

5-1-2022

## Family Functioning in Youth with Bipolar Disorder

Kayla Fobian

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FAMILY FUNCTIONING IN YOUTH WITH BIPOLAR DISORDER

By

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Bachelor of Science – Psychology  
Bachelor of Science – Human Development and Family Studies  
University of Connecticut  
2018

A thesis submitted in partial fulfillment  
of the requirements for the

Master of Arts – Clinical Psychology

Department of Psychology  
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The Graduate College

University of Nevada, Las Vegas  
May 2022



## **Thesis Approval**

The Graduate College  
The University of Nevada, Las Vegas

November 5, 2021

This thesis prepared by

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entitled

Family Functioning in Youth with Bipolar Disorder

is approved in partial fulfillment of the requirements for the degree of

Master of Arts – Clinical Psychology  
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## ABSTRACT

### **FAMILY FUNCTIONING IN YOUTH WITH BIPOLAR DISORDER**

By

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Family functioning encompasses a family's ability to maintain an environment that supports and benefits each member of the family. Among families of youth with a bipolar disorder (BD), family functioning is impaired compared to healthy controls. However, few studies have examined family functioning across psychopathology in youth. Additionally, few studies have examined which symptoms (depressive, manic, or externalizing) are most strongly associated with family functioning. Therefore, the purpose of this manuscript is to benchmark impairments in family functioning in youth with BD compared to youth with other psychiatric disorders and to examine the differential influence of depressive, manic, and comorbid externalizing behavior symptoms on family functioning in youth with BD. Youth with BD had more impaired family functioning compared to youth with behavior disorders and youth with non-mood, non-behavior disorders, but not youth with unipolar depression. Specifically, depressive and externalizing symptoms were most strongly associated with declines in both caregiver-reported and clinician-reported family functioning. (Hypo)manic symptoms were mostly unassociated with changes in family functioning. Depressive and externalizing symptoms appear to be driving declines in family functioning among youth with BD, making these symptoms critical targets for treatment of BD in youth.

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## CHAPTER 1

### LITERATURE REVIEW

Family functioning is a measure of how well a family maintains an environment that benefits and supports the members of that family (Otto, 1962; Walsh, 2012). Across severe mental illness, impaired family functioning is both common (Heru, 2000; Saunders, 2003) and associated with poorer outcomes (Jozefiak et al., 2019). Bipolar disorder (BD) is no exception, and youth with BD have impaired family functioning relative to healthy youth (MacPherson et al., 2018). As a result, most psychosocial treatments for BD target family functioning either indirectly (e.g., Knutsson et al., 2017) or directly (e.g., Miklowitz & Scott, 2009). However, the relationship between BD and family functioning is not clear. One potential pathway is that the presence of symptoms leads to impaired family functioning. Alternatively, impaired family functioning could lead to more severe BD symptoms. For youth with BD, this pathway is even more complex because youth typically experience manic, depressive, and mixed symptoms (Youngstrom et al., 2008). Additionally, youth with BD tend to have high rates of comorbidity with externalizing disorders (Frías et al., 2015), and externalizing symptoms and disorders such as attention deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD) and conduct disorder (CD) which are also associated with impaired family functioning (Weintraub et al., 2019). Therefore, identifying what (i.e., depressive, manic, or mixed symptoms or externalizing comorbidity) is driving impairment in family functioning in youth with BD is critical to informing mechanistically sound intervention pathways.

#### **Overview of Family Functioning**

Family functioning broadly encompasses how well a family environment aids individuals and the family in achieving a desired goal or outcome (Otto, 1962; Walsh, 2012). The specific tasks required of healthy families depends heavily on social and developmental factors and may vary widely across a family's timeline and between different families (Walsh, 2012). However, successful families do typically share the following hallmarks of family functioning that aid them in achieving their various goals: (a) encouragement of respectful communication, (b) adaptability of roles both within the family and with the surrounding community, (c) sensitivity to the needs of individual family members, and (d) the ability to provide family members with support and security (Otto, 1962; Walsh, 1994). The last two hallmarks can also be combined to describe family cohesion (i.e. the family's ability to balance closeness and connectedness of members along with a tolerance for individual differences; Walsh, 1994).

From these observations about healthy families, two general classes of family functioning models developed. Models such as the Circumplex Model of Marital & Family Systems (Olson et al., 1979) and Beavers System Theory (Beavers & Hampson, 2000) focus on the outcomes of family interactions. Outcome models postulate that family functioning is poor when interpersonal interactions result in poor outcomes, and they categorize families based on the general functioning outcomes expected to arise from the interpersonal processes within the family. In contrast, models such as the McMaster Model (Epstein et al., 1978) and the Process Model (Steinhauer et al., 1984) focus on the processes that family members use to interact with each other. Process models postulate that family functioning is poor when the interpersonal interactions themselves are poor, regardless of the outcome. These models conceptualize general functioning as a parallel domain separate from other interpersonal process domains, keeping focus on the process of family interactions rather than predicted general functioning outcomes

and categories. Irrespective of their outcome versus process orientation, family functioning models focus on similar core dimensions of how families interact.

Family functioning models tend to focus on communication, adaptability, and the quality of family interaction. Communication refers to the ways in which information is exchanged between family members. Good communication involves open, clear, and direct expression of a wide range of thoughts and emotions and responses that demonstrate empathy and tolerance. Adaptability centers on how well roles, ideals, and values can be adjusted in response to changes within the family and in the environment surrounding the family system. Quality of family interaction generally means the extent to which family members experience and express interest in, acceptance of, and care for one another. Therefore, despite differing in focus on the outcomes or the process of family interactions, family functioning is typically assessed by focusing on the following domains: communication patterns, the adaptability of the family system, and the quality of interactions between family members.

The importance of communication is central to all four models of family functioning. The Circumplex Model, the McMaster Model, and the Process Model all developed explicit domains outlining communication between family members (Mansfield et al., 2015; Olson et al., 2019; Steinhauer et al., 1984). These Communication domains establish the ways in which families share both affective and instrumental information. Assessments of Communication focus on the clarity of the information being communicated (i.e., whether the intended message is clear or is vague and ambiguous) and the directness with which information is conveyed (i.e. whether the information is given directly to the intended recipient or passed through several people before reaching the desired recipient). The McMaster and Process Model also contain additional domains that interweave communication into other aspects of family functioning. For example,

within the Values and Norms domain of the Process Model, the consistency with which a family's values and norms are agreed upon and upheld depends in part on the ability to successfully communicate these expectations to all members of the family (Steinhauer, 1987). Similarly, the Problem Solving domain of the McMaster Model highlights communication as an integral component of problem solving (e.g., discussing problems and whether different solutions worked; Epstein et al., 1983). The second step of problem solving considers with whom the problem is discussed and whether this was an appropriate resource to consult, establishing appropriate communication of problems as a vital component of successful family functioning. Though the Beavers System does not outline a specific communication domain, this model also weaves communication into both of its primary domains of family functioning - Family Competence and Family Style. The Beavers System defines optimal families as capable of group problem-solving, setting clear boundaries, and acknowledging and respecting the differing viewpoints among family members, all of which require successful communication (Beavers & Hampson, 2000). Therefore, among several models of family functioning, communication patterns help define how well a family is functioning.

Adaptability is another major focus in these four models of family functioning. The Flexibility and Family Competence domains of the Circumplex and Beavers Models, respectively, center around the notion that families and family roles need to adapt to the changing needs of family members and the external environment (Beavers & Hampson, 2000; Hampson et al., 1988; Olson et al., 1983, 2019). Within the McMaster Model, the ability of the family to integrate internal and external changes that influence the family system, to adjust the responsibility of family members, and to adjust the ways in which individuals are held accountable for their behavior is critical in assessing family success in the Roles and Behavior

Control domains (Epstein et al., 1978, 1983). The Task Accomplishment, Role Performance, and Control domains of the Process Model also emphasize the need for successful negotiation and adaptive flexibility within the family system (Skinner et al., 2000; Steinhauer et al., 1984). These domains demonstrate the critical need to adjust the important tasks that the family must fulfill, the roles that each family member plays in accomplishing these tasks, and how the expectations about the behavior of each family member is upheld as the needs of the family system and those within it change. Family structure, individual family members, and the environment surrounding the family are frequently changing, and the importance of adapting to these changes is central to assessing the functioning of families.

Lastly, the importance of quality interactions among family members is a critical component of several domains of these models of family functioning. The Cohesion and Family Style domains of the Circumplex and Beavers Models, respectively, include assessment of the emotional bonding among family members and the satisfaction individuals experience through interactions within the family system (Beavers, 1989; Olson, 2000; Olson & Gorall, 2003). In the McMaster Model and Process Model, bonding and satisfaction is more specifically broken down. The Affective Responsiveness and Affective Involvement of the McMaster Model emphasize the patterns of response to emotional stimuli and the extent to which the interests of family members are acknowledged and valued (Epstein et al., 1978; I. W. Miller et al., 1994). Similarly, the Affective Involvement domain of the Process model refers to both the degree to which family members express interest in one another and the extent to which interactions meet the emotional needs of family members (Steinhauer, 1987; Steinhauer et al., 1984). Overall, the quality of relationships and interactions within the family system is important in assessing the health of a family and is a critical component within several models of family functioning.

Family functioning has become an important target in the treatment of mental illness in youth. Impairments in family functioning (e.g., low parental warmth, high conflict, and low cohesion) are associated with increases in both internalizing and externalizing problems, poorer quality of life, and impaired psychosocial functioning (Freed et al., 2015; Jozefiak et al., 2019; Lau et al., 2018; Scully et al., 2019). Conversely, good family functioning is associated with better mental health in children (Scully et al., 2019). Family-focused therapies often target core features of family functioning, helping to improve processes such as communication, family flexibility (i.e. adaptability) and positive reframing of problems that likely mediate outcomes of therapy with families (MacPherson et al., 2016). Family-focused therapies are effective in the treatment of a variety of mental illnesses affecting youth including substance use (Hartnett et al., 2017), eating disorders (Couturier et al., 2013), behavior problem (van der Pol et al., 2017), and internalizing psychopathology (van der Pol et al., 2017). Therefore, improving the overall well-being of the family system can serve as a standalone treatment target instead of more traditional treatment targets that focus on an individual.

The relationship between family functioning and mental health is a bidirectional, interactional process where each affects the other across development (Silber, 1989). A child struggling with mental health difficulties may cause disruptions in family functioning. For example, hyperactive symptoms in youth predict greater family conflict, more negative and controlling maternal behavior, and less positive parent-child interactions up to eight years later (Barkley et al., 1991). However, disruptions in family functioning may also lead to a child's mental health difficulties. Among youth with attention deficit/hyperactivity disorder (ADHD), lower levels of parental involvement, parental sensitivity, and maternal positive regard and warmth are associated with more severe hyperactivity and inattention (Breaux & Harvey, 2019;

Hawes et al., 2013; Keown, 2012). Family functioning and youth mental health may also interact to support each other. For example, ODD symptoms in youth predict more negative parenting practices, and negative parenting practices predict more severe ODD symptoms over time (Burke et al., 2008). Therefore, within the context of mental illness, impaired family functioning could reflect either an outcome, cause, or co-occurring maintenance factor.

### **Family Functioning in Bipolar Disorder**

Youth with BD have impaired family functioning compared to healthy youth (MacPherson et al., 2018; Young et al., 2013). Youth with BD live in family environments marked by less cohesion, adaptability, warmth, affection, and intimacy than the family environments of healthy youth (Belardinelli et al., 2008; Nader et al., 2013; Schenkel et al., 2008; Sullivan & Miklowitz, 2010). Additionally, family cohesion declines as the length of illness increases (Belardinelli et al., 2008). Caregivers of youth with BD also report worse mental health than caregivers of youth without BD, including elevated depressive symptoms and parenting stress which could negatively impact the family environment (Algorta et al., 2018). Functionally, parents of youth with BD engage in more negative expressed emotion (Nader et al., 2013), and families of youth with BD generally experience poorer problem-solving, increased conflict, and greater use of forceful punishment compared to families with healthy youth (Belardinelli et al., 2008; Nader et al., 2013; Schenkel et al., 2008; Sullivan & Miklowitz, 2010). In summary, a BD diagnosis in youth is associated with greater impairment in family functioning when compared to healthy youth.

Impairments in family functioning are also common across other types of psychopathology (Friedmann et al., 1997). Understanding how family functioning varies across

different types of psychopathology may help providers target specific aspects of family functioning. Some literature has compared adults with BD to adults with other psychopathology. Among adults with severe mental illness, diagnosis and perceived severity of disorder do not significantly predict family functioning (Crowe & Lyness, 2014). Specifically, adults with BD experience similar impairments in family functioning compared to adults with unipolar depression (Friedmann et al., 1997; Weinstock et al., 2006), anxiety, eating disorders, and substance abuse (Friedmann et al., 1997). However, less literature has compared family functioning among youth with BD to youth with other psychiatric disorders. Whereas youth with BD report similar general family functioning compared to youth with ADHD, youth with BD also report a greater deficit in problem solving (Young et al., 2013). Additionally, youth with BD report similar family QoL compared to youth with depressive disorder (Freeman et al., 2009). However, youth with BD report worse family quality of life compared to youth with behavior, anxiety, and trauma disorders (Freeman et al., 2009). Therefore, evidence suggests that BD may not be more impairing than other more severe forms of psychopathology in adults, but more work is needed to determine whether family functioning varies across different types and severity of psychopathology among youth.

#### *Family functioning as an outcome of symptoms*

Family functioning among youth with BD is typically conceptualized as an outcome of youth symptoms. One method for understanding whether various psychopathology differentially affects family functioning is to consider comorbidity. For example, youth with BD who have history of previous suicidal ideation or attempts have worse family functioning and family quality of life than youth with BD who do not have a history of suicidal ideation (Algorta et al., 2011). Youth with BD who previously attempted suicide report worse problem solving,

communication, and general family functioning compared to youth with no lifetime history of suicide attempts (Berutti et al., 2016). In addition to suicidality, symptoms such as hyperactivity, irritability, misery, and withdrawal were associated with the highest burden on caregivers for adults with BD (Reinares et al., 2006). Among youth with BD, more severe depression symptoms are also related to poorer family relationships (Keenan-Miller et al., 2012) and poorer family QoL (Freeman et al., 2009). In summary, cross-sectional studies indicate that the presence of comorbidity through additional symptoms or suicidality is associated with more impaired family functioning.

Unlike cross-sectional studies, longitudinal studies provide an opportunity to identify the temporal ordering of symptoms and family functioning. Families who have a parent with BD report worsening cohesion, adaptability, and conflict across time (Shalev et al., 2019). Two potential mechanisms for why having a parent with BD decreases family functioning have been supported. Declines in parental functioning partially explains why families with a parent with BD have poorer family functioning than families without BD (Shalev et al., 2019). Among these families, having a child with BD is associated with even greater declines in family functioning which also partially explains the relationship between parents with BD and declines in family functioning (Shalev et al., 2019). Among youth with BD, the presence of comorbid anxiety, ADHD, and disruptive behavior disorders predict greater family conflict over time (Weintraub et al., 2019). Youth with BD who report increased energy also reported worse family functioning compared to youth with BD who report only irritability. However, as energy levels returns to normal for the youth reporting increased energy, the differences in family functioning also decline between youth with increased energy and youth with only irritability (Frazier et al.,

2020). In summary, impairments in family functioning may be an outcome of the presence or severity of BD in youth.

#### *Family functioning as a predictor of symptoms*

Family functioning has also been framed as a predictor of BD symptoms in youth. Greater family dysfunction is associated with a worse course of disorder for youth with BD. Increased rigidity (i.e., less adaptability to change) and poorer general family functioning predicts suicidal ideation in youth with BD (Algorta et al., 2011; Weinstein et al., 2015). Additionally, increased family conflict may act as an indirect pathway between the association of parental BD and the onset of offspring BD (Du Rocher Schudlich et al., 2008). Although conclusions on the directional relationship of these associations is limited due to cross-sectional research designs, the influence of family functioning on the course of BD has been further supported with longitudinal evidence. Low maternal warmth predicts more time in manic episodes over the course of several years (Geller et al., 2008), and high levels of stress in family relationships is associated with less improvement in mood symptoms over time (Kim et al., 2007). Additionally, high perceived parental burden, negative affective style, and expressed emotion increase the risk for future mood episodes (Perlick et al., 2001; Reinares et al., 2016). However, one study did find that current family functioning was not associated with subsequent changes in mood episodes (Uebelacker et al., 2006). In summary, family functioning can influence the course of mood symptoms and episodes among youth with BD.

#### *Family functioning and treatment outcomes*

Family functioning is also associated with treatment outcomes among youth with BD. Among youth receiving treatment for BD, high levels of family conflict prior to treatment are

associated with higher levels of mania over time, and high levels of cohesion and adaptability prior to treatment are associated with lower ratings of depression over time (Sullivan et al., 2012). Decreasing family conflict also predicts improvements in both mania and depression ratings across treatment; however, the rate of improvement for mania symptoms is slower in youth who report high conflict within their family (Sullivan et al., 2012). Therefore, family functioning may also act to precipitate and maintain BD symptoms, suggesting a bidirectional relationship between family functioning and BD symptom severity.

Many interventions targeting BD in youth incorporate aspects of family functioning such as family psychoeducation and communication skills building. Interventions that focus on these family dynamics and interactional processes often target the core dimensions of family functioning. For example, families receive instruction in effective communication, increasing positive and affirmative interactions (i.e., quality of family interactions), and improving problem solving and resource availability to better handle difficult changes (i.e., adaptability; Miklowitz et al., 2008; Miklowitz & Scott, 2009; Pavuluri et al., 2004). Family focused interventions are associated with more rapid recovery from initial depressive and manic symptoms, decreased caregiver burden, and improved quality of life for youth (MacPherson et al., 2016; Miklowitz et al., 2009; Miklowitz & Scott, 2009; Reinares et al., 2016; West et al., 2014). Specifically, family-focused therapy (FFT) is beneficial for reducing mania symptoms in both low- and high-conflict families (Sullivan et al., 2012), and both FFT and child- and family-focused cognitive behavioral therapy (CFF-CBT) improve family climate and psychosocial functioning in youth with BD (Knutsson et al., 2017; Reinares et al., 2016; Sullivan et al., 2012). The benefits of FFT and CFF-CBT also continue for months to years following the termination of treatment, as indicated by fewer rehospitalizations, more weeks in remission, more favorable symptom

trajectory, and better global functioning (Miklowitz et al., 2013, 2020; Miklowitz & Scott, 2009; Reinares et al., 2016; West et al., 2014). Overall, family-focused interventions can effectively improve youth psychosocial functioning, increase episode recovery rates, and improve the long-term course of BD, providing support for the importance of family functioning as a treatment target in youth with BD.

### **Family Functioning and Mood and Externalizing Symptoms**

Although there are clear patterns of dysfunction among youth with BD and family-focused interventions are empirically supported for treatment of BD, it is still unclear what symptoms are driving impairments in functioning for these families. BD consists of both internalizing symptoms (i.e., depressive symptoms) and externalizing symptoms (i.e., manic symptoms; Peters et al., 2018; Youngstrom et al., 2008). Additionally, youth with BD often experience mixed episodes of both depressive and manic symptoms (Youngstrom et al., 2008) and youth with BD tend to have comorbid externalizing disorders such as ADHD (Frías et al., 2015). The presence of depressive, manic, and externalizing behavior symptoms are all associated with worse family functioning in youth with BD (Pereira et al., 2015; Rosa et al., 2010; Weintraub et al., 2019). Poor family functioning also predicts a poorer course of depressive, manic, and externalizing behavior symptoms (Daches et al., 2018; Nelson et al., 2007; Sullivan et al., 2012). However, there is also some evidence that current mood state is not associated with family functioning in youth with BD (Young et al., 2013). Therefore, impairments in family functioning among youth with BD could be driven by depressive, manic, or externalizing behavior symptoms or by a combination of all three symptom domains.

#### *Depressive Symptoms*

Depressive symptoms may be one driver of family dysfunction in youth with BD. Families of youth with unipolar depression demonstrate patterns of impairment in family functioning similar to that found for youth with BD. Specifically, families of a child or adolescent with depression report lower communication, affective involvement and responsiveness, cohesion, and satisfaction and more family disengagement compared to families with healthy youth (Frazer & Fite, 2016; Kashani et al., 1995; Pereira et al., 2015; Simpson et al., 2018; Tamplin et al., 1998). Families of depressed adolescents also exhibit greater expressed emotion, indicating a more critical and emotionally overinvolved family environment (Asarnow et al., 1994). Among youth with depression, comorbid disruptive behavior disorders are associated with even higher critical expressed emotion (Asarnow et al., 1994) and predict worse affective involvement, communication, and problem solving compared to youth with depression alone (Tamplin et al., 1998). Furthermore, whereas youth with depression report less conflict and aggression compared to healthy youth (Pereira et al., 2015), youth with BD experience greater family conflict associated with externalizing behaviors, such as aggression (Belardinelli et al., 2008; Keenan-Miller et al., 2012; Nader et al., 2013). Therefore, depressive symptoms alone may not be enough to explain family dysfunction in youth with BD.

Family functioning can also act as a predictor of depressive symptoms in both youth with unipolar depression and youth with BD. In youth with unipolar depression, worse family functioning is positively correlated with depression symptom severity (Burnett et al., 2017; Daches et al., 2018; Sawyer et al., 2015). Similarly, in youth with BD, ratings of family cohesion, adaptability, and conflict tend to correlate with depression severity over time (Sullivan et al., 2012). Overall, impairments in family functioning may be a result of depressive symptoms as well as a facilitator of depression symptom severity over time in youth with BD.

### *Manic Symptoms*

The association between manic symptoms and family functioning has received much less attention. Manic symptoms are associated with impaired family functioning (Rosa et al., 2010). However, depressive symptoms cause more disruption in psychosocial functioning and family functioning than manic symptoms in youth with BD (Calabrese et al., 2004; Rosa et al., 2010). Changes in family functioning are also associated with changes in manic symptoms such that decreases in parent-reported conflict predict decreases in manic symptoms over time (Sullivan et al., 2012). Therefore, manic symptoms also appear to have an interactional relationship with family functioning in youth with BD.

### *Externalizing Behavior Symptoms*

Youth with BD tend to display high rates of comorbid behavior disorders, including ADHD and disruptive behavior disorders (Findling et al., 2001; Frías et al., 2015). Behavior disorders are associated with impairments in family functioning (Deault, 2010; Harvey et al., 2011; Nelson et al., 2007). ADHD is associated with worse general family functioning, greater family stress, conflicted parent-child relationships, worse problem solving, poorer communication, and less affective responsiveness and involvement (Deault, 2010; Kandemir et al., 2014). Disruptive behavior disorders are also associated with more severe depressive symptoms and greater family conflict (Weintraub et al., 2019). Additionally, the severity of externalizing symptoms is associated with impairments in family functioning. Additional oppositional and conduct problems in youth with ADHD increase conflict and negative parenting practices compared to ADHD symptoms alone (Deault, 2010; Schei et al., 2016) and children

with low prosocial behavior and high behavioral difficulties have worse parent-reported family functioning (Renzaho et al., 2013).

In youth with BD, comorbid externalizing disorders may also impact family functioning. Comorbid ADHD and disruptive behavior disorders are associated with more severe (hypo)manic and depressive symptoms, greater family conflict, and lower family cohesion in youth with BD (Esposito-smythers et al., 2006; Weintraub et al., 2019). Externalizing behaviors, such as aggression, are also correlated with poorer family functioning in youth with BD (Keenan-Miller et al., 2012). Therefore, externalizing symptoms and disorders negatively impact family functioning in youth with BD, although this may be caused by an exacerbation of manic and depressive symptoms.

Family functioning also predicts the course of externalizing behavior symptoms in youth with behavioral and emotional disorders. Poor parent-child interactions are associated with greater symptom severity and predict later development of oppositional defiant disorder symptoms in ADHD youth (Breaux & Harvey, 2019; Harvey et al., 2011), and family functioning predicts later problem behavior in children at risk for emotional and behavioral disorders (Nelson et al., 2007). However, it is still unclear how family functioning influences externalizing symptoms in youth with BD specifically. Overall, similar to depression and manic symptoms, externalizing behavior symptoms may act as both a cause and a result of disruptions in family functioning.

## CHAPTER 2

### PURPOSE OF THE PRESENT STUDY

Youth with psychopathology display poorer family functioning compared to psychiatrically healthy youth (Freed et al., 2015; Lau et al., 2018). In particular, youth with BD typically have lower cohesion, adaptability, and warmth and poorer problem solving compared to families of healthy youth (Belardinelli et al., 2008; Sullivan & Miklowitz, 2010). Additionally, parents of youth with BD report greater parenting stress and display more negative expressed emotion than parents of healthy youth (Algorta et al., 2018; Nader et al., 2013). However, little work has benchmarked deficits in family functioning across youth psychopathology. Comparing family functioning across different mental health disorders may provide insights into how different disorders differentially affect families. Therefore, aim one of this study is to examine how family functioning in youth with BD compares to youth with other psychiatric disorders.

Deficits in family functioning are commonly considered an outcome of symptoms (Keenan-Miller et al., 2012; Weintraub et al., 2019). Depressive and manic symptoms both are associated with declines in cohesion and adaptability and an increase in family conflict (Rosa et al., 2010; Sullivan et al., 2012). However, depressive symptoms may have a greater impact on family functioning than manic symptoms (Calabrese et al., 2004). Additionally, youth with BD typically have multiple comorbid disorders or presenting problems that could also impact family functioning (Frías et al., 2015). For example, comorbid externalizing disorders predict even greater declines in functioning than depression alone (Asarnow et al., 1994). The current study aims to broaden the current literature by evaluating the association between family functioning

and specific mood symptoms (i.e., depressive, manic, or mixed) as well as alternative explanations (i.e., general externalizing behavior) among youth with BD.

### **Aims and Hypotheses**

*Aim 1.* Benchmark family functioning in youth who have been diagnosed with BD against family functioning in youth who have been diagnosed with other psychiatric diagnoses.

*Hypothesis 1.* Caregivers of youth who have been diagnosed with BD will report similar family functioning compared to caregivers of youth who have been diagnosed with a unipolar depressive disorder but will report worse family functioning compared to caregivers of youth with behavior disorders or other non-behavior and non-mood disorders (e.g., anxiety disorders).

*Hypothesis 2.* Clinicians will report similar family functioning among families of youth with BD compared to families of youth with unipolar depression, but will report worse family functioning compared to families of youth with behavior disorders or other non-behavior and non-mood disorders (e.g. anxiety disorders).

*Aim 2.* Examine the association between family functioning and depressive, (hypo)manic, mixed, and externalizing behavior symptoms.

*Hypothesis 3.* As caregiver-reported severity of depressive, (hypo)manic, mixed, and externalizing behavior symptoms increases, impairments in family functioning will also increase.

*Hypothesis 4.* As clinician-rated severity of depressive, (hypo)manic, mixed, and externalizing behavior symptoms increases, impairments in family functioning will also increase.

*Aim 3.* Explore which symptom class (depressive, (hypo)manic, mixed, and externalizing behavior symptoms) is most strongly associated with family functioning.

*Hypothesis 5.* For parent-reported symptom severity, externalizing behavior symptoms will exhibit the strongest association with family functioning followed by depressive symptoms, mixed symptoms, and lastly (hypo)manic symptoms.

*Hypothesis 6.* For clinician-rated symptom severity, externalizing behavior symptoms will exhibit the strongest association with family functioning followed by depressive symptoms, mixed symptoms, and lastly (hypo)manic symptoms.

## CHAPTER 3

### METHODS

#### **Participants**

Participants for the current study consisted of approximately 828 caregiver-youth dyads recruited from either an urban community mental health center (CMHC;  $n = 626$ ) or an academic medical center (AMC;  $n = 202$ ) in Cleveland, OH. At both sites, inclusion criteria required both youth between the ages of 5 to 18 years, and their caregiver to: (a) be present for assessment, (b) be conversant in English, and (c) provide written consent and/or assent. Exclusion criteria were: (a) the presence of a developmental disorder or (b) evidence of intellectual disability.

Participants recruited at the CMHC were a consecutive case series of all new intakes for services regardless of presenting reason. Approximately 65% of the families who were approached agreed to participate and were scheduled. If participants exceeded assessment capacity, then families were selected using simple random sampling.

Participants recruited at the AMC were drawn from families seeking assessment and treatment in one of many clinical trials. Participants were recruited based on presenting symptoms and willingness to participate in treatment protocols. Target diagnoses for study recruitment included bipolar spectrum disorders (bipolar I, bipolar II, cyclothymia or bipolar not otherwise specified [NOS]), unipolar depression, ADHD, conduct disorder, and PTSD. Additionally, children were eligible to participate in the assessment phase if parents reported aggressive behavior during a phone call with the AMC, regardless of diagnosis. Interested families completed a diagnostic assessment as a screening or baseline evaluation.

## Measures

*Demographics.* Data included the youth's age, gender, race/ethnicity, number of comorbid psychiatric diagnoses (current and lifetime), and assessment site.

*The Schedule for Affective Disorders and Schizophrenia for School-aged Children (KSADS).* The KSADS Present and Lifetime version (Kaufman et al., 1997) was used to determine youth diagnoses. The KSADS-PL is a semi-structured interview that queries both parent and child about current and lifetime symptoms from common psychiatric disorders. The mood modules from the Washington University KSADS (WASH-U-KSADS; Geller et al., 2001) were integrated with the KSADS-PL to further assess associated features of depression and mania.

The KSADS depression rating scale (KDRS; Kaufman et al., 1997) and the KSADS mania rating scale (KMRS; Axelson et al., 2003) were integrated into the diagnostic interview and were used to assess clinician-rated depression and mania severity. The KDRS is based off the depression symptom intensity ratings and has demonstrated good validity (Kaufman et al., 1997). The KMRS consists of 14 questions from the KSADS plus an additional item assessing mood lability. The KMRS has good validity and internal consistency ( $\alpha = 0.94$ ; Axelson et al., 2003).

Clinician-rated externalizing symptoms were assessed using the KSADS-PL. An externalizing scale, consisting of the oppositional defiant disorder (ODD) and conduct disorder (CD) screening items, was constructed for the current study. Every participant was assessed using the KSADS-PL screening items. Only the screening items were used to create the scale because most participants were missing the supplemental items.

The KSADS-PL was completed individually by both caregivers and youth. Research assistants conducted assessments at both sites. Research assistants were primarily predoctoral psychology interns, research staff with a Master's degree or PhD in psychology, or research staff with a Master's degree in social work, and were highly trained. New raters had to pass at least five interviews with a reliable rater leading and then lead at least five interviews with a reliable rater observing. A new rater passed an interview if they achieved a symptom-level reliability of  $\kappa = .85$  and diagnosis agreement of  $\kappa = 1.0$ . New raters were trained annually ( $n = 6$  per cohort). During the length of the study only 1 rater failed standardization. All diagnoses follow criteria from the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (DSM-IV-TR; American Psychiatric Association, 1994).

*Parent General Behavior Inventory (P-GBI)*. Mood symptom severity was assessed through parent-report using the Parent General Behavior Inventory (P-GBI; Youngstrom et al., 2001). The P-GBI is adapted from the GBI (Depue et al., 1981), modifying the original 73 items to refer to the informant's child rather than to the self (e.g., "Have there been periods of time when you felt a persistent sense of gloom" was changed to "Have there been periods of time when your child felt a persistent sense of gloom"). The P-GBI items are rated on a 4-point Likert-type scale ranging from *never or hardly ever present* to *very often or almost constantly present*. The P-GBI consists of two scales, the Depression scale consisting of 46 items that assess symptoms associated with depression and the Hypomanic-Biphasic (H/B) scale consisting of 28 items that measure symptoms associated with mania in both classical and mixed forms. The H/B scale contains 21 items assessing (hypo)manic symptoms (e.g., "Have there been times when your child began many new activities with lots of enthusiasm and then found himself/herself quickly losing interest in them?") and 7 items assessing biphasic, or mixed, symptoms (e.g.,

“Has your child’s mood or energy shifted rapidly back and forth from happy to sad or high to low?”). The P-GBI has demonstrated good reliability ( $\alpha = 0.95-0.97$ ), validity, and diagnostic utility for youth ages 5 to 17 (Youngstrom et al., 2001).

*The Child Behavior Checklist (CBCL).* Parental ratings of externalizing behavior problems were established using the CBCL (Achenbach & Rescorla, 2001). The CBCL is widely used in both research and clinical work to assess behavioral problems in youth ages 6 to 18-years-old. The CBCL consists of 118 items rated on a 3-point Likert scale ranging from *Not True* to *Very True or Often True*. For youth who were 5-years-old, caregivers completed the CBCL 1.5-5.5 Years. The present study used scores from the Externalizing Problems scale, which is based on DSM-IV-TR criteria and is comprised of the Rule-breaking Behavior and Aggressive Behavior syndrome scales. The Externalizing problem scale will provide a well-established measure of disruptive and aggressive symptoms for the current study.

*The Family Assessment Device (FAD).* The FAD global scale score (Epstein et al., 1983; Kabacoff et al., 1990; I. W. Miller et al., 1985) was used to assess family functioning. The caregiver completed the 27-item FAD global scale about the family. The FAD global scale assesses communication (e.g., “When someone is upset the others know why”), problem solving (e.g., “We are able to make decisions about how to solve problems”), and general functioning (e.g., “In times of crisis we turn to each other for support”). Items are rated on a 4-point Likert scale ranging from *Strongly Agree* to *Strongly Disagree*. Total score is calculated by adding item responses together (some items are reverse-coded and must be recoded before adding together the raw scores of each domain). Higher scores indicate worse family functioning. The communication, problem solving, and general functioning scales of the FAD have demonstrated

good internal consistency ( $\alpha = 0.70-0.86$ ), test-retest reliability ( $r = 0.66-0.72$ ), and validity (Kabacoff et al., 1990; I. W. Miller et al., 1985).

*The Revised Children Quality-of-Life Questionnaire (KINDL-R).* The KINDL-R was used to assess quality of life (QoL; Ravens-Sieberer & Bullinger, 1998). The caregiver completed the 24-item KINDL-R about their child. The KINDL-R consists of the following 7 domains: Total, Physical, Emotional, Self-Esteem, Family, Friends, and School. For this study, only the Family subscale was used. An example of a Family subscale item is “During the past week we quarreled at home.” Each item is scored on a 5-point Likert scale (0 = *never*, 4 = *all the time*) and transformed to a POMP score such that higher scores indicate better QoL. The current study will use 2 different versions of the KINDL based on the age of the child; the younger version was used for youth ages 5- to 7-years-old, and the older version was used for youth 8- to 18-years-old. The KINDL Family subscale has demonstrated acceptable internal consistency ( $\alpha = .74$ ), test-retest reliability (ICC = 0.83), and validity (Erhart et al., 2009; Villalonga-Olives et al., 2015).

*Perceived Criticism Measure (PCM).* The PCM (Hooley & Teasdale, 1989) assessed negative expressed emotion. The PCM consists of two items assessing perceived criticism (PC) and criticism sensitivity (CS) (“*How critical is your family member of you?*” and “*When your family member criticizes you, how upset do you get?*”). These items are rated on a 10-point Likert scale ranging from 1= *not at all* to 10= *very much indeed*. Scores 6 or above can be categorized as high PC and CS, and scores below 6 can be categorized as low PC and CS (Hooley & Teasdale, 1989).

*The Global Family Environment Scale (GFES)*. The GFES (Rey et al., 1997) was used to assess overall family functioning as rated by the clinician. The GFES consists of a single score, ranging from 1 to 90, with higher scores indicating families with a better ability to provide physical and emotional care in a consistent and appropriate manner. Ratings should reflect the lowest quality of family environment that persisted for at least one year. The GFES has shown good test-retest ( $r = 0.91$ ) and interrater reliability ( $r = .86-.88$ ; Rey et al., 1997).

## **Procedures**

The institutional review boards of University Hospitals of Cleveland, Case Western Reserve University, Applewood Centers, and the University of North Carolina at Chapel Hill approved all procedures. Caregivers provided written consent and youth provided written/verbal assent.

The research assistant interviewed the caregiver and youth sequentially and independently with the KSADS. During the KSADS interview, the research assistant rated the KDRS, KMRS, and GFES based on the informant. After the interview was completed, the research assistant provided a final summary score integrating information received from both informants. With a separate research assistant, the caregiver completed the P-GBI, CBCL, FAD, KINDL-R, and PCM while the youth was interviewed. The separate research assistant answered any caregiver questions about the questionnaires and/or read the questionnaires to the caregiver if needed.

Diagnoses were finalized by a licensed clinical psychologist using the LEAD standard (Spitzer, 1983). The licensed clinical psychologist met with the research assistant and integrated information from the KSADS symptoms and diagnoses, family history, treatment history, intake

diagnoses, and previous psychological/psychiatric evaluation findings. Both the clinical psychologist and the research assistant were masked to the caregiver and youth completed questionnaires (CBCL, P-GBI, FAD, and PCM). Data were collected prior to the release of DSM-5. Therefore, diagnoses were made with strict accordance to DSM-IV-TR criteria.

## CHAPTER 4

### ANALYSES

#### **Data Screening**

All analyses were conducted in R v4.0 (R Core Team, 2019). The *psych* (Revelle, 2019) and *Hmisc* (Harrell et al., 2019) packages were used in the data screening process. All variables of interest were screened for erroneous data entries (e.g., implausible values), missing data, and univariate outliers. For scale scores (e.g., FAD global scale, KINDL Family, KDRS, & KMRS), if 90% of an individual case's items had responses, then mean substitution was used to create the scale scores. If a case's items had less than 90% of items completed, then the scale was treated as missing. Normality of continuous variables was assessed via Q-Q plots, skewness, kurtosis, and Shapiro-Wilks' statistic. In line with general data screening recommendations (Tabachnick & Fidell, 2007), categorical data that had more than a 90-10 split between categories and individuals with  $z$ -scores  $\pm 3.29$  were flagged as possible univariate outliers. Initial analyses included all cases and if multivariate outliers were present, then the univariate outliers were used to help identify cases that are outliers and potential solutions. Multivariate outliers that could be resolved from appropriate univariate scale transformations (e.g., square root, log), were addressed as part of follow-up analyses. Missing data for the cases that were retained were imputed using the *mice* package (Buuren & Groothuis-Oudshoorn, 2011) using predictive mean matching to impute continuous variables and logistic regression to impute dichotomous variables. A total of 20 imputed datasets were generated, each using 20 iterations to achieve convergence on the imputed values.

The impact of a diagnosis of BD was assessed in two manners. A binary variable representing the presence (1) of a bipolar spectrum disorder diagnosis (i.e., bipolar I, bipolar II, cyclothymia, & bipolar NOS) or absence (0) of a bipolar spectrum disorder diagnosis was created. A hierarchical categorical variable was also created. First, youth who met DSM-IV-TR criteria for bipolar I, bipolar II, cyclothymia, or bipolar not otherwise specified (NOS) were assigned to a BD group. Second, youth with a unipolar depressive disorder (i.e., youth who meet DSM-IV-TR criteria major depression, dysthymia, depression NOS, or adjustment disorder with depressed mood) were assigned to a unipolar depression category regardless of comorbid diagnoses. Third, youth with a behavior disorder (i.e., youth who meet DSM-IV-TR criteria for ADHD [combined, inattentive, hyperactive, NOS], oppositional defiant disorder, conduct disorder, or disruptive behavior disorder NOS) were assigned to a behavior disorder group. Fourth, youth who did not have a BD, unipolar depression, or a behavior disorder (e.g., youth with anxiety or PTSD without comorbid mood or behavior disorders) were assigned to a final group.

### **Aim 1**

Aim 1 benchmarked family functioning in youth with BD against family functioning in youth with other psychiatric diagnoses. Specifically, we compared youth with BD to youth with unipolar depression, youth with behavior disorders, and youth with other non-mood, non-behavior disorders (e.g., anxiety disorders). A multiple indicator multiple cause (MIMIC) model was fit to the data using the *lavaan* package (Rosseel, 2012) and a linear regression was fit. The MIMIC model consists of two parts: (a) a measurement model and (b) a regression model regressing the latent variable formed in the measurement model on manifest predictors and any covariates of interest (Schumacker & Lomax, 2016).

First, the measurement model was used to create a latent family functioning variable. We tested three different models using the FAD total and subscale variables, KINDL Family subscale variable, PCM total and subscale variables, and GFES variable. The first model created a latent parent-reported family functioning variable as indicated by the total scores from the FAD and PCM, and scores from the KINDL Family subscale. The second model also used parent-reported variables, but varied from the first model in that it used all subscale scores (i.e., FAD Communication, FAD Problem Solving, FAD General Functioning, PCM Self, PCM Other, and KINDL Family subscales). The third model created a latent family functioning variable using both parent- and clinician-reports. This model included the FAD Total, PCM Total, KINDL Family, and GFES scores.

Fit of the measurement model was assessed by both absolute fit indices and relative fit indices. The following absolute fit indices were used: (a) chi-squared test, (b) root mean square error of approximation (RMSEA), and (c) the standardized root mean square residual (SRMR). The chi-squared test indicates the difference between the observed and model expected covariance matrices with values closer to 0 indicating better fit. Chi-squared as an indicator of fit is sensitive to sample size with sample sizes greater than 200, often resulting in chi-squared indicating poor model fit (Kline, 2015). The RMSEA tests indicates the difference between the hypothesized model and the population covariance matrix (Hooper et al., 2008). RMSEA values range between 0 and 1 with values closer to 0 indicating better fit. RMSEA less than or equal to .06 indicate good model fit (Hu & Bentler, 1999). The SRMR indicates the discrepancy between the sample covariance matrix and the model covariance matrix. The SRMR ranges between 0 and 1 with values closer to 0 indicating better fit. SRMR less than or equal to .08 indicate good model fit (Hu & Bentler, 1999). In contrast to absolute fit indices, relative fit indices compare the

current model to a baseline null model (Brown, 2015). The comparative fit index (CFI) tests the discrepancy between the data and hypothesized model while adjusting for sample size (Bentler, 1990). CFI values range between 0 and 1 with values closer to 1 indicating better fit. CFI values greater than or equal to .95 indicate good model fit (Hu & Bentler, 1999).

Once the measurement model was determined, the parameter estimates were fixed at their estimated values in the MIMIC model to improve overall model fit. Separate MIMIC models were used to regress the latent family functioning variable on the reference coded BD diagnosis variables. Sensitivity analyses adjusted for the youth's age, gender, and number of comorbidities.

A linear regression model regressed the clinician-rated family functioning variable (i.e., GFES) on the reference coded BD diagnosis variables. Sensitivity analyses adjusted for the youth's age, gender, and number of comorbidities. Assumptions of regression (e.g., linear fit, homoscedasticity, & normality of residuals) were assessed via plotting residuals. Regression diagnostics examined multicollinearity and influential cases (i.e., multivariate outliers). No concerns of multicollinearity and influential cases were detected.

Sensitivity power analyses were conducted using GPower v 3.1.9.7 (Faul et al., 2007, 2009). Sensitivity power analyses are appropriate when secondary data analysis is being conducted to ensure that the sample is able to appropriately detect effect sizes of interest (Bierman & Bubolz, 2003; Dziak et al., 2020). Assuming a sample size of 828, four comparison groups, 80% power, and alpha of .05, a one-way ANOVA was powered to detect small effects,  $f = .12$ ,  $\eta^2 = .01$ . Assuming a sample size of 828, four comparison groups, 80% power, an alpha of .05, and two covariates, a one-way ANCOVA was powered to detect small effects,  $f = .13$ ,  $\eta^2 = .02$ .

## **Aim 2**

Aim 2 examined whether depressive, (hypo)manic, mixed, or externalizing symptoms are associated with family functioning. Once the measurement model was determined, the parameter estimates were fixed at their estimated values in the MIMIC model to improve overall model fit. Separate MIMIC models regressed the latent family functioning variable on depressive, (hypo)manic, mixed, and externalizing symptoms. Sensitivity analyses adjusted for the youth's age, gender, number of comorbidities, and BD status.

A series of linear regression models regressed the clinician-rated family functioning variable (i.e., GFES) on depressive, (hypo)manic, mixed and externalizing symptoms. Sensitivity analyses adjusted for the youth's age, gender, number of comorbidities, and BD status.

Sensitivity power analyses were conducted using GPower v 3.1.9.7 (Faul et al., 2007, 2009). Assuming 80% power, a sample size of 828,  $\alpha = .05$ , and 7 predictors (the four groups are reference coded with BP as the reference group) of which 1 is of interest (i.e., the specific symptom variable), the study was powered to detect small effects,  $f = .01$ ,  $\eta^2 = .01$ . Assuming 80% power, a sample size of 828,  $\alpha = .05$ , and 5 predictors (reference coded BP versus other diagnoses) of which 1 is of interest (i.e., the specific symptom variable), the study was powered to detect small effects,  $f = .01$ ,  $\eta^2 = .01$ .

## **Aim 3**

Aim 3 determined which type of symptoms are most strongly associated with impairments in family functioning. Once the measurement model was determined, the parameter estimates were fixed at their estimated values in the MIMIC model to improve overall model fit. A single MIMIC model regressed the latent family functioning variable on depressive,

(hypo)manic, mixed and externalizing symptoms. Sensitivity analyses adjusted for the youth's age, gender, number of comorbidities, and BD status.

A single linear regression model regressed the clinician-rated family functioning variable (i.e., GFES) on depressive, (hypo)manic, mixed and externalizing symptoms. Sensitivity analyses adjusted for the youth's age, gender, number of comorbidities, and BD status.

Sensitivity power analyses were conducted using GPower v 3.1.9.7 (Faul et al., 2007, 2009). Assuming 80% power, a sample size of 828, alpha = .05, and 10 predictors (the four groups are reference coded with BP as the reference group) of which 1 is of interest (i.e., the specific symptom variable), the study was powered to detect small effects,  $f = .01$ ,  $\eta^2 = .01$ . Assuming 80% power, a sample size of 828, alpha = .05, and 8 predictors (reference coded BP versus other diagnoses) of which 1 is of interest (i.e., the specific symptom variable), the study was powered to detect small effects,  $f = .01$ ,  $\eta^2 = .01$ .

## CHAPTER 5

### RESULTS

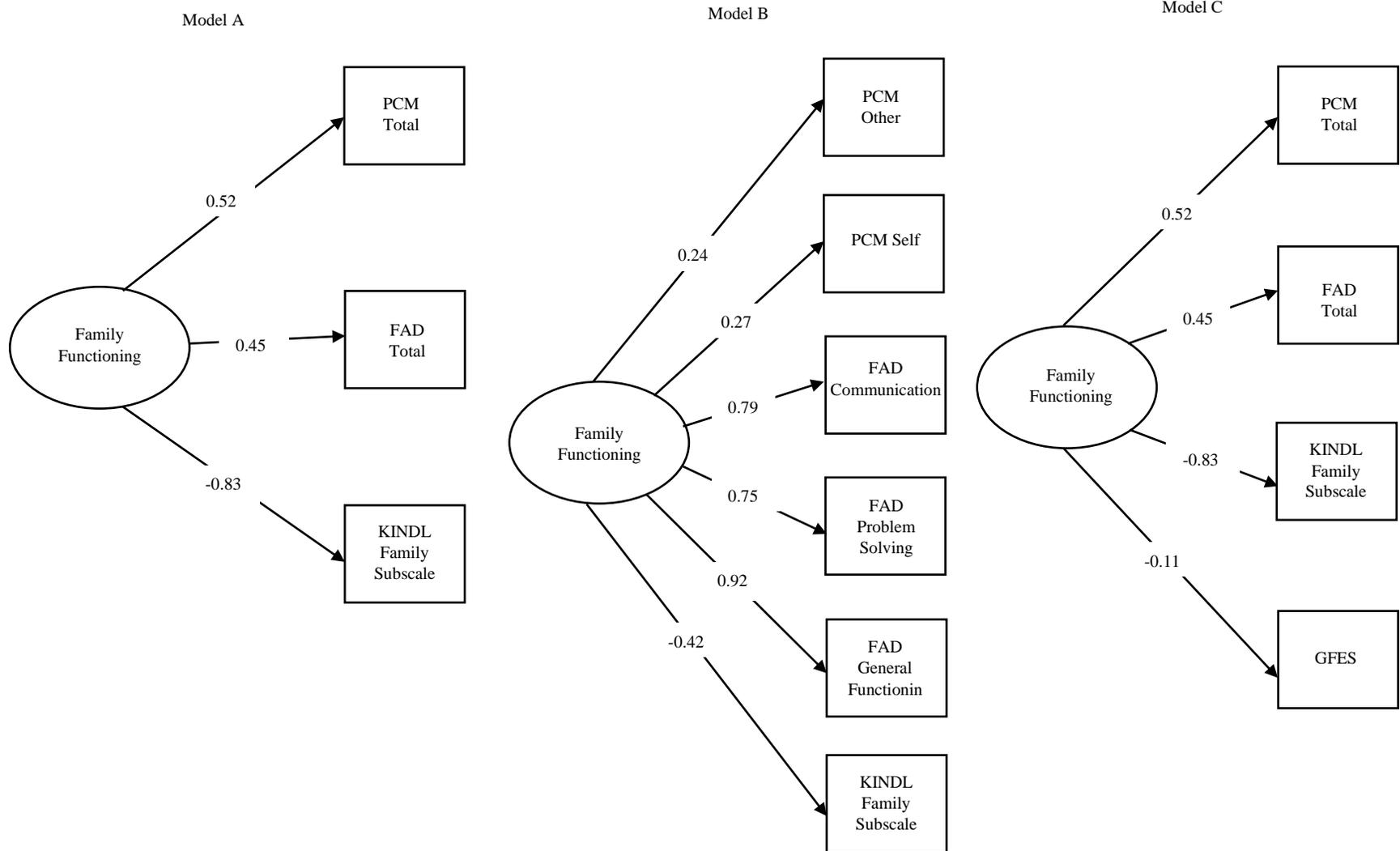
#### **Model of Caregiver-Reported Family Functioning**

Fit indices for three models of family functioning were assessed: a model utilizing parent-reported total scores (i.e., total parent model), a model utilizing parent-reported subscale scores (i.e., subscale parent model), and a model utilizing both parent- and clinician-rated total scores (i.e., combined model). The total parent model had the best overall fit, CFI = 1.00, RMSEA = .00, SRMR = .00. The total parent model consisted of a single latent family functioning variable indicated by the following parent-reported total scores: FAD Total ( $\lambda = .45$ ), PCM Total ( $\lambda = .52$ ), and KINDL Family ( $\lambda = -.83$ ) scales. Higher scores on this latent variable indicated greater impairment in parent-reported family functioning.

A combined model consisting of parent total scores and clinician total score was also tested and demonstrated excellent overall fit (CFI = 1.00, RMSEA = .00, SRMR = .01). However, the clinician-reported GFES barely loaded on the latent variable ( $\lambda = -.11$ , 95% CI [- .19, -.03]). Thus, clinician-rated and parent-reported family functioning were analyzed separately. Finally, a CFA examined the subscale parent model that consisted of three FAD subscales, two PCM subscales, and the KINDL Family scale. This model demonstrated poor fit, CFI = .92, RMSEA = .15, SRMR = .08. Although the FAD subscales and KINDL family scale demonstrated good factor loadings ( $\lambda$ s = .42 - .92), the PCM subscales demonstrated poor factor loadings ( $\lambda$ s = .24 - .27). Therefore, the CFAs supported separating family functioning by informant for substantive analyses using total scores. CFA results for each model are presented in Figure 1.

Figure 1

Confirmatory Factor Analysis Results for Models of Family Functioning



Note. CFA results for a latent family functioning variable. Model A uses parent-reported total scores from the Percieved Criticism Measure (PCM), the Family Assessment Device (FAD), and the family subscale of the Revised Children Quality-of-Life Questionnaire (KINDL-R). Model B uses parent-reported subscale scores from the PCM, the FAD, and the KINDL-R. Model C uses parent-reported total scores from the PCM, FAD, and KINDL-R family subscale, and clinician-reported total scores from the Global Family Environment Scale (GFES).

## Family Functioning in BD Versus Other Psychiatric Conditions

Tables 1 and 2 display the means and standard deviations for children with BD and other psychiatric conditions and the correlations among the family functioning scales and demographics. Family functioning weakly to moderately was negatively related to youth's age. Increases in the number of comorbid diagnoses was weakly to moderately associated with worse family functioning, with the exception of FAD total scores which were unassociated with comorbidity. Being female was associated with more expressed emotion (Cohen's  $d = .15$ ) and lower caregiver-rated family QoL (Cohen's  $d = -.21$ ) relative to male youth. Identifying as a minority was weakly associated with worse scores on clinician-rated family functioning (Cohen's  $d = .23$ ), but better caregiver-reported family functioning, Cohen's  $ds = .18 - .40$ . Participants at the community mental health clinic had worse caregiver-reported family functioning (Cohen's  $ds = .30 - .45$ ) and better clinician rated family functioning (Cohen's  $d = 0.26$ ) than the academic medical center.

Table 3 and Table 4 display the means and standard deviations for children from the two different recruitment sites. Chi-squares and linear regression were used to test for site differences. Participants from the AMC were older ( $b = 1.34$ , 95% CI [.77, 1.91],  $R^2 = .03$ ,  $p < .001$ ) and more likely to be White ( $\chi^2(1) = 373.4$ ,  $p < .001$ ). The presenting psychopathology rates also differed between the sites ( $\chi^2(1) = 8.74$ ,  $p < .01$ ), with AMC participants being more likely to have BD ( $z = 4.86$ ,  $p < .001$ ) and less likely to have an externalizing behavior disorder ( $z = -2.66$ ,  $p < .01$ ). Participants from the AMC also had poorer family functioning across all caregiver-rated and clinician-rated measures of family functioning,  $bs = 1.26 - 3.13$ ,  $ps < .01$ .

Due to the differences in samples across these demographic and psychodiagnostic features, a series of MIMIC models and linear regression models tested whether youth diagnosis

was associated with family functioning after adjusting for study site, gender, ethnicity, age, and number of comorbid diagnoses.

Table 1

*Family Functioning by Psychiatric Condition*

<b>Primary Diagnosis</b>	<b>Total n = 828</b>	<b>Bipolar n = 153</b>	<b>Depression n = 239</b>	<b>Behavior n = 362</b>	<b>Other n = 74</b>
Age					
Mean	10.9	11.1	12.2	9.95	11.0
SD	3.42	3.67	3.26	3.15	3.30
No. of					
Mean	2.65	3.42	3.06	2.35	1.16
SD	1.37	1.52	1.36	1.00	1.01
Gender, n (%)					
Boy	496 (59.9)	83 (54.3)	121 (50.7)	258 (71.1)	34 (46.5)
Girl	332 (40.1)	70 (45.7)	118 (49.3)	105 (28.9)	40 (53.5)
Ethnicity, n (%)					
White	185 (22.3)	56 (36.7)	52 (21.8)	61 (16.9)	16 (21.3)
Non-White	643 (77.7)	97 (63.3)	187 (78.2)	301 (83.1)	58 (78.7)
PCM Total					
Mean	21.9	23.4	22.6	21.3	19.3
SD	8.47	7.83	8.23	8.53	9.46
FAD Total					
Mean	2.01	2.08	2.08	1.96	1.87
SD	0.43	0.46	0.43	0.42	0.41
KINDL Family					
Mean	9.09	7.60	8.54	9.71	10.8
SD	3.35	2.80	3.35	3.31	3.05
GFES					
Mean	68.0	65.8	65.9	69.5	71.7
SD	11.7	13.0	11.2	10.8	12.5

*Note.* GFES = Global Family Environment Scale score, PCM = Perceived Criticism Measure total score, FAD = Family Assessment Device total score, and KINDL Family = The Revised Children Quality-of-Life Questionnaire (KINDL-R) family subscale score.

Table 2

*Correlations between Demographics and Family Functioning*

Variable	GFES	PCM	FAD	KINDL Family	Age	Gender	Race	Comorbid Diagnoses
GFES	1							
PCM	-.03 [-.10, .04]	1						
FAD	-.17* [-.24, -.10]	.24* [.17, .30]	1					
KINDL Family	.09* [.03, .16]	-.43* [-.49, -.37]	-.37* [-.43, -.31]	1				
Age	-.11* [-.18, -.04]	.20* [.14, .27]	.22* [.16, .29]	-.20* [-.27, -.13]	1			
Gender	.01 [-.06, .08]	.08* [.01, .15]	.07 [.00, 0.13]	-.10* [-.17, -.03]	.20* [.13, .26]	1		
Race	-.10* [-.17, -.03]	-.10* [-.17, -.03]	-.07* [-.14, .00]	.17* [.10, .23]	-.11* [-.17, -.04]	.03 [-.04, .10]	1	
Comorbid Diagnoses	-.15* [-.22, -.09]	.15* [.08, .21]	.07 [.00, .13]	-.18* [-.24, -.11]	.00 [-.06, .07]	.00 [-.07, .07]	-.04 [-.11, .03]	1
Site	.10* [.03, .17]	.15* [.08, .22]	.12* [.05, .19]	-.18* [-.25, -.12]	.16* [.09, .23]	.01 [-.06, .07]	-.67* [-.71, -.64]	-.02 [-.09, .05]

*Note.* Values in square brackets indicate the 95% confidence interval for each correlation. GFES = Global Family Environment Scale score, PCM = Perceived Criticism Measure total score, FAD = Family Assessment Device total score, and KINDL Family = The Revised Children Quality-of-Life Questionnaire (KINDL-R) family subscale score.

\* indicates  $p < .05$ .

Table 3

*Demographics and Family Functioning by Recruitment Site*

<b>Recruitment Site</b>	<b>CMHC N = 651</b>	<b>AMC N = 177</b>
Age*		
Mean	10.6	12.0
SD	3.41	3.28
No. of diagnoses		
Mean	2.66	2.60
SD	1.38	1.35
Gender, n (%)		
Boy	391 (60.1)	105 (59.3)
Girl	260 (39.9)	72 (40.7)
Ethnicity, n (%)		
White*	50 (7.7)	135 (76.3)
Non-White*	601 (92.3)	42 (23.7)
Diagnosis, n (%)		
Bipolar*	92 (14.1)	61 (34.5)
Depression	190 (29.2)	49 (27.6)
Behavior*	308 (47.3)	54 (30.8)
Other	61 (9.4)	13 (7.1)
PCM Total*		
Mean	21.2	24.4
SD	8.75	6.85
FAD Total*		
Mean	1.98	2.11
SD	0.43	0.44
KINDL Family*		
Mean	9.41	7.90
SD	3.41	2.82
GFES*		
Mean	67.4	70.3
SD	11.8	10.8

*Note.* CMHC = community mental health clinic, AMC = academic medical center, GFES = Global Family Environment Scale score, PCM = Perceived Criticism Measure total score, FAD = Family Assessment Device total score, and KINDL Family = The Revised Children Quality-of-Life Questionnaire (KINDL-R) family subscale score.

\* indicates site differences of  $p < .05$ .

Table 4

*Symptom Ratings by Recruitment Site*

<b>Recruitment Site</b>	<b>CMHC N = 651</b>	<b>AMC N = 177</b>
<b>Parent-Rated</b>		
Depression*		
Mean	25.7	34.5
SD	22.1	23.9
Hypomanic-Biphasic*		
Mean	20.5	23.2
SD	14.5	15.6
Hypomanic		
Mean	14.5	15.2
SD	10.8	11.5
Mixed*		
Mean	5.90	7.90
SD	4.41	4.90
Externalizing*		
Mean	70.3	67.1
SD	9.83	8.97
<b>Clinician-Rated</b>		
Depression*		
Mean	20.8	24.7
SD	8.72	9.35
Hypomanic-Biphasic*		
Mean	19.1	24.6
SD	9.02	10.3
Externalizing		
Mean	6.13	6.18
SD	3.74	3.68

*Note.* CMHC = community mental health clinic and AMC = academic medical center.

\* indicates site differences of  $p < .05$ .

### *Caregiver-reported family functioning*

Two sets of MIMIC models tested whether caregiver-reported family functioning in youth with BD differed from caregiver-reported family functioning of youth with other psychiatric disorders. First, youth with BD were compared to all other youth, regardless of diagnosis. Youth with BD had significantly more impaired family functioning compared to all other youth,  $\beta = 0.13$ , 95% CI [.06, .19],  $R^2 = .13$ ,  $p < .001$ . This effect persisted after adjusting for study site, gender, ethnicity, age, and number of comorbid diagnoses,  $\beta = 0.13$ , 95% CI [.06, .19],  $\Delta R^2 = .08$ ,  $p < .001$ . Second, youth with BD were compared to youth with unipolar depression, behavior disorders, or non-mood/behavior disorders. Youth with BD had significantly lower family functioning compared to youth with depression ( $\beta = -.12$ , 95% CI [-.22, -.02],  $R^2 = .10$ ,  $p = .01$ ), youth with behavior disorders ( $\beta = -0.34$ , 95% CI [-.43, -.24],  $R^2 = .10$ ,  $p < .001$ ), and youth with other non-mood and non-behavior disorders ( $\beta = -.31$ , 95% CI [-.39, -.22],  $R^2 = .10$ ,  $p < .001$ ). These differences persisted after adjusting for study site, gender, ethnicity, age, and number of comorbid diagnoses for depression ( $\beta = -.10$ , 95% CI [-.20, .00],  $\Delta R^2 = .07$ ,  $p = .04$ ), behavior disorders ( $\beta = -.19$ , 95% CI [-.29, -.09],  $\Delta R^2 = .07$ ,  $p < .001$ ), and other non-mood and non-behavior disorders ( $\beta = -.22$ , 95% CI [-.31, -.13],  $\Delta R^2 = .07$ ,  $p < .001$ ). In summary, parent of youth with BD reported greater impairments in family functioning compared to parents of youth with other psychiatric disorders.

### *Clinician-rated family functioning*

Next, a series of linear regressions tested whether clinician-rated family functioning in youth with BD differed from youth with other psychiatric diagnoses. First, youth with BD were compared to all other youth, regardless of diagnosis. Youth with BD had more impaired family

functioning compared to all other youth,  $b = -2.64$ , 95% CI [-4.72, -.56],  $R^2 = .01$ ,  $p = .01$ . After adjusting for study site, gender, ethnicity, age, and number of comorbid diagnoses, a BD diagnosis remained associated with more impaired family functioning compared to youth with other psychiatric diagnoses,  $b = -2.43$ , 95% CI [-4.58, -.26],  $\Delta R^2 = .05$ ,  $p = .03$ . Second, youth with BD were compared to youth with unipolar depression, behavior disorders, or non-mood/behavior disorders. The diagnostic groups differed in their clinician-rated family functioning,  $F(1, 828) = 22.35$ ,  $\eta^2 = .03$ ,  $p < .001$ . *Post hoc* comparisons indicated that youth with BD had significantly more impairment in clinician-reported family functioning than youth with non-mood and non-behavior disorders (Cohen's  $d = -.47$ ,  $p < .01$ ) and youth with behavior disorders, Cohen's  $d = -.31$ ,  $p < .01$ . However, youth with BD did not differ significantly in clinician-rated family functioning from youth with depression, Cohen's  $d = -.01$ ,  $p = .94$ . In summary, youth with BD had greater impairments in clinician-rated family functioning compared to youth with behavior disorders and youth with non-mood and non-behavior disorders; however, youth with BD had similar family functioning compared to youth with depression.

### **Mood and Externalizing Symptoms Impact on Family Functioning**

Tables 5 and 6 display the means and standard deviations for children with BD and other psychiatric conditions and the correlations among the family functioning scales and mood symptoms. Increases in externalizing, depressive, and caregiver-reported (hypo)manic-biphasic symptoms were weakly to strongly associated with greater impairment in clinician-rated and caregiver-reported family functioning. Increases in clinician-reported (hypo)manic-biphasic symptoms were also weakly to moderately associated with greater impairment in clinician-rated family functioning and caregiver-reported family functioning, with exception of the FAD total

scores, which were not associated with (hypo)manic-biphasic symptoms. Therefore, a series of MIMIC models and linear regression models tested whether youth symptom severity was associated with family functioning after adjusting for other symptom classes.

Table 5

*Symptom Severity by Psychiatric Condition*

<b>Primary Diagnosis</b>	<b>Total n = 828</b>	<b>Bipolar n = 153</b>	<b>Depression n = 239</b>	<b>Behavior n = 363</b>	<b>Other n = 73</b>
<b>Parent-Rated</b>					
Depression					
Mean	27.5	42.1	37.5	17.1	16.3
SD	22.7	23.8	22.8	15.9	17.4
Hypomanic- Biphasic					
Mean	21.0	32.3	21.9	17.8	11.0
SD	14.7	15.1	13.8	13.0	10.5
Hypomanic					
Mean	14.7	22.4	14.5	13.0	7.54
SD	10.9	11.4	10.5	9.77	7.59
Mixed					
Mean	6.33	9.86	7.33	4.77	3.41
SD	4.59	4.63	4.01	3.99	3.53
Externalizing					
Mean	69.6	73.8	68.4	70.9	58.9
SD	9.74	7.42	9.89	8.29	11.6
<b>Clinician-Rated</b>					
Depression					
Mean	21.6	29.3	27.6	15.4	17.1
SD	9.00	9.10	7.67	3.73	5.90
Hypomanic- Biphasic					
Mean	20.3	36.5	17.3	16.2	16.5
SD	9.58	8.90	4.82	4.23	5.63
Externalizing					
Mean	6.14	7.42	5.94	6.50	2.42
SD	3.72	3.79	3.71	3.41	2.53

Table 6

*Correlation Between Symptoms and Family Functioning*

Variable	GFES	PCM	FAD	KINDL Family	CBCL	KERS	PGBI Depression	KDRS	PGBI Mania	KMRS	PGBI Hypomania
PCM	-.03 [-.10, .04]	1									
FAD	-.17* [-.24, -.10]	.24* [.17, .30]	1								
KINDL Family	.09* [.03, .16]	-.43* [-.49, -.37]	-.37* [-.43, -.31]	1							
CBCL	-.13* [-.20, -.07]	.16* [.10, .23]	.10* [.03, .17]	-.33* [-.40, -.27]	1						
KERS	-.22* [-.29, -.15]	.23* [.16, .29]	.13* [.06, .20]	-.36* [-.42, -.29]	.57* [.52, .61]	1					
PGBI Depression	-.10* [-.17, -.03]	.25* [.18, .31]	.23* [.16, .29]	-.39* [-.44, -.33]	.25* [.19, .32]	.13* [.06, .20]	1				
KDRS	-.20* [-.26, -.13]	.16* [.10, .23]	.18* [.11, .24]	-.29* [-.35, -.22]	.05 [-.02, .12]	.07* [.00, .14]	.58* [.53, .63]	1			
PGBI Mania	-.11* [-.18, -.04]	.21* [.14, .27]	.11* [.04, .18]	-.34* [-.40, -.28]	.45* [.40, .51]	.29* [.22, .35]	.74* [.70, .77]	.30* [.24, .36]	1		
KMRS	-.12* [-.19, -.05]	.11* [.04, .17]	.05 [-.01, .12]	-.26* [-.32, -.19]	.22* [.15, .29]	.22* [.15, .28]	.37* [.30, .43]	.51* [.46, .56]	.43* [.37, .48]	1	
PGBI (Hypo)mania	-.11* [-.18, -.04]	.17* [.10, .23]	.08* [.01, .14]	-.29* [-.35, -.23]	.47* [.41, .52]	.30* [.24, .36]	.67* [.62, .71]	.22* [.16, .29]	.98* [.98, .98]	.39* [.33, .45]	1
PGBI Mixed	-.09* [-.16, -.02]	.27* [.20, .33]	.17* [.10, .23]	-.39* [-.45, -.33]	.34* [.28, .40]	.21* [.14, .28]	.79* [.76, .82]	.44* [.38, .49]	.88* [.86, .90]	.44* [.38, .49]	.77* [.74, .80]

*Note.* Values in square brackets indicate the 95% confidence interval for each correlation. GFES = Global Family Environment Scale score. PCM = Perceived Criticism Measure total score. FAD = Family Assessment Device total score. KINDL Family = The Revised Children Quality-of-Life Questionnaire (KINDL-R) family subscale score. CBCL = Child Behavior Checklist externalizing subscale. KERS = K-SADS Externalizing Rating Scale. PGBI = Parent General Behavior Inventory. KDRS = K-SADS Depression Rating Scale. KMRS = K-SADS Mania Rating Scale.

\* indicates  $p < .05$ .

### *Caregiver-reported family functioning*

Caregiver-Reported Symptom Severity: A series of MIMIC models tested whether caregiver-reported symptoms were associated with greater impairment in caregiver-reported family functioning. The first three models each adjusted for site gender, ethnicity, age, number of comorbid diagnoses, and BD diagnosis. Each of the three models added only one symptom dimension (i.e., externalizing, depressive, or (hypo)manic-biphasic symptoms) individually. Externalizing symptoms ( $\beta = .37$ , 95% CI [.31, .43],  $\Delta R^2 = .11$ ,  $p < .001$ ), depressive symptoms ( $\beta = .32$ , 95% CI [.26, .39],  $\Delta R^2 = .06$ ,  $p < .001$ ), and hypomanic-biphasic symptoms ( $\beta = .30$ , 95% CI [.24, .37],  $\Delta R^2 = .06$ ,  $p < .001$ ) were each individually associated with greater impairment in family functioning. The next model added all symptom dimensions (i.e., externalizing, depressive, and (hypo)manic-biphasic) simultaneously. After adjusting for all covariates and other symptom dimensions, externalizing symptoms ( $\beta = .32$ , 95% CI [.26, .37],  $\Delta R^2 = .11$ ,  $p < .001$ ) and depressive symptoms ( $\beta = .24$ , 95% CI [.15, .34],  $\Delta R^2 = .11$ ,  $p < .001$ ) were associated with greater family functioning impairment. Hypomanic-biphasic symptoms were no longer associated with family functioning,  $\beta = .01$ , 95% CI [-.09, .10],  $\Delta R^2 = .11$ ,  $p = .85$ . In summary, caregiver-reported externalizing and depressive symptoms were uniquely associated with impairments in caregiver-reported family functioning.

Next, we separated caregiver-reported (hypo)manic-biphasic symptoms into two subscales. The (hypo)manic subscale consisted of only increased manic symptom content (e.g., “Have there been times lasting several days or more when your child felt he/she must have lots of excitement, and he/she actually did a lot of new or different things?”). The mixed subscale consisted of items that had both manic and depressive symptom content (e.g., “Has your child’s mood or energy shifted rapidly back and forth from happy to sad or high to low?”). The first two

models each adjusted for site gender, ethnicity, age, number of comorbid diagnoses, and BD diagnosis. Each of the two models added a different symptom dimension (i.e., (hypo)manic or mixed symptoms) individually. Hypomanic ( $\beta = .26$ , 95% CI [.20, .33],  $\Delta R^2 = .05$ ,  $p < .001$ ) and mixed symptoms ( $\beta = .35$ , 95% CI [.28, .41],  $\Delta R^2 = .08$ ,  $p < .001$ ) were each individually associated with greater impairment in caregiver-reported family functioning. The next model tested whether hypomanic and mixed symptoms were associated with decreased family functioning after adjusting for study site, gender, ethnicity, age, number of comorbid diagnoses, BD diagnosis, externalizing symptoms, and depressive symptoms. (Hypo)manic symptoms were associated with increases in family functioning ( $\beta = -.14$ , 95% CI [-.20, -.07],  $\Delta R^2 = .19$ ,  $p < .001$ ), whereas mixed symptoms were associated with decreases in family functioning,  $\beta = .23$ , 95% CI [.15, .32],  $\Delta R^2 = .19$ ,  $p < .001$ . Therefore, combining (hypo)manic and mixed symptoms in the same scale washed out their opposing relationships with family functioning. Mixed symptoms were associated with reduced family functioning, whereas (hypo)manic symptoms were associated with improved family functioning.

Clinician-Rated Symptom Severity: Another series of MIMIC models tested whether clinician-rated symptoms were associated with decreases in caregiver-reported family functioning. The first three models each adjusted for site gender, ethnicity, age, number of comorbid diagnoses, and BD diagnosis. Each of the three models added a different symptom dimension (i.e., externalizing, depressive, or (hypo)manic-biphasic symptoms) individually. Externalizing symptoms ( $\beta = .33$ , 95% CI [.27, .39],  $\Delta R^2 = .08$ ,  $p < .001$ ), hypomanic-biphasic symptoms ( $\beta = .18$ , 95% CI [.07, .29],  $\Delta R^2 = .00$ ,  $p < .01$ ), and depressive symptoms ( $\beta = .15$ , 95% CI [.07, .22],  $\Delta R^2 = .00$ ,  $p < .001$ ), were each individually associated with greater impairment in family functioning. The next model added all symptom dimensions (e.g.

externalizing, depressive, and hypo(manic)-biphasic) simultaneously. After adjusting for all covariates and other symptom dimensions, externalizing symptoms ( $\beta = .34$ , 95% CI [.28, .40],  $\Delta R^2 = .09$ ,  $p < .001$ ) and depressive symptoms ( $\beta = .18$ , 95% CI [.11, .26],  $\Delta R^2 = .09$ ,  $p < .001$ ) were associated with greater family functioning impairment. Hypomanic-biphasic symptoms were no longer associated with caregiver-reported family functioning,  $\beta = .02$ , 95% CI [-.08, .12],  $\Delta R^2 = .09$ ,  $p = .65$ . In summary, clinician-rated externalizing and depressive symptoms are associated with impairments in caregiver-reported family functioning.

Unlike parent-reported symptoms, clinician-reported (hypo)manic-biphasic symptoms were unable to be separated into two subscales. The KMRS contains only one item that either confirms or denies the presence of mixed symptoms. Therefore, this item does not allow for an assessment of mixed symptom severity ratings such as that measured by the PGBI.

#### *Clinician-rated family functioning*

Caregiver-Reported Symptom Severity: A series of linear regressions tested whether caregiver-reported symptoms were associated with decreases in clinician-rated family functioning. The first three models each adjusted for site gender, ethnicity, age, number of comorbid diagnoses, and BD diagnosis. Each of the three models added a different symptom dimension (i.e., externalizing, depressive, or (hypo)manic-biphasic symptoms) individually. Caregiver-reported externalizing symptoms were individually associated with decreases in GFES scores,  $\beta = -.08$ , 95% CI [-.17, -.00],  $\Delta R^2 = .01$ ,  $p = .03$ . Neither depressive symptoms ( $\beta = -.02$ , 95% CI [-.06, .02],  $\Delta R^2 = .00$ ,  $p = .20$ ) nor hypomanic-biphasic ( $\beta = -.05$ , 95% CI [-.11, .01],  $\Delta R^2 = .00$ ,  $p = .10$ ) symptoms were individually associated with changes in GFES scores. In the next model, externalizing, depressive, and hypo(manic)-biphasic symptoms were added simultaneously. After adjusting for all covariates and other symptom dimensions, no symptoms

(i.e., externalizing, depressive, and (hypo)manic-biphasic) were associated with GFES scores,  $\beta$ s = -.08 to -.01,  $\Delta R^2 = .01$ ,  $p$ s > .08. In the final model, the interaction between depressive and (hypo)manic-biphasic symptoms were added. There were no significant interactions between depressive and (hypo)manic-biphasic symptoms,  $\beta = .00$ , 95% CI [.00, .00],  $\Delta R^2 = .00$ ,  $p = .94$ . In summary, caregiver-reported externalizing, depressive, and (hypo)manic symptoms were not uniquely associated with clinician-reported family functioning.

In the next series of linear regressions, caregiver-reported (hypo)manic-biphasic symptoms were separated into two subscales. The first two models each adjusted for site gender, ethnicity, age, number of comorbid diagnoses, and BD diagnosis. Each of the two models added a different symptom dimension (i.e., (hypo)manic or mixed symptoms) individually.

(Hypo)manic ( $\beta = -.07$ , 95% CI [-.15, .01],  $\Delta R^2 = .00$ ,  $p = .10$ ) and mixed symptoms ( $\beta = -.12$ , 95% CI [-.32, .08],  $\Delta R^2 = .00$ ,  $p = .19$ ) were not individually associated with GFES scores. The next model tested whether hypomanic and mixed symptoms were associated with decreased family functioning after adjusting for study site, gender, ethnicity, age, number of comorbid diagnoses, BD diagnosis, externalizing symptoms, and depressive symptoms. (Hypo)manic symptoms ( $\beta = -.03$ , 95% CI [-.17, .11],  $\Delta R^2 = .01$ ,  $p = .66$ ) and mixed symptoms ( $\beta = .02$ , 95% CI [-.31, .35],  $\Delta R^2 = .01$ ,  $p = .93$ ) remained unassociated with GFES scores. In summary, caregiver-reported (hypo)manic and mixed symptoms were not associated with clinician-rated family functioning.

Clinician-Rated Symptom Severity: A series of linear regressions tested whether clinician-rated symptoms were associated with decreases in clinician-rated family functioning. The first three models tested whether clinician-rated externalizing, depressive, and hypomanic-biphasic symptoms, respectively, were associated with changes in family functioning after

adjusting for site, gender, ethnicity, age, number of comorbid diagnoses, and BD diagnosis. Depressive symptoms ( $\beta = -.16$ , 95% CI [-.25, -.07],  $\Delta R^2 = .06$ ,  $p < .001$ ), externalizing symptoms ( $\beta = -.17$ , 95% CI [-.24, -.09],  $\Delta R^2 = .02$ ,  $p < .001$ ), and hypomanic-biphasic symptoms ( $\beta = -.16$ , 95% CI [-.27, -.04],  $\Delta R^2 = .01$ ,  $p = .01$ ) were all individually associated with decreases in GFES scores. In the next model, externalizing, depressive, and hypo(manic)-biphasic symptoms were added simultaneously. After adjusting for all covariates and other symptom classes, depressive symptoms ( $\beta = -.19$ , 95% CI [-.28, -.04],  $\Delta R^2 = .05$ ,  $p < .001$ ) and externalizing symptoms ( $\beta = -.18$ , 95% CI [-.26, -.11],  $\Delta R^2 = .05$ ,  $p < .001$ ) were associated with decreased GFES scores. Hypomanic-biphasic symptoms were not associated with changes in GFES scores,  $\beta = -.03$ , 95% CI [-.17, .11],  $\Delta R^2 = .05$ ,  $p = .73$ . In the final model, the interaction between depressive and (hypo)manic symptoms were added. There were no significant interactions between depressive and (hypo)manic-biphasic symptoms,  $\beta = .00$ , 95% CI [-.01, .01],  $\Delta R^2 = .00$ ,  $p = .87$ . In summary, clinician-rated externalizing and depressive symptoms were associated with impaired clinician-rated family functioning.

## CHAPTER 6

### DISCUSSION

Youth with psychopathology tend to have impaired family functioning. In particular, leading theories of mood disorder development in children and adolescents rely on disruptions in family functioning for both the development, maintenance, and treatment of the difficulties (Frazier et al., 2020; MacPherson et al., 2016; Reinares et al., 2016; Weintraub et al., 2019). BD represents a unique mood disorder in that it features an externalizing-oriented mood (i.e., mania) and nearly always an internalizing mood (i.e., depression; Peters et al., 2018; Youngstrom et al., 2008). BD in youth also tends to be highly comorbid with externalizing problems (e.g., oppositionality & defiance; Frías et al., 2015). Depressive, (hypo)manic, and externalizing behavior symptoms all demonstrate independent influences on family functioning (Deault, 2010; Rosa et al., 2009; Sullivan et al., 2012). Therefore, the current study aimed to identify the relative contributions of each symptom set (i.e., depressive, (hypo)manic, externalizing, or a combination) to disruptions in family functioning.

#### **Diagnostic Associations with Family Functioning**

Family functioning among youth with BD was first compared to family functioning among youth with other common psychiatric disorders presenting to outpatient clinics. Both caregivers and clinicians of youth with BD reported worse family functioning compared to caregivers and clinicians of youth with behavior disorders (e.g., oppositional defiant disorder, conduct disorder, attention-deficit/hyperactivity disorder) and youth with other non-mood/non-behavior disorders (e.g., anxiety disorders). However, only caregivers reported worse family functioning compared to youth with unipolar depression. These findings were mostly consistent

with Freeman and colleagues (2009), who demonstrated that youth with bipolar disorder show a similar profile of impairment in family quality of life. Individuals with BD spend more time in depressive episodes compared to manic episodes (Judd et al., 2002; Kupka et al., 2007), and depressive symptoms have been linked to greater functional impairment among youth with BD (Akbaş et al., 2017; Rosa et al., 2009). In the current sample, youth were more likely to be in a depressive episode than a manic episode. Our findings reinforce that depressive episodes may be the driver of psychosocial impairment.

Similar to the current findings, Young and colleagues (2013) demonstrated that youth with BD report worse problem solving compared to youth with ADHD (which falls within the externalizing spectrum of behavioral disorders; (Kotov et al., 2021). However, unlike the current results, youth with BD and ADHD reported similar general family functioning (Young et al., 2013). One potential explanation for this difference may be the way family functioning was measured in each study. Young and colleagues (2013) utilized only the FAD as a measure of family functioning. In contrast, the current study used factor analysis to create a latent family functioning variable. One benefit of creating a latent variable is that it removes measurement error which can attenuate the relationship. Additionally, of the three measures of family functioning incorporated in this factor analysis, the FAD demonstrated the lowest loading on this latent family functioning variable. This comparatively low loading may have precluded Young and colleagues (2003) from finding a representation of this relationship similar to that demonstrated in the current study. In summary, families of youth with BD are perceived as having significantly worse family functioning compared to families of youth with behavior disorders and youth with other non-mood and non-behavior disorders, but not necessarily youth

with unipolar depression. These findings emphasize the importance depressive symptoms may play in the well-being of youth with BD.

### **Symptomatic Associations with Family Functioning: Hypomania as a Protective Factor?**

The association between impairments in family functioning and depressive, (hypo)manic, and externalizing symptom severity was examined. Both clinician and caregiver-reported depressive and externalizing symptoms were most associated with decline in both caregiver-reported and clinician-reported family functioning. These findings were consistent with previous research, which found that youth with depression report poorer family functioning (Frazer & Fite, 2016; Kashani et al., 1995; Pereira et al., 2015; Simpson et al., 2018; Tamplin et al., 1998). Furthermore, among youth with BD, the presence of comorbid behavior disorders and externalizing behaviors like aggression is associated with greater impairments in family functioning (Esposito-smythers et al., 2006; Keenan-Miller et al., 2012; Weintraub et al., 2019). In contrast, after adjusting for depressive and externalizing symptoms, (hypo)manic-biphasic symptoms were unassociated with family functioning regardless of reporting source. However, when (hypo)manic-biphasic symptoms were separated, (hypo)manic symptoms were associated with greater caregiver-rated family functioning, whereas mixed symptoms were associated with worse caregiver-rated family functioning. This is inconsistent with previous research, which found that manic symptoms impair family functioning (Calabrese et al., 2004; Rosa et al., 2010).

The current results may indicate that the unique variance in mild (hypo)mania may be protective in the context of depression, whereas the presence of mixed symptoms could be impairing. These findings suggest that the specific profile of “pure” versus “mixed” (hypo)mania symptoms may matter when assessing the impact of mania on a person’s psychosocial functioning, whereas the current findings support that depressive symptoms may be the primary

driver of impairment for youth with BD. Alternatively, these findings may be a result of suppression stemming from a significant overlap between the impact of depression and mania symptoms, such that the residual variance in (hypo)manic symptoms in this study is fundamentally different from the clinical presentation of (hypo)mania (G. A. Miller & Chapman, 2001). In summary, depressive and externalizing symptoms may be driving declines in family functioning among youth with BD.

### **Treatment Implications**

Overall, youth with BD have impaired family functioning that appears to be most consistently associated with depressive and externalizing symptoms. Targeting depressive symptoms (e.g., behavior activation in high positive emotion family activities; Martin & Oliver, 2019) and externalizing symptoms (e.g., caregiver training for behavior management; Michelson et al., 2013) may be critical for improving family functioning. We can also consider the effect that targeting family functioning may have on symptom severity. Family systems theory proposes a reciprocal relationship between an individual's functioning and the family environment (Kerr & Bowen, 1988). Therefore, targeting family functioning (e.g., improving communication, increasing adaptability, and enhancing relationship quality; MacPherson et al., 2016) may also improve depressive and externalizing symptoms. In summary, although (hypo)mania is a cardinal symptom of BD, depressive and externalizing symptoms may be a more appropriate treatment target among youth with BD.

### **Limitations and Future Directions**

Limitations of the present study include the use of a cross-sectional design. Caregiver and clinician reports varied in respect to the mood symptom and family functioning time frames,

such that the timelines for reports of family functioning and reports of symptom severity were not perfectly overlapping (i.e., some time periods were shorter and others longer). Cross-sectional designs with low temporal specificity result in limitations regarding the temporal association between mood and externalizing symptoms and family functioning. A longitudinal research design would aid in tracking youth across different mood episodes to identify the precise effects of mood symptoms on family functioning in youth with BD. Similar designs have been used to determine the temporal order of expressed emotion and psychotic symptoms among individuals with schizophrenia resulting in findings that family functioning is both a causal factor and a sequela of psychotic symptoms (Ma et al., 2021).

Clinicians and caregivers also reported youth mood and externalizing symptoms using different filtering methods. Clinicians filtered symptoms to assess them for individual disorders, a method that is associated with more reliable diagnostic decision making and specificity (Yee et al., 2015). In contrast, caregiver ratings of youth symptoms were based on unfiltered reports across a number of measures, a method that is associated with greater sensitivity but poorer specificity. This may have resulted in inconsistencies between caregiver and clinician reports and altered the relationships found between family functioning and symptom severity (e.g., the lack of relationships found between all caregiver-reported symptoms and clinician-rated family functioning). However, despite differences among informants, the core findings remained the same, with depressive and externalizing symptoms being the most impairing.

Another limitation that warrants consideration in the current study is the possibility of distortions in self-reported symptoms due to elevated mental health stigma in racial and ethnic minority groups. The current sample was largely Black (with Black participants comprising 90% of the non-White sample). Though this diversity strengthens the ability to generalize our findings

beyond White Americans, it may have also led to a depression of parent-reported symptom severity. Black Americans and other ethnic minority groups within America express greater societal and self-stigma regarding mental health compared to White Americans (Misra et al., 2021). This could result in greater concealment and non-disclosure of symptoms and symptom severity among Black participants within the current study compared to White participants. However, the diversity of our sample is also a large strength of the current study. Previous research has shown significant differences in parenting practices, family dynamics, and associated child outcomes among ethnic and racial minority families (Dunbar et al., 2017; Tang & Davis-Kean, 2015).

These differences may also influence how we can define family functioning and how family functioning relates to child psychopathology within racially and ethnically diverse families. Much of the research on family functioning and psychopathology utilizes samples consisting of largely White participants. Therefore, our current knowledge of the relationship between family functioning and psychopathology may not accurately describe the experience of racial and ethnic minorities. Nevertheless, findings from our Black-majority sample remained largely consistent with previous conclusions about the associations between family functioning, psychopathology, and symptom severity. Although we were not specifically looking at racial and ethnic differences in family functioning and psychopathology, the current findings provide more confidence in the ability to generalize results on family functioning and symptom severity to Black youth and families.

## **Conclusions**

In summary, this study provides evidence that family functioning is impaired in youth with BD relative to youth with behavioral disorders and other non-mood, non-behavioral

disorders. Additionally, depressive and externalizing symptoms may be particularly related to family functioning among youth with BD. Though manic symptoms are thought of as a cardinal symptom of BD, other symptoms (e.g., depressive and externalizing) may have a greater influence on the functioning of youth with BD and their families and serve as important targets of treatment. Overall, family functioning is an important indicator of well-being for both youth and their families, and expanding our understanding of the relationship between BD symptoms (i.e., depressive, manic, mixed, and externalizing symptoms) and family functioning can help inform mechanistically sound family-based interventions and advance treatment for youth with BD.

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## CURRICULUM VITAE

### **Kayla Fobian**

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Las Vegas, NV 89154  
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### **EDUCATION**

#### **University of Connecticut**

Storrs, CT

*Bachelor of Science*

Graduation Date: May 6, 2018

Summa Cum Laude

Psychological Sciences, Honors Degree

Human Development and Family Studies, Double Major

Minor in Neuroscience

GPA: 3.99/4.00

#### **University of Nevada, Las Vegas**

Las Vegas, NV

*Doctor of Philosophy*

Diploma Expected: May 2025

Clinical Psychology

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### **RESEARCH EXPERIENCE**

#### **University of Nevada, Las Vegas (UNLV), Psychology Department**

Ecological Momentary Assessment of Mood and Family Functioning      January 2021 – Present

PI: Stephen Benning, Ph.D.

*Researcher*

- Use ecological momentary assessment (EMA) to explore daily patterns of mood and family functioning.
- Review previous EMA literature to inform appropriate study procedures.
- Develop EMA items to assess family functioning and general mood.
- Assist in developing an app to distribute EMA questionnaires to participants.
- Train research assistants on data collection procedures.

Self-Reported Quality of Life in Youth with Bipolar Disorder

September 2019- Present

PI: Andrew Freeman, Ph.D.

*Researcher*

- Investigate the impact of bipolar disorder on the quality of life in youth using secondary data analysis.
- Conduct literature reviews and form hypotheses.
- Design and run all analyses in R.
- Collaborate with co-authors on a paper to be submitted for publication.

Family Functioning in Youth with Bipolar Disorder  
Master's Thesis Project

August 2019- Present

PI: Stephen Benning, Ph.D.

*Researcher*

- Investigate the impact of bipolar disorder on family functioning in youth using secondary data analysis.
- Conduct literature reviews and form hypotheses.
- Design and run all analyses in R.
- Write a proposal document and present proposal to Advisory Committee in October 2020.
- Edit and revise proposal document for final thesis defense.
- Presented defense, estimated November 2021.

Young Adults Attending to Emotions

August 2019- March 2020

PI: Andrew Freeman, Ph.D.

Dissertation Project of Breanna Garcia

*Lab Manager*

- Ran participants through the study procedure including completion of multiple self-report questionnaires and eye tracking tasks, the Five-Minute Speech Sample (FMSS), and the Mini- International Neuropsychiatric Interview (M.I.N.I.).
- Managed undergraduate research assistants (e.g. train on study procedures, schedule and manage lab hours, and provide direct supervision and guidance during daily lab tasks).
- Create transcription and coding protocols for the FMSS data, train research assistants on transcription and coding. procedure, and manage transcribed and coded FMSS data (e.g. provide guidance for research assistants and review the final transcribed and coded data).

### **University of Connecticut (UConn), Psychology Department**

Early Detection of Autism Spectrum Disorders

PI: Deborah Fein, Ph.D.

*Research Coordinator*

May 2018- May 2019

- Primary research coordinator for a long-term, multi-site, research project investigating the early detection of autism spectrum disorder (ASD). This project aims to determine the optimal screening schedule for ASD, in order to achieve the earliest possible detection while still maintaining low false positive rates.
- Coordinated with research assistants at other study sites to ensure all universities remain informed of research progress
- Corresponded with participating pediatrician sites to refill screening packets and to schedule travel evaluations at the pediatrician offices.
- Managed undergraduate research assistants (e.g. train on study procedures, schedule and manage lab hours, and provide direct supervision and guidance during daily lab tasks).
- Conducted follow-up phone interviews for participants who failed the M-CHAT-R or were missing items on screening measures.
- Coordinated with graduate students and clinicians to create the evaluation calendar each school year and schedule evaluations.
- Scored screening and evaluation measures (i.e. M-CHAT-R, FYI, ITC, Vineland-II, CBCL, ITSEA, and MCDI)
- General study management (e.g. organize lab meetings and order study materials).

*Undergraduate Research Assistant*

August 2015-May 2018

- Conducted follow-up phone interviews for participants who failed the M-CHAT-R or were missing items on screening measures.
- Determined participant eligibility and scheduled evaluations.
- Helped to prepare and organize study materials.
- Assisted on several additional graduate student projects by pulling and analyzing data.

Evaluation Follow-Up Study

June 2018- May 2019

PI: Deborah Fein

Dissertation Project of Cara Cordeaux

*Researcher*

- Exploring the associations between parent resolution to diagnosis status, parent and child characteristics (i.e. parent self-efficacy, child symptom severity), and treatment-seeking behaviors soon after receiving a child's developmental disorder diagnosis (i.e. ASD, global developmental delay, language disorder) and recommendations for intervention.
- Transcribe and code parent responses to the Reaction to Diagnosis Interview and determine the extent to which the parent has resolved the experience of their child's diagnosis.

Novelty and Familiarity Preference in Toddlers

January 2017-May 2018

Honors Thesis Project

PI and thesis advisor: Deborah Fein, Ph.D.

*Researcher*

- Investigated the preference behavior of toddlers and compared the novelty preference behavior of children with autism spectrum disorder, children with other developmental delays, and typically developing children.
- Supervised directly by a graduate student and faculty advisor.
- Gathered background literature, formed a hypothesis, and designed a procedure to test the hypothesis.
- Organized and submitted proper IRB documentation.
- Completed play-based evaluations investigating play behavior with novel toys.
- Developed a coding system for play behavior and coded all evaluation videos.
- Trained other undergraduate students for the administration of evaluations.
- Completed a final research paper to present findings to my thesis advisor and the UConn honors office.

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**CLINICAL EXPERIENCE**

**The Evidenced Based Practice of Nevada**

June 2021 – Present

Las Vegas, NV

Role: *Psychological Trainee*

- Individual Therapy
  - Utilize both telehealth and in-person services to provide evidence-based psychotherapy to youth ages 6 to 12.

- Provided services informed by cognitive-behavioral therapy, exposure therapy, and parent management training.
- Conduct risk assessments for clients endorsing suicidal ideation.
- Conduct intake interviews and formulate diagnostic impressions and treatment recommendations for new clients.
- Didactic Training
  - Discuss a variety of topics aimed at enhancing professional development including, interprofessional collaboration and adjunctive services, evidence-based practice, biological rhythms, escalation cycle and crisis management, caregiver involvement in therapy, and ethical guidelines.

**The PRACTICE at UNLV**

August 2020-June 2021

Las Vegas, NV

Role: *Psychological Trainee*

- Individual Therapy
  - Utilize telehealth procedures to provide individual services that align with the CDC COVID-19 recommendations.
  - Conduct individual therapy with children ages 7 to 17 and their parents, utilizing Trauma-Focused Cognitive Behavioral Therapy, Dialectical Behavioral Therapy, mindfulness, and behavioral training techniques.
  - Collaborate with Community in Schools (CIS) to conduct individual therapy with children from rural areas of Nevada.
  - When appropriate, collaborate with other clinicians working with family members of my clients (e.g., parents or child) to coordinate client care.
  - Conduct risk assessments and develop safety plans for clients endorsing suicidal ideation.
  - Conduct intake interviews and formulate treatment recommendations for new clients.
- Group Therapy
  - Co-facilitate Befriending Emotions (B.E.) Kids group therapy with children ages 8 – 11 years old, a group which teaches and empowers children to identify, communicate, and cope with emotions.
  - Co-facilitate Making Connections © grief and loss support groups for children and caregivers. Utilize an evidence-based curriculum to support grieving children and caregivers who have experienced the death of a special person.
  - Helped develop a social emotional learning curriculum for children ages 7 – 11 years old.
- Assessment
  - Conduct psychological assessments to assess cognitive, academic, language, and social-emotional functioning. Adjust assessment procedures to align with COVID-19 recommendations endorsed by the CDC.
  - Collaborate with a Spanish-speaking clinician, to determine diagnoses and recommendations for a Spanish speaking client and family. Conduct interviews with teachers and school staff for the purpose of assessment and diagnosis.
- Ensure administrative requirements are fulfilled by clients (e.g. payments and required paperwork).

**UNLV Research - Young Adults Attending to Emotions** August 2019- March 2020  
Las Vegas, NV

Role: *Lab Manager*

- Complete structured diagnostic interviews (M.I.N.I.).
- Work with study P.I. to conduct risk assessments for participants endorsing suicidal ideation.

**UConn Research – Early Detection of Autism Spectrum Disorders** May 2018 – May 2019  
Storrs, CT

Role: *Research Coordinator*

- Conducted developmental evaluations on children age 15 months to 40 months using the ADOS-2, MSEL, and BOSCC.
- Collaborated with a graduate student and licensed clinician to determine diagnoses and complete a diagnostic report.

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## CLINICAL TRAINING

**Everyday Parenting: The ABCs of Child Rearing** 2021  
Web-based training, Alan E. Kazdin, Ph.D., ABPP

**TF-CBT Web 2.0: A Course for Trauma-Focused Cognitive Behavioral Therapy** 2021  
Web-based training, certificate of completion granted by Daniel W. Smith, Course Director

**Making Connections® Bereavement Group Facilitator and Assistant Training** 2020  
Virtual training over Zoom, presented by Robin F. Goodman, Ph.D., A.T.R.-BC

**American Psychological Association, Telepsychology Best Practices 101** 2020  
*Clinical Evaluation and Care: Cultural Competencies; Documentation - Segment #1*  
*About the Tech... Video, Email, Text Messaging & Apps - Segment #2*  
*Legal, Regulatory & Ethical Rules of the Road - Segment #3*  
*Getting Paid: Reimbursement Strategies & Marketing Your Professional Services Online - Segment #4*  
All segments completed as virtual training over Zoom, presented by Marlene M. Maheu, PhD

**Not all that blows up is Bipolar: Evidence-Based Assessment and Treatment for Bipolar Disorder in Youth and Young Adults** 2019  
Las Vegas, NV; workshop hosted by the Nevada Psychological Association; presented by Eric Youngstrom, Ph.D.

**Autism Diagnostic Observation Schedule (ADOS-2) Training** 2018  
*Modules 1-4*, University of Connecticut, presented by So Hyun Kim, Ph.D.  
*Toddler Module*, Center for Autism and the Developing Brain, presented So Hyun Kim, Ph.D.

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**CONFERENCE TALKS AND POSTER PRESENTATIONS**

Dennis, S. J., Fobian, K., Poston, M., Wrenn, K. H., and Benning, S. D. (May, 2021). Meanness and Sacrificial Moral Decision Making. Poster to be presented at the Society for the Scientific Study of Psychopathy Early Career Event, virtually held.

Fobian, K., Janos, J., Youngstrom, J., Findling, R., Youngstrom, E.A., and Freeman, A. (November, 2020). Self-Reported Quality of Life in Youth with Bipolar Disorder. Presented as part of the symposium session, **“From Symptoms to Functioning in Children and Adolescents Across Care Settings” at the Association for Behavioral and Cognitive Therapies Annual Convention, virtually held.**

Fobian, K., Coulter, K., Robins, D. L., Barton, M., & Fein, D. (2019, May). Sex differences in CARS Scores for Toddlers with Autism Spectrum Disorders. Poster presented at the International Society for Autism Research Annual Meeting, Montreal, Canada.

Fobian, K. (2017, October). Novelty and Familiarity Preference in Toddlers. Poster presented at the Fall Frontiers in Undergraduate Research Poster Session, University of Connecticut, Storrs, CT.

Fobian, K., Fernandez, J., & Mansfield, A. (2015, December). The Effects of Emotion on Helping Behavior. Poster presented at the PSYC 2100WQ Poster Night, University of Connecticut, Storrs, CT.

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**HONORS, AWARDS, AND GRANTS**

Summer Undergraduate Research Fund (SURF) Award, University of Connecticut  
*Summer 2017*

CLAS Alan R. Bennett Honors Funds, UConn Fall Frontiers poster production grant  
*Fall 2017*

UConn Babbidge Scholar  
*2014-2018*

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**PROFESSIONAL MEMBERSHIP**

American Psychological Association	2021 - Current
Nevada Psychological Association	2019-Current
American Psychological Association, Division 53	2019-Current
Psi Chi National Honors Society	2016-Current
National Society of Collegiate Scholars	2014-Current
Alpha Lambda Delta Honor Society	2014-Current

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**ADDITIONAL SKILLS:**

- *Eye-tracking:* Tobii
- *Databases:* Filemaker, OpenSesame
- *Data Analysis:* SPSS, R