Outcomes

DRS Total scores, MRS Total scores and Youth-SAS Friend scores were positively skewed in our dataset (DRS Total score skew=1.27; MRS Total score skew=2.89; Youth-SAS Friend score skew=1.45). To reduce the influence of outliers and allow for accurate interpretation of the analyses detailed below, these score distributions were corrected with a logarithmic transformation. All subsequent reference to DRS, MRS and Youth-SAS Friend scores indicates use of the logarithmic transformation of these scores, unless noted.

Given the significant difference in age between control and BD subjects, and the substantial literature suggesting the influence of gender on social variables (for review, see Rose & Rudolph, 2006), group comparison models included subject age, gender, and their two- and three-way interactions with diagnosis (BD versus control). The low prevalence of racial and ethnic minorities in the study sample precluded meaningful comparisons on these variables. As high school education (F (1,58)=95.37, p<.0001, $R^2 = .62$), college education (F(1,58)=7.70, p=.007, $R^2 = .12$) and employment (F(1,58)=13.86, p<.001, $R^2 = .19$) and living situation (F(1,58)=32.80, p<.0001, $R^2 = .36$) variables were highly correlated with age, they were not included in these models to avoid redundancy among covariates, unless specifically noted.

Initial analyses demonstrated that, controlling for participant group, subjects’ living situations predicted UCLA-SAS Impairment scores (F(1,57)=9.29, p=.003, $R^2=.26$), such that living with parents or guardians was associated with greater impairment in social functioning. Further analyses suggested this relationship was reflected in items assessing relationship quality (F(1,57)=14.16, p=.001, $R^2=.25$), but not engagement in activities (F(1,57)=0.32, p=.572, $R^2=.19$). Living situation was therefore included as a covariate in subsequent models predicting
UCLA-SAS Impairment scores. High school, college and living situation variables were otherwise not related to social functioning.

With respect to psychopharmacology, no control subjects reported currently taking psychotropic medication. Nearly half of the BD subjects reported that they were not currently taking a psychotropic medication (N=13); five BD subjects reported use of mood stabilizer(s) only, two reported using antipsychotic(s) only, seven reported use of both antipsychotic(s) and mood stabilizer(s), and two reported taking other kinds of medication (e.g., Prozac and a stimulant). For all measures, use of psychotropic medication (i.e., binary yes/no) did not predict social functioning (p > .05 for all). Regarding patient characteristics, bipolar diagnosis (i.e., bipolar I vs bipolar II), age of symptom onset (for both depression and mania), and severity of most significant past episode were unrelated to current measures of social functioning (p > .05 for all). As such, medication usage, BD diagnosis, age of symptom onset, and most severe past episode were not included as covariates in study analyses.

Specific Aim #1: Group Comparison of Social Functioning Variables

Hypothesis 1: Relative to controls, participants with early-onset BD will report greater difficulties in specific social skill areas relevant to peer relationships, specifically: assertiveness, impulsivity, overconfidence, jealousy and withdrawal, and social appropriateness. Supporting our hypothesis, BD subjects’ Total MESSY scores reflected significantly more impairment than those of control subjects, controlling for age, gender, and their interactions (F(1,51)=10.63, p = .002, d = .73; see Figure 1). Although BD and control subjects did not differ significantly on measures of appropriate social skills (F(1,51)=0.41, p=.525, d=-.20), BD subjects reported significantly greater impairment in the areas of impulsivity (F(1,51)=12.76, p <.001, d=.81), jealousy and withdrawal (F(1,51)=6.63, p=.013,
d=.71), and inappropriate assertiveness (F(1,51)=7.76, p=.007, d=.56). The reported difference between BD and control subjects in the area of overconfidence was at the trend level ((F(1,51)=3.85, p=.055, d=.47).

Gender differences across subject age and group reflected endorsement of poorer functioning by males as compared to females. Specifically, significant differences were noted in the Total MESSY score (F(1,51)=6.76, p=.012, d=.80), as well as measures of appropriate social skills (F(1,51)=5.66, p=.021, d=.73) and inappropriate assertiveness (F(1,51)=6.45, p=.014, d=.74; see Figure 2). Controlling for group membership and age, male and female participants did not significantly differ on self-reported impulsivity, overconfidence, or jealousy and withdrawal.

There was a significant three-way interaction between group, gender, and age in participant reports of impulsivity (F(1,51)=8.18, p=.006), such that males’ reports of impulsivity reduced as age increased within the control group, while males’ reports of impulsivity increased with age within the BD subject group. For females, impulsivity did not differ across ages within the control group, and scores were lower as age increased for BD subjects. No additional two- or three-way interactions were significant. For all analyses, there were no significant main effects of age on MESSY total or domain scores.

Hypothesis 2: Relative to controls, participants with early-onset BD will show more impairment in social functioning with peers, as reflected by decreased involvement in peer-related activities and a lower quantity of relationships (friendships and romantic relationships). Across subject age, gender, and living situation, total UCLA-SAS Impairment scores reflected lower quality of social functioning in BD subjects, as compared to control subjects (Control: M=7.15, SD=3.85; BD: M=11, SD=5.81; F(1,49)=9.33, p=.004,
Figure 1: Total Matson Evaluation of Social Skills with Youth (MESSY) and Domain Mean Scores\textsuperscript{a}: Group Comparison

\* = p < .05
\* * = p ≤ .01

\textsuperscript{a}Unlike remaining subscales, greater Appropriate Social Skills scores reflect \textit{less} impairment
Figure 2: Total Matson Evaluation of Social Skills with Youth (MESSY) and Domain Mean Scores\(^a\): Gender Comparison

\[^a\text{Unlike remaining subscales, greater Appropriate Social Skills scores reflect less impairment}\]

Cohen’s \(d = .78\); see Figure 3). This discrepancy occurred in items reflecting relationship quality (Control: \(M = 6.55, SD = 3.57\); BD: \(M = 8.46, SD = 3.99\); \(F(1,49) = 4.26, p = .044\), Cohen’s \(d = .51\)) and level of engagement in activities with peers (Control: \(M = 0.6, SD = 1.22\); BD: \(M = 2.53, SD = 2.66\); \(F(1,49) = 11.50, p = .001\), Cohen’s \(d = .93\)). Across groups, subject age and gender were unrelated to UCLA-SAS Impairment scores (although the interaction between age and group did not reach statistical significance, exploratory analyses demonstrated that greater age was...
correlated with decreased social impairment within the control group, but not the BD group; see Appendix A).

**Figure 3: UCLA - Social Attainment Survey (UCLA-SAS) Impairment Scores By Group**

![Bar graph showing UCLA-SAS Impairment Scores by Group](image)

* *= p ≤ .01

Results from Youth-SAS Friend scores also reflected group differences in self-reported friendship quality. Controlling for subject age and gender and their interactions, BD subjects’ higher average scores reflected lower self-reported friendship quality than that reported by control subjects (Control: $M=1.64, SD=1.24$; BD: $M=1.97, SD=1.34$; $F(1,50)=6.25, p=.016$, Cohen’s $d = .74$; see Figure 4; for ease of interpretation, Figure 4 and summary statistics reported here are presented with logarithm scores transformed to the original Youth SAS-SR metric). Again, subject age and gender were unrelated to Youth-SAS Friend scores.
Figure 4: Youth Social Adjustment Survey – Self-Report Friendship Scores By Group

Specific Aim #2: Concurrent Predictors of Social Functioning

Hypothesis 3: Participants’ symptoms of depression and mania will predict concurrent social functioning with peers, such that more severe symptom scores will predict greater levels of social impairment with peers. To assess the relationship between affective symptoms and social functioning across subject groups, models predicting social functioning included MRS or DRS total scores, subject group, and the interaction between group and affective symptom score. Controlling for subject gender and group, the severity of participants’ depression predicted Total MESSY scores (F(1,54)=9.19, p<.0001, $R^2=.40$), such
that greater symptom severity was associated with poorer social functioning.\(^1\) In contrast, after controlling for subject group, severity of current depressive symptoms did not predict UCLA-SAS Impairment scores or Youth-SAS Friend scores (\(p>.05\) for both). For each model, the relationship between affective symptoms and social functioning was consistent between groups (i.e., the DRS \(\times\) group interaction terms were nonsignificant). Severity of manic symptoms was unrelated to measures of current social functioning (for all, \(p>.05\)).

To further explore the relationships between symptoms and social functioning, a subset of BD subjects who endorsed few or no depressive symptoms (i.e., DRS raw scores of less than 9; \(N = 14\)) was compared to the control group\(^2\). Although the BD subgroup and control subjects did not differ significantly on endorsed depressive symptoms, living situation or gender (\(p>.05\) for all), BD subjects’ scores reflected significantly greater social impairment on UCLA-SAS Impairment scores (Control: \(M=7.15, SD=3.85\); BD: \(M=10.99, SD=6.29\); \(F(1,42)=6.26, p=.016, \) Cohen’s \(d=.74\)), with trends towards a similar difference in Total MESSY scores (Control: \(M=109.78, SD=15.84\); BD: \(M=119.75, SD=20.47\); \(F(1,42)=3.13, p=.084, \) Cohen’s \(d=.54\)) and Youth-SAS Friend scores (Control: \(M=0.49, SD=0.22\); BD: \(M=0.67, SD=0.34\); \(F(1,41)=3.96, p=.053, \) Cohen’s \(d=.63\)).

A subset of BD subjects who endorsed no or few current manic symptoms (i.e., MRS raw scores of less than 9; \(N = 17\)) was also compared to the control group. While the BD subgroup and control subjects did not differ significantly on endorsed manic symptoms, living situation or gender (\(p>.05\) for all), BD subjects’ scores reflected significantly greater social impairment on UCLA-SAS Impairment scores (Control: \(M=7.15, SD=3.85\); BD: \(M=10.99, SD=6.29\); \(F(1,42)=6.26, p=.016, \) Cohen’s \(d=.74\)), with trends towards a similar difference in Total MESSY scores (Control: \(M=109.78, SD=15.84\); BD: \(M=119.75, SD=20.47\); \(F(1,42)=3.13, p=.084, \) Cohen’s \(d=.54\)) and Youth-SAS Friend scores (Control: \(M=0.49, SD=0.22\); BD: \(M=0.67, SD=0.34\); \(F(1,41)=3.96, p=.053, \) Cohen’s \(d=.63\)).

---

\(^1\) Given previously-reported results, models predicting Total MESSY score also included subject gender as a covariate, and models predicting UCLA-SAS Impairment score also included living situation as a covariate.

\(^2\) As there is not an established DRS cutoff score indicating “low” depressive symptoms, scores less than 9 were used to maximize statistical power. More specifically, use of different cutoffs was explored in this sample; BD subjects with DRS scores less than 9 produced the largest group whose depressive symptoms were not significantly different from depressive symptoms reported by controls.
gender (p > .05 for all), BD subjects’ scores reflected significantly greater social impairment on the Total MESSY score (Control: $M=109.78$, $SD=15.84$; BD: $M=123.97$, $SD=21.19$; $F(1,44)=6.59$, $p=.014$, Cohen’s $d=.76$), UCLA-SAS Impairment score (Control: $M=7.15$, $SD=3.85$; BD: $M=10.45$, $SD=6.02$; $F(1,45)=5.27$, $p=.026$, Cohen’s $d=.65$), and the Youth-SAS Friend score (Control: $M=.49$, $SD=0.22$; BD: $M=.69$, $SD=0.31$; $F(1,43)=6.07$, $p=.018$, Cohen’s $d=.74$).

Because the low-depression BD subgroup endorsed significantly greater manic symptoms than control subjects (and similarly, the low-mania BD group endorsed greater experience with depressive symptoms than controls, $p < .05$ for both), a further subgroup of BD subjects (N=9) were selected who endorsed having few or no manic or depressive symptoms (i.e., total scores less than 9 for the DRS and MRS). Again, this low-symptom BD subgroup did not differ from control subjects on gender, living situation or affective symptom measures ($p > .05$ for both). BD subjects’ scores reflected significantly greater social impairment on the UCLA-SAS Impairment score (Control: $M=27.85$, $SD=3.85$; BD: $M=22.11$, $SD=7.19$; $F(1,37)=10.01$, $p=.003$, Cohen’s $d=.99$; see Figure 5). Groups did not significantly differ on Total MESSY scores (Control: $M=109.78$, $SD=15.84$; BD: $M=119.39$, $SD=21.54$; $F(1,37)=2.15$, $p=.15$, Cohen’s $d=.51$) and Youth-SAS Friend scores did not significantly differ between groups (Control: $M=.49$, $SD=.22$; BD: $M=.65$, $SD=.34$; $F(1,36)=2.82$, $p=.10$, Cohen’s $d=.58$).

Additional analyses examined the role of current comorbidity in subjects’ social functioning. As the incidence of psychiatric diagnosis was prohibitively low among control subjects, models testing for the role of comorbid diagnosis included only BD subjects. Comorbidity (i.e., whether a subject has no, one, or multiple comorbid diagnoses) was unrelated to all measures of social functioning (for all, $p > .05$).
Specific Aim #3: Longitudinal Predictors of Social Functioning

Hypothesis 4: Patients’ intake measures of social functioning with peers will predict current social functioning, such that greater levels of social impairment at original treatment study intake will predict greater levels of current social impairment with peers. Parent-reported measures of patients’ social functioning at study intake were unrelated to current self-reported measures of social functioning. More specifically, scores from the CBCL Social Syndrome Scale and the CBCL Social Competence Scale did not predict BD subjects’ current...
Total MESSY scores, UCLA-SAS Impairment scores, or Youth-SAS Friend scores (p > .05 for all).

**Hypothesis 5: Patients’ intake measures of family functioning will predict current social functioning, such that greater levels of family functioning impairment at original treatment study intake will predict greater levels of current social impairment with peers.**

Controlling for patient gender, parent-reported FACES-II cohesion significantly predicted current Total MESSY scores ($F(1,23)=12.46, p=.002, R^2=.47$; see Figure 5). This relationship remained significant in models covarying intake and current levels of depression and mania ($F(1,19) = 8.18, p=.01, R^2=.62$). Parent-reported FACES-II cohesion scores at intake were unrelated to current UCLA-SAS Impairment scores and Youth-SAS Friend scores. Remaining family functioning scores gathered at original treatment study intake, namely FACES-II adaptability and CBQ conflict scores, were unrelated to current measures of social functioning (p >.05 for all).

Controlling for patient gender, teen-reported FACES-II cohesion predicted Total MESSY scores ($F(1,20) = 17.72, p=.0004, R^2=.68$; see Figure 6), following removal of an outlier from the dataset (Studentized Deleted Residual = -3.9, Cook’s D = 0.4). This relationship remained significant in models covarying intake and current levels of mania and depression ($F(1,16) = 6.00, p=.026, R^2=.75$). Teen-reported adaptability at intake predicted current Total MESSY scores at the trend level ($F(1,21)=3.57 p=.072, R^2=.38$), although this relationship was not significant.

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3 Both Studentized Residual and Cook’s D are measurements of the error contributed by an individual data point within a given statistical model. Specifically, a Studentized Deleted Residual is the t statistic resulting from a comparison between a statistical model and the same model with an additional parameter specifically addressing a single observation. Conversely, Cook’s D is a comparison of model predictions including and excluding a given data point. In both cases, larger numbers indicate greater likelihood that an observation is an outlier for the analysis.
significant once intake and current levels of mania and depression were included as covariates (p > .50). For these relationships, better family functioning scores at intake predicted more adaptive social functioning. Teen-reported cohesion and adaptability at intake were unrelated to current UCLA-SAS Impairment scores or Youth-SAS Friendship scores. Teen-reported family conflict at intake was not related to any measure of current social functioning. See Appendix B for path analyses reflecting the relationships between Total MESSY Scores and the covariates detailed above in Hypotheses 4 & 5.

Figure 6: Parent- and Teen-Reported FACES-II Cohesion and Total MESSY Score

FACES-II = Family Adaptability and Cohesion Evaluation Scale, II; MESSY = Matson Evaluation of Social Skills with Youngsters

Specific Aim #4: Social Functioning at Intake as Predictor of Current Symptoms
Hypothesis 6: Patients’ intake scores of social functioning, after covarying intake levels of depressive severity, will predict current depressive severity. Specifically, greater levels of social impairment at treatment study intake will predict increased severity of depression. In our sample, previous social functioning scores did not predict current depressive symptom severity. This was true for models using the CBCL Social Syndrome and Social Competence Scale T-scores as independent predictors, models that included intake levels of depression as covariates, and models that additionally covaried current levels of social functioning (p > .05 for all). Exploratory analyses including previous and current manic symptoms demonstrated similar results (p > .05 for all).

Instead, following removal of an outlier data point (Studentized Deleted Residual=-4.26; Cook’s D=.37), DRS scores collected at FFT-A study intake significantly predicted current DRS scores (F(1,26)=11.77, p=.002, $R^2=.31$). Additionally, exploratory analyses (see Appendix A) demonstrated that teen-reported cohesion (F(1,22)=4.62, p=.043, $R^2=.17$) and adaptability (F(1,22)=4.73, p=.041, $R^2=.18$) at intake into the original treatment study also predicted current DRS scores. However, these family functioning variables no longer significantly predicted current depressive severity once intake DRS scores were entered as model covariates. Intake MRS scores from the original treatment study were unrelated to current MRS scores (p > .05).
CHAPTER 4

DISCUSSION

This study investigated the severity and stability of social impairment in early-onset bipolar disorder, and the relationships between social functioning, affective symptoms, and family functioning. Our analyses examined concurrent and longitudinal associations between study variables, including data from approximately 3.5 years earlier, collected during entry into a randomized controlled trial of family-focused treatment for adolescents (FFT-A). Participants with BD endorsed greater impairment in the use of specific social skills, as well as decreased relationship engagement and quality, as compared to gender-matched controls. Greater depressive symptom severity predicted poorer self-reported use of social skills; social functioning was otherwise independent of concurrent affective symptom severity and patient diagnostic characteristics (e.g., comorbidities, age of symptom onset, Bipolar I vs. Bipolar II diagnosis). Parent-reported social functioning, collected during FFT-A study intake, was unrelated to current self-reported social functioning of BD participants. In contrast, intake parent- and teen-reported cohesion predicted current self-reported social skills. Finally, intake measures of bipolar depression – and not prior assessments of social functioning – predicted current levels of bipolar depression.

Specific Aim #1: Group Comparison of Social Functioning Variables

Social functioning differences between gender-matched BD patients and controls were reflected in interview and self-report measures, as well as assessments of brief (e.g., Youth SAS-SR: last two weeks) and extended (UCLA-SAS: throughout adolescence) timeframes. Our inclusion of an older population suggests that previously-noted social deficits extend into early-
Although BD patients in our study were significantly older than control participants, measures of social functioning were independent of participant age. This finding contrasts with previous studies reporting that older participants report greater levels of psychosocial impairment (e.g., Biederman et al., 2005; Goldstein et al., 2009). These studies, however, typically included child and adolescent patients. Our findings from a population of older adolescents and young adults with early-onset BD may suggest that social impairment is more consistent during these developmental periods.

Participant gender significantly predicted some, but not all, of our social functioning outcome variables. Specifically, across participant groups, female participants endorsed superior use of specific social skills as compared to male participants. Gender differences were not found, however, in measures assessing social functioning more broadly (i.e., UCLA-SAS Impairment or Youth-SAS Friend scores). This disagreement within the current study, as well as contrasting findings from prior studies (e.g., Goldstein et al., 2006 did not find gender differences on the MESSY), suggest the need for additional research to clarify the influence of gender on peer relationships in early-onset BD.

Our findings may suggest that patients with early-onset BD experience a broad range of social functioning impairments throughout adolescents and early adulthood. Indeed, similar results have been interpreted as such throughout much of the literature (e.g., Goldstein et al., 2006; Goldstein et al., 2009). Alternately, however, group differences in our study may instead reflect an overestimation of social functioning by healthy controls. Research into depressive realism – the hypothesis that individuals with depression have a more accurate perception of reality than non-depressed individuals (Alloy & Abramson, 1979, 1988) – suggests that individuals with depressive diagnoses may actually provide more accurate self-assessments of
their social functioning, with non-depressed individuals providing overly optimistic reports of their social strengths and abilities (e.g., Edison & Adams, 1992; Lewinsohn, Mischel, Chaplin & Barton, 1980). These studies have not been replicated with bipolar populations; as such, it is currently unclear to what extent depressive realism may play a role in our findings. Our current study design did not allow us to determine if group differences reflect genuine functional impairment in BD subjects, over-reporting of social strength by healthy controls, or a combination of the two. Research including multiple ratings of participants’ social functioning (e.g., friend-, observer-, or clinician-report) or observational measures of social behavior would be better able to address this question.

Regarding self-reported functioning in specific domains of the MESSY, participants with early-onset BD differed from control subjects on all subscales (i.e., Inappropriate Assertiveness, Jealousy/Withdrawal, Impulsivity, and Overconfidence) except Appropriate Social Skills. Interestingly, this pattern of results mirrors the findings of Goldstein et al. (2006), in their comparison of BD adolescents (ages 13-18 years) and healthy control subjects. These results may suggest that deficits in social functioning among those with early-onset BD are characterized not by a lack of appropriate skill use, but instead by the addition of inappropriate behaviors that may decrease the likelihood of developing close peer relationships (e.g., being overly assertive with others). Alternately, adolescents and young adults with BD may be less aware of deficits in their use of appropriate social skills than they are of deficits in other domains of social behavior. Replication of these findings in datasets using a broader range of methodologies (e.g., parent- or clinician-report assessment) would permit a full interpretation of these results.

Research on typical adolescent development suggests that impaired social functioning, such as that associated with early-onset BD, may have significant negative consequences. Rather
than an overall increase in autonomy, adolescence is more accurately viewed as a shift from reliance on parents and family members towards a reliance on same-age peers (Steinberg & Silverberg, 1986). Adolescents seek support more frequently from peer, rather than family, networks as they mature (e.g., Helsen, Vollebergh & Meeus, 2000), particularly during times of increased stress (Harter, Stocker & Robinson, 1996; Stevens & Prinstein, 2005; Brechwald & Prinstein, 2011). Relatedly, increased quality of peer relationships has also been associated with improved coping mechanisms for stress (Savin-Williams & Berndt, 1990). Limited peer support may be particularly difficult for those with early-onset BD, who experience more frequent stressful life events and fewer positive life events than healthy control subjects or patients with other psychiatric diagnoses (Romero et al., 2009; Tillman et al., 2003).

The availability of consistent, healthy peer relationships can also have protective behavioral effects (e.g., increases in pro-social behavior), encourage the development of a favorable sense of self, and increase well-being (Brechwald & Prinstein, 2011; Harter et al., 1996; Oberle et al., 2010; Prinstein & La Greca, 2004; Savin-Williams & Berndt, 1990). Increased time with peers also presents increased opportunities for observing and modeling appropriate social behavior; the greater the intimacy of these peer relationships, the greater the peer influence on these pro-social behaviors (Prinstein, 2007; Savin-Williams & Berndt, 1990; Wentzel & Caldwell, 1997). Adolescents receive rapid feedback from peers regarding the appropriateness of their interactions; through this feedback, teenagers learn the most adaptive methods of navigating the social world they are entering (Brechwald & Prinstein, 2011; Oberle, Schonert-Reichl & Thomson, 2010; Savin-Williams & Berndt, 1990; Sullivan, 1953). Peers may also encourage participation in social activities (e.g. after school clubs, sports), which in turn fosters the further development of adolescent social skills (Savin-Williams & Berndt, 1990).
Conversely, deficits in social functioning with peers have been correlated with adjustment problems in later adolescence and adulthood (Parker & Asher, 1993; Trentacosta & Shaw, 2009; Woodward & Fergusson, 1999). The specific ways in which early-onset BD symptoms interfere with this developmental process remains unknown. Early in development, the experience of sub-clinical BD symptoms – such as those seen within high-risk populations – has been associated with decreased social awareness and motivation (e.g., Whitney et al., in press), which negatively affects a child’s ability to establish healthy peer relationships. More severe, acute affective symptoms (e.g., withdrawal, anhedonia, and irritability during depression; inappropriate behaviors during hypomania or mania) may result in damage to or termination of any established friendships. Alternately, patients may experience an incomplete or abnormal development of social behavior, which impairs interpersonal functioning regardless of mood state. For example, while research has suggested that the acquisition of social skills knowledge is unimpaired in BD adolescents, patients experience difficulty with appropriately acting on this knowledge (Goldstein et al., 2006).

**Specific Aim #2: Concurrent Predictors of Social Functioning**

Our second question concerned the concurrent relationships between affective symptoms and social functioning in adolescents and young adults. Initial whole-group analyses (N=60) suggested that greater depression severity predicted greater social impairment across all measures. Levels of mania were unrelated to social functioning; this may reflect the limited variance of manic symptoms within our sample, although similar findings suggesting independence between manic symptoms and social functioning have been reported elsewhere (Goldstein et al., 2009; Weinstock & Miller, 2008; Weinstock & Miller, 2010).
The relationship between DRS scores and Total MESSY scores remained significant when analyses were restricted to BD patients, suggesting that those with more severe depression endorsed poorer concurrent use of social skills. In contrast, initial analyses indicating that depressive symptoms predicted concurrent UCLA-SAS Impairment Scores and Youth-SAS Friend scores resulted wholly from subject group differences, as these relationships were no longer significant when restricted to only BD subjects (post-hoc analyses reflected similar findings within the intake data for the original treatment study; see Appendix A). These findings may reflect a difference in constructs measured by social functioning assessments in the current study. Possibly, an individual’s use of particular social skills (as measured by the MESSY) is more immediately responsive to concurrent mood symptoms. For example, an individual may be more inappropriately assertive with peers (i.e., “I hurt others’ feelings when teasing them”) if they are feeling irritable while depressed. The UCLA-SAS and Youth-SAS may instead measure aspects of social functioning that develop over time (e.g., quality of close friendships; involvement in recreational activities; degree of emotional closeness with others), which are less sensitive to fluctuations in mood.

Our analyses also demonstrated that a subset of BD subjects endorsing few or no affective symptoms reported poorer scores on some measures of social functioning than control subjects. A growing literature of research with adult (e.g., de Almeida Rocca et al., 2008, Ibanez et al., 2012; Malhi et al, 2008; Perez, Riggio & Kopelowicz, 2007) and pediatric (e.g., DelBello et al., 2007; Keenan-Miller, Peris, Axelson, Kowatch & Miklowitz, 2011; McClure et al., 2005) BD patients has suggested independence between symptom severity and social functioning, and the experience of notable impairment regardless of the presence or absence of affective symptoms. Research including adolescents with major depressive disorder has also indicated
inter-episode social impairment (e.g., Geller, Zimmerman, Williams, Bolhofner & Craney, 2001; Lewinsohn, Rohde, Seeley, Klein & Gotlib, 2003); however, the unipolar depression literature does generally indicate that greater severity of adolescent depression is correlated with poorer social functioning (e.g., Cole, Martin, Powers & Truglio, 1996; Fauber, Forehand, Long, Burke & Faust, 1987; Garland & Fitzgerald, 1998; Helsel & Matson, 1984; Judd et al., 2000; La Greca, & Harrison, 2005; Lewinsohn, Solomon, Seeley & Zeiss, 2000; Spirito, Hart, Overholser & Halverson, 1990).

Neurobiological research in BD populations may help to explain our findings, which suggest that at some aspects of social impairment may not be mood-dependent. The previously-discussed functional and structural neuroimaging studies - suggesting that early-onset BD is associated with cortical asymmetries, decreased neuronal density, and deficits in facial affect recognition – reflect neurobiological abnormalities which may influence functioning regardless of mood state. An additional area of research interest has been myelination within the BD adolescent brain. The normative period of increased myelination experienced during adolescence, particularly myelination increases within regions important in the coordination of speech and complex processing (i.e., corpus callosum), result in improved ability to organize complex processes in the teenage brain. This increased ability to quickly handle complexity may prime the adolescent for better social learning (Blakemore & Choudhury, 2006; Steinberg, 2005). Research has demonstrated overall deficits in neuronal myelination in individuals with BD and schizophrenia (e.g., Bellani et al., 2012; McIntosh, Hall, Lymer, Sussmann & Lawrie, 2009; Tkachev et al., 2003) but not unipolar depression (Brambilla et al., 2004). Myelination abnormalities within the corpus callosum have been noted in BD adult (Walterfang et al., 2009),
pediatric (Caetano et al., 2008; Frazier et al., 2007), and high risk (Versace et al., 2010) populations.

Social deficits seen in BD patients may also result from impaired development of functional neural networks. Nelson, Leibenluft, McClure and Pine (2005) emphasize the importance of the Social Information Processing Network (SIPN), which is responsive to hormone and other pubertal changes that occur during adolescence. One component of this network, the affective ‘node’ (e.g., amygdala, ventral striatum, septum, hypothalamus), is sensitive to hormonal changes, and completes both an anatomical and functional reorganization during adolescence. The cognitive and regulatory node (e.g., ventral, medial and dorsomedial prefrontal cortex, orbitofrontal cortex) in contrast, is relatively insensitive to gonadal hormones, and develops via neural pruning – a slower, competitive process that is dependent on social learning. This cognitive node is thought to be involved with the development of social cognition and ultimately, the control of social behavior (Blakemore & Choudhury, 2006; Nelson et al., 2005).

Evidence suggests that the above model nodes communicate in a complex, bidirectional manner (Nelson et al., 2005); the relevant social areas and associated affective networks in the brain are similarly closely intertwined, with social processes informing emotional experience and the reverse (Blakemore & Choudhury, 2006). Relatedly, Nelson and colleagues (2005) suggest that the SIPN neuronal nodes may play important roles in the experience of mood symptoms in adolescents with an affective psychiatric disorder. As the hormone-driven shift in the affective node precedes the slower development of the cognitive node, emotional regulation may be particularly difficult for individuals with strong affective experiences. Thus, an inability to
control or inhibit responses in ways that are contextually appropriate may lead to behavioral
difficulties – and ultimately, general social difficulties – in adolescents with BD.

The described neurobiological functional deficits associated with early-onset BD are
likely exacerbated by the experience of mood symptoms, but continue to influence interpersonal
behavior between affective episodes. Although the current study could not speak to the possible
neurobiological underpinnings of demonstrated social deficits, the increasing feasibility of
incorporating neuroimaging techniques into psychiatric research will likely ease the integration
of behavioral and biological data in future studies of early-onset BD.

**Specific Aim #3: Longitudinal Predictors of Social Functioning**

Contrary to our hypothesis, our results did not reflect endurance of social impairments
over time. Instead, measures of social functioning collected at intake into the original FFT-A
treatment study were unrelated to current social functioning in participants with early-onset BD.
This contrasts with previous research, which generally suggests that social functioning remains
longitudinally consistent in patients with a mood disorder (e.g., Cole et al., 1996; Weinstock &
Miller, 2008). As effect sizes were limited for these analyses ($R^2 < 0.17$ for all), our small sample
size ($N=22$) – and subsequently, reduced statistical power – is not likely the sole explanation for
non-significant findings (post hoc analyses indicated that a sample size of 55 subjects would
provide 90% statistical power to detect an effect size of $R^2=.17$, using $\alpha = .05$). Instead, our
results likely reflect methodological aspects of our study, including the use of different measures
to assess social functioning (i.e., CBCL versus MESSY, UCLA-SAS, and Youth SAS-SR),
discrepant respondents (i.e., parent-reported social functioning at FFT-A study intake versus self-
reported social functioning in the current study), during developmentally distinct periods (i.e.,
average age 15.5 years vs. 19.07 years).
As prior research indicating the consistency of social functioning has included a briefer follow-up period than that used with the present study (i.e., one year or less, compared to approximately 3.5 years), it remains possible that our findings reflect a genuine independence between social functioning during mid-adolescence and early adulthood. More specifically, the use of particular social behaviors (assessed by the CBCL subscales used in this study) may not be related to later use of specific social behaviors or relationship quality over extended timeframes. Longitudinal studies using multiple measures over lengthier assessment periods are needed to determine the consistency of social functioning in early-onset BD.

Previous research has suggested that family functioning variables may longitudinally predict affective symptoms in early-onset BD (e.g., Geller, Tillman, Bolhofner & Zimerman, 2008). To the author’s knowledge, the current study is the first longitudinal demonstration of the predictive power of family functioning regarding social functioning in BD adolescents. Intake measures of family relationships (i.e., FACES-II cohesion), as reported by both parents and teens, predicted patients’ current use of social skills (i.e., Total MESSY Scores). Perhaps relationships within more cohesive families provide healthy models for appropriate interpersonal behavior, leading to the successful development of adolescent social skills. Conversely, strained or rigid parental relationships in a home environment - likely also characterized by high EE (Sullivan & Miklowitz, 2010) - may undermine an adolescent’s social confidence and increase the likelihood of social withdrawal or isolation, subsequently reducing opportunities to learn social behavior. The specific influence of family cohesion on self-reported use of social skills, and not other measures of social functioning used in our analyses, demands further study.

Helsen et al. (2000) reviewed three models outlined in the literature relating the quality of family and peer relationships for typical adolescents. In the compensation model, quality of
family and peer relationships are related inversely - those who receive little or no support from family relationships will ‘overcome’ by turning to their friendships to receive additional support. The reinforcement model suggests that those with high-quality familial relationships will be able to develop high-quality peer relationships; those with poor family relationships will be less likely to develop supportive relationships with peers. According to the additive model, family and peer relationship quality is independent of each other. Although they have been the frequent topic of theoretical debate, none of these models has consistently received empirical support.

In their study of family and peer support and well-being in adolescence, Helsen and colleagues provided some support for the additive model, as measures of peer and family support did not significantly correlate over time. However, the relationship between peer support and subjects’ well-being was not independent of family support. Instead, their findings suggested a complex relationship in which family support may moderate the relationship between peer relationships and participants’ well-being. Ultimately, the authors concluded that family relationships were the most important factor in participants’ self-reported well-being; levels of familial support influenced participant well-being, independent of peer support, in subjects ranging in age from early adolescence into early adulthood.

In combination with our current study, these findings suggest that familial and peer environments may be closely interconnected, even as individuals with early-onset BD enter late adolescence and early adulthood. Our findings may provide support for the reinforcement model detailed above, and suggest that individuals with early-onset BD are most successful with peer relationships when they have appropriate, healthy models of supportive relationships in the home. Relationships established within the family throughout childhood and adolescence may
have a more enduring and far-reaching impact on peer friendships, and potentially overall well-being, than previous appreciated.

Given indications of the longitudinal endurance of family cohesion and adaptability (Sullivan, Judd, Axelson & Miklowitz, 2012), and adolescent social functioning (although not replicated in the present study), we might expect that this relationship between family functioning and interpersonal behavior would remain fairly consistent; however, a recent study of concurrent family and social functioning demonstrated independence between family cohesion, adaptability, and conflict, and BD teens’ peer relationship quality (Keenan-Miller et al., 2011). Again, longitudinal analyses including multiple assessments of these variables may be better able to examine this relationship between family functioning and social behavior in early-onset BD.

Specific Aim #4: Social Functioning at Intake as Predictor of Current Symptoms

Finally, we examined the longitudinal relationships between social functioning at intake into the original treatment study and patients’ current affective symptoms. Contrary to hypothesis, current levels of depression were predicted by previous levels of depression, and were unrelated to prior measures of social functioning (effect size range for non-significant models: $R^2 < .04$ for all). While our data did not support Weinstock and Miller’s (2008, 2010) hypothesis, research including subjects with unipolar depression has generally suggested that greater social impairment longitudinally predicts greater severity of depressive symptoms (e.g., Cole et al, 1996; Lewinsohn, Rohde, Seeley, Klein & Gotlib, 2000; Panak and Garbar, 1992; Wierzbicki and McCabe, 1988). Their hypothesis also garners theoretical support from etiological models of depression. For example, Lewinsohn’s (1975) model suggests that depression results from a deficit in ‘response contingent reinforcement,’ which is likely
diminished by impaired social skills; similarly, McLean (1976) posits that depression results from an inability to influence one’s social environment. Certainly, the theoretical underpinnings of Interpersonal Therapy (IPT) rest on this hypothesis that social functioning relates directly to depressive trajectory (Mufson, Weissman, Moreau & Garfinkel, 1999; Mufson Dorta, Moreau & Weissman, 2004), as emphasized when it is described as a preventative treatment for depressive symptoms (Young, Mufson & Davies, 2006).

Limitations

Results from this study are best understood within the context of several limitations. First, the generalizability of our results may be limited due to characteristics of our sample. Both subject groups included little ethnic or racial diversity, and were limited in size. Control subjects were recruited exclusively from a community generally characterized by high educational attainment, achievement orientation, and socioeconomic status. Although rates of substance use (Wilens et al., 2004), social anxiety (Pini et al., 2006), and other comorbid diagnoses (Lewinsohn et al., 1995) were within expected ranges for our patient group, it is likely that the BD participants were generally higher-functioning, as a number of patients (N=8) declined participation or were unable to be contacted due to ongoing residential treatment or excessive affective symptoms (i.e., “John is too ill to complete an interview”). While inclusion of higher-functioning patients with early-onset BD may negatively impact generalizability, it increases our confidence in reported group differences in social functioning, as we would anticipate that the inclusion of lower-functioning patients would likely strengthen the noted group discrepancies.

Although our use of both cross-sectional and longitudinal data is a methodological strength of the current study, our use of two time points limited the complexity of the relationships between study variables that we could measure (for example, tests for curvilinear
changes over time). In addition, our current study measured the concurrent relationship between symptoms and social functioning at a single point in time. Inclusion of multiple assessments for each participant, completed when patients were symptomatic and asymptomatic, would permit a more thorough examination of the relationship between affective symptoms and social functioning. More specifically, longitudinal studies utilizing more frequently-collected affective (e.g., ratings of depressive and manic symptoms) and social (e.g., number of completed recreational activities, ratings of friendship quality, etc.) data will be better equipped to clarify the complex relationship between affective symptoms and interpersonal impairment in early-onset BD. Emerging methodologies, such as data collection via weekly text messaging (Bopp, Miklowitz, Goodwin, Stevens, Rendell & Geddes, 2010), may be particularly useful in these endeavors.

All study interviews were conducted by the author, who was not blinded to participant group when completing subject assessments. As such, it is possible that social functioning data gathered by the UCLA-SAS interview was biased by knowledge of patient diagnosis. Although findings reflected frequent agreement between analyses using UCLA-SAS and self-report measures (e.g., group comparisons), replication of these findings would benefit from the use of assessors who were blind to subject group membership.

It was a challenge to assess social functioning in participant groups including both adolescents and young adults. Traditionally, measures of interpersonal functioning have been designed for use with either child and adolescent or adult populations. The measures used in the present study were selected based on previous use with similar populations (e.g., Goldstein et al., 2006; Weinstock & Miller, 2008). However, our results should be interpreted with some caution, as subjects’ ages sometimes exceeded those for which study questionnaires were designed
(specifically, the MESSY and Youth SAS-SR). The study of social development in early-onset BD would be greatly aided by the availability of measures assessing both adolescent and adult behavior. The clear need for longitudinal research in this area – which would be considerably strengthened by use of consistent measures across time intervals – highlights the need to develop social functioning assessments that can be used across broader age ranges.

The current study included use of self-report and interview measures of social functioning. Ultimately, our understanding of social functioning in BD will be enhanced by the incorporation of data from multiple assessment methods, including self-, other-, and clinician-report, as well as behavioral observations. In-vivo observations of social behavior (e.g., Interactional Skills Ratings; Becker and Bellack, 1987), or tasks that place demands on adolescents’ abilities to flexibly and spontaneously use learned social skills (e.g., The Social Performance Rating Scale; Trower, Bryant & Argyle, 1978) have been underutilized in research with BD populations, and would likely provide unique contributions to our understanding of social impairment among BD patients.

Implications for Treatment

Our results add to the literature suggesting that social impairment is an important treatment target for patients with early-onset BD, distinct from the traditional focus on symptom reduction and relapse prevention (Keenan-Miller et al., 2011). Although some studies have suggested that social impairment does improve over time with the reduction of depressive and manic symptoms, the direction of these relationships is not clear (Sullivan, Miklowitz & Axelson, 2011). In addition, treatment studies have demonstrated that patients with improved medication adherence and reduced severity of affective symptoms frequently continue to
experience poor social outcomes (e.g., Goldstein, Axelson, Birmaher & Brent, 2007; Tsai, Chen, Kuo, Lee, Lee & Strakowski, 2001).

Effective treatment programs have been developed to address social skill deficits for adult patients with schizophrenia (e.g., Social Skills Training for Schizophrenia; Bellack, Mueser, Gingerich, & Agresta, 1997). These protocols typically focus on very specific, concrete skills that improve patients’ ability to navigate various aspects of social functioning (e.g., maintaining appropriate hygiene, keeping appropriate eye contact, starting a conversation with an unfamiliar person). It is unclear, however, if highly-concretized, basic skill-oriented protocols would be the most effective method to improve social functioning for individuals with early-onset BD. These programs traditionally focus on the development of fundamental prosocial skills; our current findings suggest that those with early-onset BD do not significantly differ from controls regarding use of appropriate social skills.

Instead, those with early-onset BD may be most effectively treated with protocols targeting the complex social skills required to develop and maintain close friendships. Dialectical behavior therapy (DBT; Linehan, 1993), an evidence-based, skill-focused treatment that includes modules focused on interpersonal effectiveness, emotion regulation, mindfulness and distress tolerance. Although the protocol has been shown to effectively improve functioning in a range of patient populations, it has only recently been used in the treatment of early-onset bipolar disorder. Goldstein and colleagues (2007) enrolled ten adolescents with BD in a year-long open trial of DBT; participation in the DBT treatment (which included both individual and family skills training sessions) was associated with improvements in parent-reported emotion regulation in BD patients, as well as reductions in suicidality, nonsuicidal self-injurious behavior, and depressive symptoms. Interpersonal improvements were also noted in self- and parent-report
measures (Cohen’s $d = 0.2-0.4$), although these findings were not statistically significant, given the study’s small sample size. The authors note that these areas of improvement mirror those reported in trials of DBT with adult and adolescent populations with other psychiatric diagnoses (e.g., unipolar depression, Rathus & Miller, 2002), suggesting that DBT may be effectively used with adolescents with BD.

Patient stigma may be an additional fruitful treatment target for early-onset BD populations. Perlick et al., (2001) suggested that the stigma associated with a BD diagnosis resulted in decreased assurance in social situations and social isolation. Relatedly, a number of participants in the current study commented on their negative response to receiving a BD diagnosis, and the resulting lack of confidence and social impairment (e.g., “After I was diagnosed in 6th grade, I didn’t want to talk with anyone. I was super shy because I was embarrassed.”). Although currently few in number, several treatments have been developed to specifically target patient self-stigma. Use of group treatment protocols has been successful in the reduction of stigma in adult patients with schizophrenia (Fung, Tsang & Cheung, 2011; Shin & Lukens, 2002). In both studies, reductions in stigma were associated with additional treatment gains (e.g., increased self esteem, decreased symptom severity), although post-treatment maintenance of these gains was limited (Fung et al., 2011) or untested (Shin et al., 2002). Participation in an online psychoeducational or cognitive behavioral skills program was associated with a reduction in stigmatizing attitudes among adults with depressive symptoms (Griffiths, Christensen, Jorm, Evans & Groves, 2004). Thus, additional research may indicate that the reduction of self-stigma is a fruitful target for the reduction of social impairment in early-onset BD (Brohan, Gauci, Sartorius & Throncroft, 2011).
Reduction of self-stigma may also be achieved via acceptance-based protocols. Acceptance and Commitment Therapy (ACT; Hayes, Strosahl & Wilson, 1999), as described by Hayes and colleagues (2006), does not focus on change of unwanted thoughts or circumstances, but instead encourages the patient to engage in a fulfilling life that is more in line with self-determined values. In the present study, patients made spontaneous comments connecting their acceptance of their bipolar diagnosis and their social functioning. Specifically, a number of patients noted that they felt able to engage socially only once they felt “ok with my moods.” One participant noted, “When I first told friends….they couldn’t deal with it because I couldn’t deal with it. My acceptance led to being able to explain it. Once I wasn’t hateful of myself for having it, I could talk about it” (additional patient comments regarding the relationship between BD diagnosis and social functioning are provided in Appendix C).

There are currently no published investigations of the use of ACT with BD adult or adolescent populations. In addition, studies of ACT typically report symptom reduction or decrease in experiential avoidance (e.g., school refusal), and have rarely included specific measures of social functioning. However, preliminary results suggest that participation in ACT may improve psychosocial functioning among adult schizophrenic patients (Gaudiano & Herbert, 2006), adult (McCracken, Vowles & Eccleston, 2005) and pediatric (Masuda, Cohen, Wicksell, Kemani, & Johnson, 2011; Wicksell, Melin, Lekander & Olsson, 2009) patients with chronic pain, and adolescents with subclinical anxiety (Brown & Hooper, 2009). For use with early-onset BD populations, ACT protocols including family participation may be particularly effective (Coyne, McHugh & Martinez, 2011).

Finally, our findings also suggest that treatments aimed at increasing family cohesion may improve social functioning of individuals with early-onset BD. However, few protocols
have been developed to specifically target family cohesion, and the variable has very rarely been included as an outcome variable in treatment studies. Instead, levels of family ‘togetherness’ are typically included in models as covariates (e.g., moderators, mediators, etc.) predicting psychiatric symptoms or other outcome variables (e.g., Cumsille & Epstein, 1994; Lewandowski, Verdelli, Wickramaratne, Warner, Mancini & Weissman, 2013; Lucia & Breslau, 2006). Longitudinal, repeated assessment of family cohesion within the context is quite rare; one recent study including adolescents with early-onset BD suggested that levels of family cohesion remained consistent throughout participation in FFT-A (Sullivan et al., 2012). Additional research is needed to identify protocols that support and enhance the development of cohesion within a family.

**Conclusion**

Study assessments indicated notable, wide-reaching deficits in the social functioning of patients with early-onset BD. The severity of these social impairments was largely independent on concurrent affective symptom severity and prior measures of social functioning. Although not directly ascertained from study measures, patients’ comments reflected a lack of social confidence, frequent experience with peer rejection, and avoidance of social environments. Certainly, the negative impacts of loneliness are widely appreciated. As noted by Mother Theresa:

> There is much suffering in the world – physical, material, mental…. The material and physical suffering is suffering from hunger, from homelessness, from all kinds of diseases. But the greatest suffering is being lonely, feeling unloved, having no one. I have come more and more
to realize that it is being unwanted that is the worst disease that any human
being can ever experience (p. 55).

The combination of social isolation with the severe, rapid-cycling mood symptoms in early-onset
BD creates a particularly challenging experience in this population. As noted by one study
participant, “[Bipolar symptoms] destroyed the majority of ties I had. I was described as a very
unlovable person.”

The present study contributes to the rapidly-expanding literature that emphasizes the need
to consider social impairments as a distinct treatment target in BD populations. Analyses suggest
that improvement of depressive symptoms or family cohesiveness may lead to improved use of
social skills. It remains unclear which variables are most likely to influence BD patients’
engagement in social recreational activities, friendship quality, or satisfaction with romantic
relationships. Research designed to clarify the complex relationships between social impairment,
affective symptoms, and family functioning will be an important next step towards the
development of informed treatment protocols with this population. The traditional focus on
affective symptom reduction does not adequately address all domains of impairment in early-
onset BD; a combination of treatment targets that includes improvement of social skills, peer
relationship quality, and social engagement will more effectively improve the quality of life of
adolescents and young adults with BD.
REFERENCES


Biederman, J., Kwon, A., Wozniak, J., Mick, E., Markowitz, S., Fazio, V. & Faraone, S.V. 

Clinical features and pathogenesis. In K.I. Shulman, M. Tohen & S.P. Kutcher (Eds.), Mood 

executive function and social cognition. *Journal of Child Psychology and Psychiatry, 47*(3), 
296-312.

The longitudinal course of bipolar disorder as revealed through weekly text messaging: A 


Brambilla, P., Nicoletti, M., Sassi, R.B., Mallinger, A.G., Frank, E., Keshavan, M.S. & Soares, 
*Journal of*

Brohan, E., Gauci, D., Sartorius, N. & Thornicroft, G. (2011). Self-stigma, empowerment and 
perceived discrimination among people with bipolar disorder or depression in 13 european 

the threshold: The developing adolescent* (pp. 277-307). Cambridge, MA: Harvard 
University Press.


risk youth: Links to child depression status and longitudinal course. *Journal of Clinical Child and Adolescent Psychology, 38*(1), 36-47.


Wilens, T.E., Biederman, J., Kwon, A. Ditterline, J., Forkner, P., Moore, H….Faraone, S.


### Appendix A  
Correlations Between Study Variables by Subject Group

Table A1. Correlations Between Intake and Follow-Up Variables Among Participants with Bipolar Disorder

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<th>Variable</th>
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<td>1. Gender</td>
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<td>2. Age</td>
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<td>3. Follow-up DRS</td>
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<td>4. Follow-up MRS</td>
<td>.26</td>
<td>-.02</td>
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<td>5. Total MESSY score</td>
<td>-.47*</td>
<td>.05</td>
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<td>6. UCLA-SAS Impairment score</td>
<td>-.14</td>
<td>-.05</td>
<td>.23</td>
<td>.08</td>
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<td>7. Youth-SAS Friend score</td>
<td>-.21</td>
<td>.13</td>
<td>.18</td>
<td>.00</td>
<td>.46*</td>
<td>.31</td>
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<td>8. DRS at FFT-A study intake</td>
<td>.17</td>
<td>.11</td>
<td>.39*</td>
<td>-.18</td>
<td>.22</td>
<td>.11</td>
<td>-.01</td>
<td>1.0</td>
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<td>9. MRS at FFT-A study intake</td>
<td>.15</td>
<td>.01</td>
<td>-.01</td>
<td>.19</td>
<td>-.16</td>
<td>-.01</td>
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<td>10. CBCL-Social Syndrome Scale</td>
<td>-.38</td>
<td>.06</td>
<td>.13</td>
<td>-.19</td>
<td>.32</td>
<td>.19</td>
<td>.41</td>
<td>.00</td>
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<tr>
<td>11. CBCL-Social Competence Scale</td>
<td>-.13</td>
<td>-.18</td>
<td>-.07</td>
<td>-.24</td>
<td>-.20</td>
<td>-.19</td>
<td>-.33</td>
<td>-.07</td>
<td>-.27</td>
<td>-.51*</td>
<td>1.0</td>
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<tr>
<td>12. FACES-II Cohesion – parent</td>
<td>-.02</td>
<td>.08</td>
<td>-.23</td>
<td>-.25</td>
<td>-.53*</td>
<td>-.04</td>
<td>.03</td>
<td>-.16</td>
<td>.28</td>
<td>.02</td>
<td>-.05</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. FACES-II Cohesion – teen</td>
<td>.22</td>
<td>.11</td>
<td>-.42*</td>
<td>.27</td>
<td>-.56*</td>
<td>-.03</td>
<td>-.04</td>
<td>-.59*</td>
<td>.31</td>
<td>.03</td>
<td>-.32</td>
<td>.42*</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. FACES-II Adapt – parent</td>
<td>.24</td>
<td>.19</td>
<td>-.01</td>
<td>.09</td>
<td>-.23</td>
<td>-.08</td>
<td>.05</td>
<td>.00</td>
<td>.16</td>
<td>-.18</td>
<td>-.06</td>
<td>.53*</td>
<td>.36</td>
<td>1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. FACES-II Adapt – teen</td>
<td>.31</td>
<td>.08</td>
<td>-.42*</td>
<td>.27</td>
<td>-.47*</td>
<td>.07</td>
<td>-.11</td>
<td>-.53*</td>
<td>.05</td>
<td>-.15</td>
<td>-.22</td>
<td>.13</td>
<td>.84*</td>
<td>.23</td>
<td>1.0</td>
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<tr>
<td>16. CBQ – parent</td>
<td>-.26</td>
<td>-.48*</td>
<td>.03</td>
<td>-.07</td>
<td>.25</td>
<td>-.08</td>
<td>.06</td>
<td>-.08</td>
<td>-.14</td>
<td>.38</td>
<td>-.23</td>
<td>-.28</td>
<td>-.36</td>
<td>-.49*</td>
<td>-.33</td>
<td>1.0</td>
</tr>
<tr>
<td>17. CBQ – teen</td>
<td>-.22</td>
<td>-.27</td>
<td>.33</td>
<td>-.29</td>
<td>.14</td>
<td>-.25</td>
<td>.00</td>
<td>.35</td>
<td>-.19</td>
<td>.24</td>
<td>.35</td>
<td>-.21</td>
<td>-.67*</td>
<td>-.39</td>
<td>-.76*</td>
<td>.57*</td>
</tr>
</tbody>
</table>

DRS = Kiddie Schedule for Affective Disorders Depression Rating Scale; MRS = Kiddie Schedule for Affective Disorders Mania Rating Scale; MESSY = Matson Evaluation of Social Skills with Youngsters; UCLA-SAS = UCLA Social Attainment Survey; Youth-SAS = Youth Social Adjustment Scale – Self Report, Short Form Score from Friendship Subscale; FFT-A = Family-Focused Treatment for Adolescents; FACES-II = Family Adaptability and Cohesion Evaluation Scales, II; CBQ = Conflict Behavior Scale.
Table A2. Correlations Between Study Variables Among Control Group Participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Gender</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Age</td>
<td>.06</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Follow-up DRS</td>
<td>.09</td>
<td>.14</td>
<td>1.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Follow-up MRS</td>
<td>-.18</td>
<td>-.04</td>
<td>-.17</td>
<td>1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Total MESSY Score</td>
<td>-.36*</td>
<td>-.19</td>
<td>.25</td>
<td>-.04</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>6. UCLA-SAS Impairment score</td>
<td>-.19</td>
<td>-.45*</td>
<td>.09</td>
<td>-.35</td>
<td>.43*</td>
<td>1.0</td>
</tr>
<tr>
<td>7. Youth-SAS Friend Score</td>
<td>-.04</td>
<td>-.08</td>
<td>.15</td>
<td>.09</td>
<td>.66*</td>
<td>.25</td>
</tr>
</tbody>
</table>

DRS = Kiddie Schedule for Affective Disorders Depression Rating Scale; MRS = Kiddie Schedule for Affective Disorders Mania Rating Scale; MESSY = Matson Evaluation of Social Skills with Youngsters; UCLA-SAS = UCLA Social Attainment Survey; Youth-SAS = Youth Social Adjustment Scale – Self Report, Short Form Score from Friendship Subscale;
APPENDIX B
Path Analyses

Figure B1. Path analysis of parent-reported FACES-II cohesion and additional covariates predicting Total MESSY Score

DRS = Depression Rating Scale; MRS = Mania Rating Scale; CBCL Social Functioning at Intake = single variable combining Child Behavior Checklist Social Competence and Social Problem subscales; FACES-II Cohesion = Family Adaptability and Cohesion Evaluation Scales – II; Cohesion Subtotal; MESSY = Matson Evaluation of Social Skills in Youngsters
Figure B2. Path analysis of self-reported FACES-II cohesion and additional covariates predicting Total MESSY Score

DRS = Depression Rating Scale; MRS = Mania Rating Scale; CBCL Social Functioning at Intake = single variable combining Child Behavior Checklist Social Competence and Social Problem subscales; FACES-II Cohesion = Family Adaptability and Cohesion Evaluation Scales – II; Cohesion Subtotal; MESSY = Matson Evaluation of Social Skills in Youngsters
## Appendix C
Comments Regarding the Relationship Between Social Functioning and Bipolar Disorder

Table C1. Positive Responses to the open-ended question: Do you believe there is a relationship between your bipolar symptoms and your social relationships?

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Participant Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>21</td>
<td>“Yeah – the biggest thing is that [friendships] initially start out great. But it seems like the more they get to know me, the more they find out about my instability, and then they really clearly back off.”</td>
</tr>
<tr>
<td>F</td>
<td>22</td>
<td>“It was definitely a factor…I think [having bipolar disorder] makes me less comfortable in new situations and keeps me from meeting new people. Sometimes it’s hard to feel like others understand. I don’t want to have to always explain why I’m not doing something. They think I’m just blowing them off. But that’s not always the case. Sometimes it’s too much to handle to go out with people.”</td>
</tr>
<tr>
<td>F</td>
<td>20</td>
<td>“I don’t have any friends, but it’s because I’m on these meds now. [The side effects] really get in the way of everything. I have no idea how to make friends now.”</td>
</tr>
<tr>
<td>F</td>
<td>23</td>
<td>“Yes. When I’m manic, I want to be around people, and I love attention. Friends don’t like how [mania] makes me really competitive. I get super social…but when I’m depressed I totally withdraw. When I go from being so socially out there to not returning a call, I can see why people get pissed. They think I’m shady or creepy….and they don’t really want to get close after that.”</td>
</tr>
<tr>
<td>F</td>
<td>19</td>
<td>“My irritability has really kept me away from people, I think. I’m very particular about who I spend time with. …If I’m worried about my mood in a [social] situation, I’ll just stay away.”</td>
</tr>
<tr>
<td>M</td>
<td>21</td>
<td>“Before a year ago, [mood symptoms] destroyed the majority of ties I had. I’d been described as a very unlovable person. I agreed with that. I was really struggling, and I needed to make better decisions.”</td>
</tr>
<tr>
<td>F</td>
<td>18</td>
<td>“They’re absolutely related. Changes in mood are immediately noticeable to friends. My emotions control each of my relationships. When I’m irritable, I can’t keep from snapping and people just stay away. It has made me more on guard and reluctant to let people in. I’ve learned it’s best for me to keep people at a distance. They have problems understanding my moods.”</td>
</tr>
<tr>
<td>M</td>
<td>17</td>
<td>“[Mood symptoms have] gotten in the way sometimes – I’ll just disconnect socially. Friends must be aware of [mood swings] at some level, but I haven’t told them or anything.”</td>
</tr>
<tr>
<td>M</td>
<td>20</td>
<td>“Yeah – I pretty much destroyed my phone when I was manic, and now I don’t have anyone’s phone numbers any more. I don’t really make friends anymore now anyways. When I’m gone for a while, like if I’m hospitalized, people wonder ‘where is he?!’ Someone has to come up with an answer and it’s awkward.”</td>
</tr>
<tr>
<td>F</td>
<td>18</td>
<td>“Yep. Friendships are hard – I have enough emotions for both of us.”</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>Statement</td>
</tr>
<tr>
<td>---</td>
<td>-----</td>
<td>-----------</td>
</tr>
<tr>
<td>M</td>
<td>16</td>
<td>“Mood stuff is definitely triggered by social problems, for sure. Being lonely is a huge part of it, too. It can be isolating having bipolar disorder. People would stay away and I would get upset, and I would get upset and moody, which made them stay away more.”</td>
</tr>
<tr>
<td>F</td>
<td>22</td>
<td>“Without a doubt they’re related. In dealing with other people, bipolar influences my actions, reactions, beliefs and principles”</td>
</tr>
<tr>
<td>M</td>
<td>22</td>
<td>“They do tend to be related. Some emotions may be understood by some people some of the time, but lots of people don’t get it. Swings in mood tend to be pretty quick, and are usually associated with anger. That doesn’t usually go over well with people. My long-term friend just seems to understand it and we balance each other out. I get him, too. I’ve told him about it and he knows me and how I am sometimes.”</td>
</tr>
<tr>
<td>F</td>
<td>17</td>
<td>“I think a lot of my social problems stem from bipolar disorder. The mood swings have made it really hard for me to make friends. When I change too much, people have said, ‘Did someone die?’ or ‘Why are you so hyper??!’ or ‘I don’t know how to deal with you.’”</td>
</tr>
<tr>
<td>F</td>
<td>17</td>
<td>“Yeah – but the people I surround myself with know that I can flip on a switch. So they get it. It was harder when I was younger – now I have an ability to keep my moods under a little better control.”</td>
</tr>
<tr>
<td>F</td>
<td>19</td>
<td>“I’ve always tried to separate myself from my disorder – I don’t want to identify only with it. For me, bipolar disorder does not have a big influence on my social life right now. In the past, much more so…but right now [mood symptoms are] something I control, so it doesn’t affect things socially.”</td>
</tr>
<tr>
<td>M</td>
<td>21</td>
<td>“It’s associated with both challenges and strengths. My closest friends got closer because of it; casual friends get freaked out if I get…snippy and stay away. My romantic relationships have probably been shorter because of it – people don’t know what’s going on sometimes.”</td>
</tr>
<tr>
<td>F</td>
<td>19</td>
<td>“It has helped me in a sense –it feels like in some ways I’m closer with my friends because I do have bipolar disorder. It has made me stronger, but it made things really hard for a while. After I was diagnosed in 6th grade, I didn’t want to talk with anyone. I was super shy because I was embarrassed. I’ve had some hard times when I didn’t want to talk with people – but then as I gained confidence I started working through my shyness.”</td>
</tr>
<tr>
<td>F</td>
<td>18</td>
<td>“I think [bipolar disorder] has helped, actually. It made it easier to make friends after I did everything with the mood and learned skills. I was never that amazing at making friends, but moodiness made things waaaayy worse. I think working through it all was really helpful-not the mood stuff itself but being able to work through it boosted my self-confidence. A lot, actually. Being able to know that I could do things to improve my own mood was really useful. Confidence really followed my ability to help my mood out. Then I could make friends.”</td>
</tr>
<tr>
<td>F</td>
<td>21</td>
<td>“It took me until I was ok with [the bipolar diagnosis] before I was ok with telling others about it. When I first told friends, some people didn’t know how to deal with it. I think they couldn’t deal with it because I didn’t know how to deal with it. I put distance between us. My acceptance led to being able to explain it. Once I wasn’t hateful of myself for having it, I could talk about it”</td>
</tr>
</tbody>
</table>
Table C2. Negative Responses to the open-ended question: Do you believe there is a relationship between your bipolar symptoms and your social relationships?

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Participant Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>20</td>
<td>“No, I’ve found it to be quite independent from my mood. I can talk with my friends about [my symptoms] and he understands.”</td>
</tr>
<tr>
<td>F</td>
<td>18</td>
<td>“No, I don't think so. I don't think it’s my mood—I just don’t like being around people.”</td>
</tr>
<tr>
<td>F</td>
<td>22</td>
<td>“No, I don’t think they’re related at all.”</td>
</tr>
<tr>
<td>M</td>
<td>17</td>
<td>“My symptoms are weird and kind of sporadic – I don’t think they influence friendships at all.”</td>
</tr>
<tr>
<td>M</td>
<td>15</td>
<td>“I don’t think they’re related – I don’t think bipolar interferes with friends at all.”</td>
</tr>
</tbody>
</table>