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Parenting practices in middle childhood mediate the relation between growing up with a parent having bipolar disorder and offspring psychopathology from childhood into early adulthood

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Abstract

The offspring of parents with bipolar disorder (OBD) are at high risk for developing mental disorders. In addition to genetic factors, environmental risk is purported to be associated with these negative outcomes. However, few studies have examined this relation. Using concurrent and longitudinal data, we examined if support, structure, and control provided by parents in middle childhood mediated the relation between having a parent with or without bipolar disorder, and offspring mental health. The sample included 145 offspring (77 OBD, 68 controls) aged 4 to 14 years and their parents. Parent and teacher ratings of child behavior were collected, and diagnostic assessments were conducted in offspring 12 years later ($n = 101$). Bootstrapping analyses showed that low levels of structure mediated the relation between having a parent with bipolar disorder and elevated internalizing and externalizing difficulties during middle childhood. For the longitudinal outcomes, parental control emerged as the strongest mediator of the relation between parents' bipolar disorder and offspring psychopathology. Suboptimal childrearing may have different immediate and enduring consequences on mental health outcomes in the OBD. Parental structure has robust effects on emotional and behavioral problems in middle childhood, while levels of control promote psychological adjustment in the OBD as they mature.

Bipolar disorder (BD) is a chronic and debilitating psychiatric illness. It is among the top 10 leading causes of disability worldwide (World Health Organization, 2001), by virtue of its association with severe psychosocial dysfunction, suicidality, and high comorbidity (Hodgins, Faucher, Zarac, & Ellenbogen, 2002; Schaffer, Cairney, Cheung, Veldhuizen, & Levitt, 2006). As such, it entails important societal and economic costs (Begley et al., 2001; Das Gupta & Guest, 2002), including particularly heavy burdens for the offspring of parents with BD (OBD). There is substantial evidence of adjustment and mental health problems among the OBD. The OBD display rates of attention, disruptive behavioral, and anxiety disorders in childhood two to nine times those observed among children with healthy parents (Birmaher et al., 2009; Singh et al., 2007). Recent clinical staging models suggest that the OBD are likely to exhibit age-specific emotional and behavioral difficulties prior to the development of subclinical affective symptoms and later major affective disorders (Duffy, Alda, Hajek, Sherry, & Grof, 2010; Duffy et al., 2014; Miklowitz & Cicchetti, 2006, 2010). By young adulthood, approximately 30%–50% of the OBD will have developed a major affective disorder compared with only 10% of the offspring of parents with no affective disorder (ONAD; Mesman, Nolen, Reichart, Wals, & Hillegers, 2013; Nijjar, Ellenbogen, & Hodgins, 2014; Rasic, Hajek, Alda, & Uher, 2013).

Adverse outcomes for the OBD likely stem from a complex interaction between inherited traits and exposure to suboptimal childrearing environments (Brietzke et al., 2012). Genetic predisposition is the primary vulnerability factor reported in twin and adoption studies, with heritability estimates ranging between 60% and 93% (Kieseppa, Partonen, Haukka, Kaprio, & Lonnqvist, 2014; McGuffin et al., 2003). At least one part of the genetic risk may be the transmission of alleles that increase sensitivity to both negative and positive environmental factors (Nilsson, Comasco, Hodgins, Oeland, & Åslund, 2015). In addition, since genetic risk does not entirely account for the intergenerational transmission of psychopathology, growing up with a parent who periodically becomes psychotic, dysfunctional, neglectful, or abusive has also been described as a dominant influence on the development of the OBD (Alloy et al., 2005; Post, Leverich, Xing, & Weiss, 2001). Disruptions in caregiving in families in which one parent has BD are well documented,

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as indicated by high levels of marital discord and separation (Dore & Romans, 2001; Lam, Donaldson, Brown, & Malliaris, 2005), parental absenteeism (e.g., due to hospitalizations; Pini et al., 2005), nonoptimal parenting (e.g., use of lax disciplinary techniques; Calam, Jones, Sanders, Dempsey, & Sadhnani, 2012), insecure offspring attachment (DeMulder & Radke-Yarrow, 1991), and negative communication styles (Inoff-Germain, Nottelmann, & Radke-Yarrow, 1992; Meyer et al., 2006; Vance, Huntley, Espie, Bentall, & Tai, 2008). With regard to general family dynamics, lower levels of cohesion and organization, and elevated conflict characterizes families in which a parent has BD relative to control samples (e.g., Barron et al., 2014; Chang, Blasey, Ketter, & Steiner, 2001; Ferreira et al., 2013; Romero, Delbello, Soutullo, Stanford, & Strakowski, 2005).

Dysfunctional caregiving environments in childhood are well-established risk factors for a broad array of mental disorders across the life span (see Yap & Jorm, 2015; Yap, Pilkington, Ryan, & Jorm, 2014, for meta-analyses). Within the literature on affectively ill parents, there is robust evidence that unipolar depression in a parent contributes to illness onset in children at genetic risk via an impaired caregiving environment (see Beardslee, Gladstone, & O'Connor, 2011, for a review). Among parents with BD, negative bidirectional correlations have been described between the quality of the childrearing environment and the experience of emotional and behavioral problems in the OBD (Calam et al., 2012; Ferreira et al., 2013; Freed et al., 2015). In a longitudinal investigation of the children of mothers with mood disorders (Meyer et al., 2006; Radke-Yarrow, 1998), extreme displays of negative affect and attitudes among mothers with BD during early childhood were predictive of risk for bipolar illness approximately 20 years later via negative alterations in frontal lobe functioning. Pioneering work by Miklowitz and colleagues has also shed light on the characteristics of the family environment relevant to BD, linking high levels of expressed emotion (i.e., criticism, hostility, and/or emotional overinvolvement) in family members to an increased likelihood of relapse (Miklowitz, Goldstein, Nuechterlein, Snyder, & Mintz, 1988) as well as longer and more severe illness episodes (Miklowitz, Wisiewski, Miyahara, Otto, & Sachs, 2005) in adolescents and adults diagnosed with BD. However, knowledge of the childrearing mechanisms underlying the parent–child transmission of psychopathology within the context of parental bipolar illness remains scarce.

To date, only one cross-sectional study has examined and found a small, but statistically significant indirect pathway from parents' BD to current BD in offspring via heightened levels of family conflict (Schudlich, Youngstrom, Calabrese, & Findling, 2008), suggesting that the OBD may be especially susceptible to being exposed to a caregiving environment characterized by high levels of stress, chaos, and instability. Relative to control offspring, the OBD tend to experience more frequent and severe stressful life events (Ostiguy et al., 2009). Among those who eventually develop a mental disorder, negative life events often precede onset (Hillegers et al., 2004; Wals et al., 2005). High levels of the trait neuroticism (i.e., a tendency to react with elevated emotionality to stressors) in parents with BD have also been postulated to elicit unstable and disorganized caregiving environments in middle childhood that increases the risk for later high-risk sexual behaviors and poor interpersonal functioning in the OBD (Ellenbogen & Hodgins, 2004; Nijjar, Ellenbogen, & Hodgins, 2016; Ostiguy, Ellenbogen, & Hodgins, 2012). Likewise, structure provided by parents in middle childhood has been shown to influence cortisol reactivity in adolescence among the OBD (Ellenbogen & Hodgins, 2009; Ellenbogen, Hodgins, & Walker, 2004). In turn, persistent abnormalities in individuals' biological sensitivity to stress have been associated with an increased vulnerability for the development of an affective disorder (Ellenbogen, Hodgins, Linnen, & Ostiguy, 2011; Goodyer, Bacon, Ban, Croudace, & Herbert, 2009). Thus, stability, predictability, and cohesion in the childrearing environment may be especially important environmental predictors of health outcomes in youth who have a parent with BD, although knowledge of specific risk mechanisms remains elusive.

Ultimately, the current literature base highlights the need for additional research exploring the causal pathways linking the caregiving environment to mental health outcomes in the OBD. Specifically, longitudinal designs would help determine causality between both factors. It is also possible that disrupted caregiving processes that stem from parental affective disorder convey risk for a variety of maladaptive psychiatric outcomes in youth, and not just for the development of BD. In accordance with clinical staging models of BD, alternative, nonmood disorders should also be investigated as potential adverse outcomes for the OBD. Accordingly, the objectives of the current study were twofold: (a) to compare the quality of childrearing between families with a parent having BD and control families where neither parent has an affective disorder, and (b) to determine if the quality of the childrearing environment would mediate the relation between offspring's risk status (having a parent with BD vs. no affective disorder) and concurrent and prospective internalizing and externalizing symptoms in their offspring. Impairments in the caregiving environment were evaluated across three domains of parenting practices as rated by all parents within a family: mean levels of support (emotional warmth), structure (i.e., organization and consistency), and control (i.e., disciplinary practices). We hypothesized that parents with BD would display nonoptimal parenting in all domains relative to control parents. Moreover, we expected that having a parent with BD would increase offspring's likelihood of experiencing emotional and behavioral problems by way of increased disruptions in parenting practices during middle childhood. We explored these patterns at the Time 1 assessment, using concurrent measures, and prospectively by examining if parenting practices in middle childhood would continue to mediate the association between offspring risk status (OBD vs. ONAD) and offspring symptomatology approximately 12 years later as they transitioned into adulthood. In light of pre-

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vious research showing a greater effect of structure, relative to support and control, provided by parents in middle childhood on stress reactivity in the adolescent OBD (Ellenbogen & Hodgins, 2009), we postulated that the presence of low structure would yield the strongest predictive relations with symptoms of psychopathology among the OBD relative to the ONAD. Given that there are sex differences in vulnerability to specific internalizing (Angold, Erkanli, Silberg, Eaves, & Costello, 2002) and externalizing (Broidy et al., 2003) pathologies in youth, we also explored whether sex of the offspring moderated the aforementioned statistical associations. To account for the large age range in the current sample (4 to 13 years at Time 1), and the fact the quality of family functioning varies with socioeconomic status (SES; Bradley & Corwyn, 2002), age of the offspring and family income (used as a proxy of SES) were controlled for in all analyses. In order to account for the continuity of mental health problems, issues of bidirectionality, and the possibility of evocative effects in the parent–child relationship (Larsson, Viding, Rijsdijk, & Plomin, 2008), psychiatric diagnoses in offspring in middle childhood were controlled for in the mediation analyses. Parents' antisocial personality traits were also included as a covariate to control for the possibility of genetically or environmentally transmitted effects involving parents' antisocial behavior (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005). To the best of our knowledge, this is the first study to evaluate the mediating role of parenting practices on both concurrent and longitudinal mental health outcomes in the OBD.

Method

Participants

Participants were recruited for a longitudinal investigation comparing the development of the OBD with that of the ONAD. Parents with a diagnosis of BD were recruited from psychiatric outpatient clinics in the province of Québec, as well as from advocacy and support groups. Control families, where neither parent had a lifetime diagnosis of a major affective disorder or any current Axis I diagnosis, were recruited from the same geographic locations via physicians' offices and community organizations. Parental diagnoses were confirmed by an experienced clinician using the Structured Clinical Interview for DSM-III-R (SCID-I; Spitzer, Williams, Gibbon, & Michael, 1992) and from an examination of psychiatric records. The Structured Clinical Interview for DSM-III-R Axis II personality disorders (SCID-II; Spitzer et al., 1992) was also administered to assess symptoms of antisocial personality disorder in parents. For inclusion, families were required to be fluent in French or English, and have at least one biological child between the ages of 4 and 14 years who had been raised and educated in Canada. Families in which either parents or children presented with a chronic physical condition, physical handicap, or IQ below 70 were excluded from the study.

Assessments were made at two time points, approximately 12 years apart (11.7 ± 1.0, range = 10–14). The initial sample was recruited between 1996 and 1998, and included 145 (76 female) offspring between the ages of 4 and 13 years ($M = 7.89$, $SD = 2.41$) from 103 families (58 OBD, 45 ONAD). Parents in this sample were mostly Caucasian, middle class, and French Canadian. Detailed demographic and psychosocial information on the original sample is described in Ellenbogen and Hodgins (2004). One hundred and one (52 female) offspring between the ages of 15 and 21 years ($M = 19.71$, $SD = 2.50$) from 74 families (42 OBD, 32 ONAD) returned for assessment at least 10 years later, consisting of 70% of the original sample. Twenty-nine offspring (19 OBD, 10 ONAD) met DSM-IV (American Psychiatric Association, 1994) criteria for a *current* diagnosis, including an affective disorder (2 OBD, 0 ONAD), anxiety disorder (15 OBD, 9 ONAD), substance use disorder (6 OBD, 2 ONAD), and 4 other diagnoses (1 OBD with hypochondriasis, 1 OBD with ADHD, 1 OBD with conduct disorder, and 1 ONAD with Tourette syndrome). Seventeen OBD (33%) and 5 (10%) ONAD had a *lifetime* diagnoses of an affective disorder. No differences were observed between the original sample and those who dropped out 12 years later with regard to offspring problem behavior and IQ, as well as parents' scores across three dimensions of parenting (all $ps > .05$).

Measures

Time 1 offspring aged 4–13 years. Parents completed the Parenting Dimensions Inventory (PDI; Slater & Power, 1987) as a measure of levels of (a) support (i.e., parental warmth, nurturance, and emotional expressiveness; "My child and I have warm, close moments together"), (b) structure (i.e., organization, consistency, and predictability; "Once I decide how to deal with a misbehavior of my child, I follow through on it"), and (c) control (i.e., frequency and type of disciplinary strategies; "I do not allow my child to get angry with me") in the home. Parents endorsed items on a scale ranging from 1 (*not at all characteristic of me*) to 6 (*very characteristic of me*). Scores for each subscale of the PDI were mean ratings across all parents within a family. In the current sample, the PDI showed adequate internal consistency ($\alpha = 0.80$).

The Achenbach System of Empirically Based Assessment (Achenbach & Rescorla, 2001) is designed to assess children's internalizing and externalizing difficulties across eight dimensions of functioning at home and in school. For the purposes of this study, both the parent-reported Child Behavior Checklist (CBCL) and the Teacher Report Form (TRF) were administered. Composite scores were derived from three subscales (i.e., anxious/depressed, withdrawn/depressed, and somatic complaints) and two subscales (i.e., rule-breaking behavior and aggressive behavior) to assess internalizing and externalizing symptoms in youth, respectively. CBCL scores were averaged across all parents. The TRF was obtained for a subsample of children ($n = 105$; 55 OBD, 50 controls). The Achenbach System of Empirically Based

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Assessment shows adequate test–retest reliability ($\kappa = 0.64$–0.95) and high internal consistencies ($\alpha = 0.90$; Achenbach & Rescorla, 2001). Concurrent validity has also been established between the CBCL and other parent-reported behavior scales and diagnostic interviews for children (Barkley, 1998).

The Child Assessment Schedule (CAS; Hodges, 1981; Hodges, Mcknew, Cytryn, Stern, & Kline, 1982) is a semi-structured, diagnostic interview that was used by trained clinicians to assess for the number of psychiatric symptoms in offspring based on DSM-III (American Psychiatric Association, 1980) criteria. A total score representing the number of current symptoms that met the threshold for clinical significance across all affective, anxious, and disruptive behavior disorders was created. Only parent-reported symptoms were used for the purposes of this study. There is substantial evidence of interrater reliability and internal consistency for the CAS (Hodges, 1981; Hodges et al., 1982). Diagnostic agreement between child and parent informants has also been established (Verhulst, Althaus, & Berden, 1987).

Time 2 offspring aged 15–21 years. The SCID-I (First et al., 2002) and Kiddie Schedule for Affective Disorders and Schizophrenia—Present and Lifetime version (K-SADS-PL; Kaufman & Schweder, 2004) were used to assess for the number of current (past month) symptoms of mental disorders in adult and adolescent offspring, respectively. Interviews were conducted by experienced clinicians trained and supervised in the use of the official French and English versions of the SCID-I or K-SADS-PL. In the present study, outcome at the follow-up was defined by the number of symptoms of major depressive disorder, anxiety disorders (generalized anxiety disorder, obsessive–compulsive disorder, social and specific phobia, and posttraumatic stress disorder), and substance use disorders recorded. Given the low diagnosis rate in the current sample (see participant description above), the number of symptoms reaching the subthreshold or threshold for clinical significance was tallied for each category. Thus, the final tally represents a count of only those symptoms that were deemed "severe" enough to be meaningful clinically, with higher numbers indicating a greater number of symptoms. Both diagnostic instruments demonstrate adequate psychometric properties (Basco et al., 2000; First et al., 2002; Kaufman & Schweder, 2004). Interrater reliability obtained for 15% of interviews in the current sample was excellent ($\kappa = 0.82$).

Procedure

Following a telephone screening, parents with BD were administered the SCID-I and SCID-II interviews in the laboratory or at their homes, as well as a number of questionnaires. Parents with BD were euthymic during testing. Current spouses and ex-partners were also contacted and requested to complete the same interviews and questionnaires. Next, each parent independently completed the PDI and CBCL, and one parent from each family underwent the CAS interview for each of their children. Finally, parents provided consent and contact information for their child's teacher to complete the TRF. Control parents underwent the same procedures as families with a parent having BD.

Approximately 12 years later, parents were approached by telephone to provide consent for their adolescent and adult offspring to be contacted by study personnel. Offspring were then scheduled to return to the laboratory to undergo a diagnostic assessment, complete a battery of questionnaires (Nijjar et al., 2014) and information processing tasks, as well as partake in three days of saliva collection in their natural environment (Ostiguy, Ellenbogen, Walker, Walker, & Hodgins, 2011). Only the diagnostic data is used in the present manuscript. Informed written consent was obtained from parents at Time 1 and from both parents and offspring at Time 2. Offspring participants received an honorarium of $150 CAN at Time 2 for participating in the full data collection. All procedures at Time 1 and Time 2 were approved by the Ethics Committee of the Université de Montréal and the Human Research Ethics Committee of Concordia University (Montréal, Canada), respectively.

Data analysis

Prior to conducting the analyses, data were screened and corrected for outliers and distributional anomalies that violated statistical assumptions. Cases with missing data at Time 2 were deleted listwise. Comparison of the OBD and ONAD on study variables are presented in Table 1.

A multivariate analysis of covariance was first conducted to examine differences in the caregiving environment of the OBD relative to the ONAD in middle childhood across mean levels of parents' support, structure, and control, controlling for offspring age and SES. Multivariate significance was determined using Wilk's λ. A statistically significant multivariate effect was followed-up by three tests of univariate analysis of covariance conducted on each dependent variable.

Two overarching, parallel multiple mediation models guided the main analytical procedure for this study, as represented in Figure 1. Parallel multiple mediation models the effects of a predictor on an outcome variable through two or more mediators. The output yields specific indirect effects for each mediator variable included in the statistical model, controlling for their intercorrelations. Hayes (2013) provides a SPSS macro (PROCESS version 2.15) that calculates total (Path c; strength of the relation between a predictor and outcome variable prior to accounting for the mediator effects), direct (Path c^1; strength of the relation between a predictor and outcome variable accounting for the mediator effects), and indirect (Path ab; strength of each mediating pathway) effects, as well as describes the relation between the predictor and mediator variables (Path a), and the mediator and outcome variables (Path b). PROCESS conducts tests of significance by constructing 95% bias-corrected confidence intervals. If the confidence interval does not include zero,

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Table 1. *Comparison of offspring of parents with bipolar disorder (OBD) and offspring of parents with no affective disorder (ONAD)*

	OBD	ONAD	
Time 1 (1996–1998)			
N	77	68	
Sex of offspring (female/male ratio)	37:40	39:29	
	M (*SD*)	*M* (*SD*)	*F*
Offspring age	8.38 (2.46)	7.34 (2.24)	6.98**
Parenting practices (PDI)[a]			
Support	13.98 (1.08)	14.66 (1.10)	14.16**
Structure	8.66 (1.21)	9.21 (0.71)	10.78**
Control	5.40 (0.85)	5.71 (0.88)	4.48*
Offspring CBCL symptoms[a,b]			
Internalizing	53.28 (8.34)	47.28 (8.34)	15.22**
Externalizing	52.82 (10.91)	44.98 (9.87)	20.38**
Offspring TRF symptoms[b,c]			
Internalizing	54.22 (10.91)	50.24 (9.12)	4.06*
Externalizing	52.56 (8.59)	49.06 (7.42)	4.96*
Offspring no. of CAS psychiatric symptoms[d,e]	8.08 (7.13)	3.75 (3.90)	17.12**
Time 2 (2006–2008)			
N	53	48	
Sex of offspring (female/male ratio)	24:29	25:23	
	M (*SD*)	*M* (*SD*)	*F*
Offspring age	20.09 (2.56)	18.88 (2.53)	5.92*
Offspring no. of psychiatric symptoms[f,g]			
Depressive	0.47 (1.15)	0.17 (0.64)	2.53
Anxiety	2.02 (2.77)	1.40 (2.47)	1.42
Substance use	1.72 (3.54)	0.52 (1.90)	4.33*

Note: PDI, Parenting Dimensions Inventory; CBCL, Child Behavior Checklist; TRF, Teacher Report Form; CAS, Child Assessment Schedule.
[a]A mean score across all parents within a family.
[b]A *t* score.
[c]$n = 55$ OBD, 50 controls.
[d]From the CAS parent interview.
[e]Includes clinical symptoms across all affective, anxious, and disruptive behavior disorders.
[f]From the Structured Clinical Interview for DSM-IV or Kiddie Schedule for Affective Disorders and Schizophrenia.
[g]Includes present (past month) subclinical and clinical symptoms for each disorder.
*$p < .05$. **$p < .01$.

the indirect effect is considered statistically significant at the .05 level. For this study, the bootstrap sample was set at 5,000 iterations. Finally, the output also yields partially standardized effect sizes (C_{ps}) for the indirect effects, which represent the number of standard deviations by which the dependent variable is expected to increase or decrease per a change in the mediator equal to the size of Path a.

For all mediation analyses, the following covariates, all measured at Time 1, were included: offspring age, family income, offspring psychiatric diagnoses on the CAS, and parents' antisocial traits as defined by the mean number of SCID-II threshold and subthreshold symptoms of antisocial personality disorder. To protect against violations of the assumption of independent observations, analyses were conducted with and without siblings (using random deletion). As the mediation analyses yielded similar findings, all participants were included in the final analyses. Finally, to determine if the mediated associations varied with offspring sex, the above-mentioned analyses were repeated using moderated mediation. However, offspring sex did not moderate any of the cross-sectional or longitudinal mediated analyses, and was dropped from the final statistical plan (data not shown).

Results

Group comparison of parenting practices in middle childhood

Mean levels of parent-rated support, structure, and control in middle childhood were compared between the BD and con-

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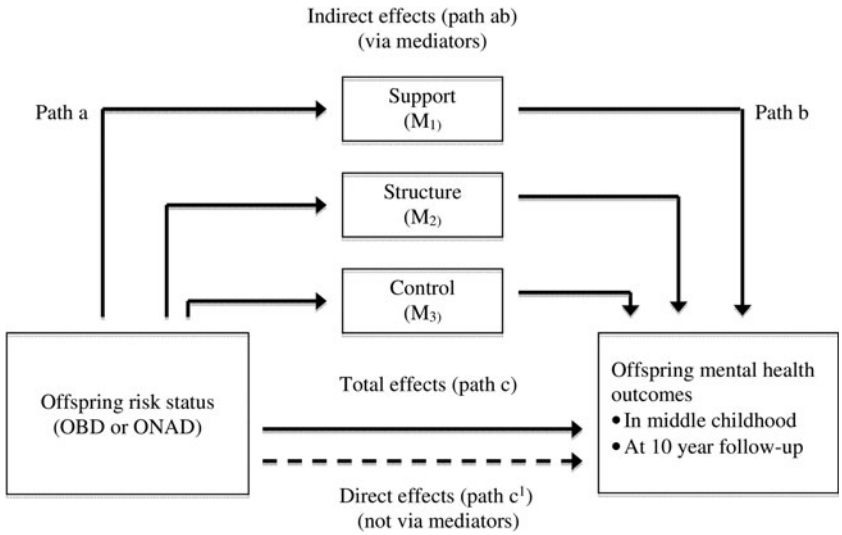

Figure 1. Multiple mediation models.

trol families. After controlling for between-group differences in SES and offspring age, a multivariate analysis of covariance revealed a main effect of group for parenting practices, F (4, 138) = 1,167.76, p = .000. Post hoc analyses indicated significantly lower levels of support, t (143) = –3.76, p = .00, d = 0.63, structure, t (143) = –3.28, p = .00, d = 0.55, and control, t (143) = –2.12, p = .04, d = 0.36, in BD relative to control families (see Table 1 for means and standard deviations).

Mediation of the relation between offspring risk status and internalizing and externalizing problems in middle childhood

Pearson correlations between independent and dependent variables are shown in Table 2. Coefficients of the associations between predictor and mediator variables (Paths a in Figure 1), and mediator and outcome variables (Paths b in Figure 1) are summarized in Table 3. Coefficients for total, direct, and indirect effects (Paths c, c^1, and ab in Figure 1) are presented in Tables 4 and 5 for the cross-sectional and longitudinal mediation analyses, respectively. The results for the indirect effects are highlighted in the text below, as a significant indirect effect is exclusively needed to establish mediation (Rucker, Preacher, Tormala, & Petty, 2011).

Mean levels of support, structure, and control provided by parents in middle childhood were tested as potential parallel mediators of the relation between offspring risk status (OBD or ONAD) and parents' report of internalizing and externalizing symptoms on the CBCL. Of the three mediators, structure showed a significant indirect effect of the relation between offspring risk status and rates of internalizing, ab = 0.97, SE = 0.56, 95% confidence interval (CI) [0.15, 2.4]; C_{ps} = 0.11, and externalizing, ab = 1.31, SE = 0.64, 95% CI [0.38, 2.88]; C_{ps} = 0.13, symptoms. Weaker, but statistically significant indirect effects on externalizing symptoms were detected for support, ab = 0.71, SE = 0.47, 95% CI [0.01, 2.0], C_{ps} = 0.07, and control, ab = 0.76, SE = 0.48, 95% CI [–1.99, –0.03]; C_{ps} = –0.08 (see Table 4). Similar to parent report, significant indirect effects of structure were detected when predicting the level of teacher-reported internalizing, ab = 1.16, SE = 0.73, 95% CI [0.11, 3.12]; C_{ps} = 0.11, and externalizing, ab = 1.1, SE = 0.68, 95% CI [0.10, 2.95]; C_{ps} = 0.13, symptoms on the TRF, as well as the total number of clinician-rated symptoms on the CAS, ab = 0.52, SE = 0.32, 95% CI [0.06, 1.38]; C_{ps} = 0.09 (see Table 4). In sum, having a parent with BD was associated with lower levels of structure in middle childhood that, in turn, predicted higher rates of parent-reported, teacher-reported, and clinician-rated internalizing and externalizing symptoms in the OBD (see Table 3). With the exception of parent-report externalizing symptoms, the indirect effects of support and control were nonsignificant (all 95% CI contained zero) across all cross-sectional analyses.

Mediation of the relation between offspring risk status and internalizing and externalizing problems in early adolescence and young adulthood

Mean levels of support, structure, and control provided by parents in middle childhood were tested as potential parallel mediators of the relation between offspring risk status and current symptoms of depressive, anxiety, and substance use disorders in late adolescence and early adulthood on the SCID-1 or K-SADS-PL. Of the three mediators, control in middle childhood mediated the association between offspring risk status and the number of symptoms of depressive, ab = 0.16, SE = 0.09, 95% CI [0.02, 0.43]; C_{ps} = 0.14, and substance use, ab = 0.70, SE = 0.36, 95% CI [0.19, 1.67]; C_{ps} = 0.24, disorders 12 years later. Although weaker than for control, the indirect effect of structure on present symptoms of depression was also statistically significant, ab = –0.17,

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Table 2. *Pearson correlation coefficients for study variables*

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Offspring risk status	—	–.30**	–.27**	–.17*	.31**	.35**	.20*	.21*	.35**	.13	.12	.21*
2. PDI support		—	.27**	–.20*	–.19*	–.32**	–.08	–0.12	–.19*	.03	.02	–.10
3. PDI structure			—	.21**	–.35**	–40**	–.26**	–.29**	–.29**	.19	–.06	–.13
4. PDI control				—	–.06	.05	–.09	–.05	–.08	–.09	–.10	–.36**
Offspring outcomes at Time 1												
5. CBCL internalizing symptoms					—	.69**	.49**	.22*	.61**	.03	.06	.06
6. CBCL externalizing symptoms						—	.36**	.50**	.60**	.13	.12	.19
7. TRF internalizing symptoms							—	.43**	.33**	–.14	.09	.24*
8. TRF externalizing symptoms								—	.36**	–.06	.13	.20
9. No. of CAS psychiatric symptoms									—	.18	.04	.03
Offspring outcomes at Time 2												
10. No. of depressive symptoms										—	.12	.10
11. No. of anxiety symptoms											—	.18
12. No. of substance use symptoms												—

Note: PDI, Parenting Dimensions Inventory; CBCL, Child Behavior Checklist; TRF, Teacher Report Form; CAS, Child Assessment Schedule.
*$p < .05$. **$p < .01$.

$SE = 0.09$, 95% CI [–0.44, –0.04]; $C_{ps} = –0.03$ (see Table 5). Similar to the cross-sectional analyses of parenting and offspring symptoms in middle childhood, low levels of control in middle childhood predicted higher rates of offspring depressive and substance use symptoms in late adolescence and young adulthood. Conversely, high levels of structure in middle childhood were associated with higher rates of depressive symptoms (see Table 3). Across all remaining longitudinal analyses, the indirect effects of structure and support were nonsignificant (all 95% CI contained 0).

Discussion

The present prospective study showed that parenting practices in middle childhood mediated the associations between having a parent with or without BD, and internalizing and externalizing symptoms in offspring in middle childhood and approximately 12 years later. As expected, parents with BD displayed significant impairments in parenting practices relative to control parents, providing less support, structure, and control to their offspring in middle childhood. This is consistent with previous studies of parenting by adults with an affective disorder (Alloy, Abramson, Smith, Gibb, & Neeren, 2006; Dix & Meunier, 2009; Radke-Yarrow, 1998; Wilson & Durbin, 2010). During middle childhood, parental structure, to a greater extent than support or control, underlay the relation between parents' diagnosis of BD and a range of internalizing and externalizing difficulties in their offspring. Specifically, parents with BD were more likely than healthy control parents to provide low levels of organization, consistency, and stability in the home during middle childhood that, in turn, predicted higher rates of psychopathology in the OBD. These associations remained significant for parent-report, teacher-report, and clinician ratings of offspring psychopathology. In contrast, parental control in middle childhood emerged as the strongest predictor of offspring psychopathology in late adolescence and young adulthood. That is, parents' inability to ensure adequate supervision and role boundaries, as well as set appropriate expectations, limits, and consequences for child misbehavior during middle childhood, mediated the association between having a parent with BD and symptoms of substance use and depressive disorders approximately 12 years later.

The results of the mediation analyses are consistent with the view that poor parenting practices in families that include a parent with BD represent a putative causal mechanism for internalizing and externalizing problems in the OBD (Alloy et al., 2005, 2006; Ellenbogen & Hodgins, 2004; Meyer et al., 2006). The present findings extend previous findings from this sample. We showed that levels of neuroticism were elevated among parents with BD and associated with a range of nonoptimal parenting practices including low warmth, low autonomy support, and ineffective behavioral control, as well as a less organized and consistent parenting style (Ellenbogen & Hodgins, 2004). Parents' traits of neuroticism and agreeableness were robust predictors of internaliz-

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Table 3. *Unstandardized coefficients between independent and mediator variables (Path a), and mediator and dependent variables (Path b) for the cross-sectional and longitudinal mediation models*

	Mediator Variables		
	Support	Structure	Control
Independent[a] → Mediator (Path a)			
For Cross-Sectional Analyses	β (*SE*)	β (*SE*)	β (*SE*)
Predicting CBCL internalizing symptoms	−0.58** (0.19)	−0.48** (0.18)	−0.41** (0.14)
Predicting CBCL externalizing symptoms	−0.58** (0.19)	−0.48** (0.18)	−0.41** (0.14)
Predicting TRF internalizing symptoms	−0.62* (0.24)	−0.65** (0.22)	−0.30 (0.18)
Predicting TRF externalizing symptoms	−0.62* (0.24)	−0.65** (0.22)	−0.30 (0.18)
Predicting no. of CAS psychiatric symptoms[b]	−0.59** (0.19)	−0.51** (0.18)	−0.42** (0.14)
For Longitudinal Analyses			
Predicting no. of psychiatric symptoms[c,d]			
Depression	−0.54** (0.20)	−0.49* (0.21)	−0.51** (0.15)
Anxiety	−0.55** (0.20)	−0.49* (0.21)	−0.50** (0.15)
Substance Use	−0.55** (0.20)	−0.49* (0.21)	−0.50** (0.15)
Mediator → Dependent (Path b)			
For Cross-Sectional Analyses			
Predicting CBCL internalizing symptoms	−0.01 (0.73)	−2.00** (0.75)	0.37 (0.94)
Predicting CBCL externalizing symptoms	−1.22 (0.80)	−2.72** (0.82)	1.82 (1.03)
Predicting TRF internalizing symptoms	−0.04 (0.95)	−1.80 (0.99)	−0.49 (1.26)
Predicting TRF externalizing symptoms	−0.12 (0.75)	−1.64* (0.78)	0.39 (0.99)
Predicting no. of CAS psychiatric symptoms[b]	−0.57 (0.49)	−1.02* (0.50)	−0.28 (0.64)
For Longitudinal Analyses			
Predicting no. of psychiatric symptoms[c,d]			
Depression	−0.03 (0.13)	0.32** (0.12)	−0.30 (0.17)
Anxiety	0.30 (0.31)	−0.05 (0.27)	−0.00 (0.40)
Substance use	−0.05 (32)	−0.06 (0.28)	−1.40** (0.42)

Note: CBCL, Child Behavior Checklist; TRF, Teacher Report Form; CAS, Child's Assessment Schedule.
[a]Across all mediation models, the independent variable is offspring risk status (having a parent with bipolar disorder or not).
[b]Includes clinical symptoms across all affective, anxious, and disruptive behavior disorders.
[c]From the Structured Clinical Interview for DSM-IV or Kiddie Schedule for Affective Disorders and Schizophrenia.
[d]Includes present (past month) subclinical and clinical symptoms for each disorder.
*$p < .05$. **$p < .01$.

ing and externalizing problems among their offspring in middle childhood (Nijjar et al., 2016; Ostiguy et al., 2012). These externalizing problems, in turn, place the OBD on trajectories leading to a variety of difficulties in adolescence and young adulthood, such as poor interpersonal functioning and high-risk sexual behaviors. Further, we found that the OBD were more sensitive to the deleterious effects of their parents' neuroticism and its associated environmental consequences than the ONAD (Nijjar et al., 2016; Ostiguy et al., 2012), further highlighting the important role of the home environment in preventing negative outcomes among the OBD.

It is interesting that the type of parenting practice that was an important predictor of behavioral outcomes differed depending on whether the outcome of interest was assessed concurrently in middle childhood or 12 years later when offspring were in late adolescence and young adulthood. There is evidence that a child's susceptibility to specific disruptions in caregiving depends on their age at exposure (Frick, Christian, & Wootton, 1999). Parenting practices that offer an organized, predictable, and consistent framework for daily living have been primarily associated with enhanced psychological well-being and psychosocial development among younger children. For instance, the use of child and family routines provide a buffer against hyperactivity, impulsivity, and noncompliance in low-income, ethnic minority children (Lanza & Drabick, 2011), and internalizing problems in school-aged children (Bridley & Jordan, 2012). Likewise, home environments characterized by high levels of disorganization, chaos, and noise have been associated with a range of internalizing, externalizing, and attention problems in preschool and

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Table 4. *Unstandardized coefficients for the total, direct, and indirect effects of offspring risk status on internalizing, externalizing, and total psychiatric symptoms at baseline (cross-sectional analyses) via mean levels of parents' support, structure, and control in middle childhood*

Parent Report (n = 145)				
Effect	β	*SE*	*p*	
Total				
Predicting CBCL internalizing symptoms	4.06	1.51	.01**	
Predicting CBCL externalizing symptoms	3.39	1.66	.00**	
Predicting no. of CAS psychiatric symptoms	3.90	1.02	.00**	
Direct				
Predicting CBCL internalizing symptoms	3.23	1.62	.05*	
Predicting CBCL externalizing symptoms	2.37	1.78	.01**	
Predicting no. of CAS psychiatric symptoms	2.93	1.10	.01**	
Indirect (Via Mediators)	β	*SE*	95% CI	C_{ps}
Predicting CBCL internalizing symptoms				
Support	0.01	0.42	−0.83, 0.91	0.00
Structure	0.97	0.56	0.15, 2.38*	0.11
Control	−0.15	0.41	−1.09, 0.58	−0.02
Predicting CBCL externalizing symptoms				
Support	0.71	0.47	0.01, 1.98*	0.07
Structure	1.31	0.64	0.38, 2.88*	0.13
Control	−0.76	0.48	−1.99, –0.03*	−0.08
Predicting no. of CAS psychiatric symptoms				
Support	0.34	0.30	−0.13, 1.12	0.06
Structure	0.52	0.32	0.06, 1.38*	0.09
Control	0.12	0.27	−0.30, 0.83	0.02
Teacher Report (n = 105)				
Effect	β	*SE*	*p*	
Total				
Predicting TRF internalizing symptoms	3.60	2.10	.09	
Predicting TRF externalizing symptoms	3.39	1.66	.04*	
Direct				
Predicting TRF internalizing symptoms	2.27	2.27	.32	
Predicting TRF externalizing symptoms	2.37	1.78	.19	
Indirect (Via Mediators)	β	*SE*	95% CI	C_{ps}
Predicting TRF internalizing symptoms				
Support	0.03	0.62	−1.09, 1.43	0.00
Structure	1.16	0.73	0.11, 3.12*	0.11
Control	0.15	0.45	−0.47, 1.49	0.01
Predicting TRF externalizing symptoms				
Support	0.07	0.54	−0.94, 1.29	0.01
Structure	1.07	0.68	0.10, 2.95*	0.13
Control	−0.12	0.38	−1.25, 0.40	−0.01

Note: CBCL, Child Behavior Checklist; CAS, Child Assessment Schedule; TRF, Teacher Report Form; C_{ps}, partially standardized effect size.
*$p < .05$. **$p < .01$.

school-aged children (Coldwell, Pike, & Dunn, 2006; Deater-Deckard et al., 2009). Among adolescent populations, low levels of parental behavioral control, including inadequate regulation, monitoring, and supervision of adolescent activities, have been repeatedly linked to increased delinquency and engagement in high-risk behaviors (DeVore & Ginsburg, 2005; Hoeve et al., 2009), greater internalizing problems and steeper trajectories toward externalizing difficulties (Galambos, Barker, & Almeida, 2003), as well as higher rates of antisocial behaviors and depression (Bacchini, Miranda, & Affuso, 2011).

In the present study, while a caregiving environment low in parental structure had an immediate impact on the OBD's men-

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Table 5. *Unstandardized coefficients for the total, direct, and indirect effects of offspring risk status on symptoms of depressive, anxiety, and substance use disorders[a,b] in late adolescence and early adulthood (longitudinal analyses) via mean levels of parents' support, structure, and control in middle childhood*

Effect	β	*SE*	*p*	
Total				
Predicting depressive symptoms	0.31	0.24	.21	
Predicting anxiety symptoms	0.56	0.53	.29	
Predicting substance use symptoms	1.30	0.59	.03*	
Direct				
Predicting depressive symptoms	0.29	0.27	.28	
Predicting anxiety symptoms	0.70	0.62	.26	
Predicting substance use symptoms	0.29	0.65	.65	
Indirect (Via Mediators)	β	*SE*	95% CI	C_{ps}
Predicting depressive symptoms				
Support	0.03	0.06	−0.07, 0.19	0.03
Structure	−0.17	0.09	−0.44, –0.04*	−0.03
Control	0.16	0.09	0.02, 0.43*	0.33
Predicting anxiety symptoms				
Support	−0.17	0.24	−0.75, 0.25	−0.06
Structure	0.03	0.21	−0.33, 0.55	0.01
Control	0.00	0.28	−0.53, 0.61	0.00
Predicting substance use symptoms				
Support	0.28	0.24	−0.04, 0.98	0.09
Structure	0.03	0.18	−0.26, 0.49	0.01
Control	0.70	0.36	0.19, 1.67*	0.24

Note: $n = 101$; C_{ps}, partially standardized effect size.
[a]From the Structured Clinical Interview for DSM-IV or Kiddie Schedule for Affective Disorders and Schizophrenia.
[b]Includes present (past month) subclinical and clinical symptoms for each disorder.
*$p < .05$.

tal health, the adverse psychological effects of insufficient parental control during middle childhood remained latent until the OBD reached late adolescence and early adulthood. The enduring effects of repeated exposure to suboptimal caregiving environments on offspring's psychological well-being have been well documented (e.g., Hoeve et al., 2009; Morgan, Brugha, Fryers, & Stewart-Brown, 2012; Weich, Patterson, Shaw, & Stewart-Brown, 2009). The current data extend previous findings by suggesting that the impact of early negative caregiving not only persists but also may not be apparent until much later in the course of offspring development. Alternatively, it is possible that the longitudinal findings reflect consistency in parenting practices from childhood into late adolescence. Although not measured in the current study, parenting philosophies and practices tend to persist over time (Carrasco, Rodriguez, Del Barrio, & Holgado, 2011; Else-Quest, Clark, & Tresch Owen, 2011). This may be especially characteristic of the childrearing practices adopted by parents with BD, as behavioral and cognitive rigidity is a feature common to many psychopathologies (Schultz & Searleman, 2002). Thus, the OBD who were exposed to low levels of parental control in middle childhood may have continued to experience inadequate parental control as they grew older that, in turn, contributed to the development of substance use and depressive symptoms.

Although parents with BD provided less support to their offspring than control parents, parental support in middle childhood did not emerge as a central pathway through which BD in a parent led to offspring internalizing and externalizing symptoms in either middle childhood or late adolescence and early adulthood. The finding that parental support failed to independently contribute to internalizing problems in the OBD was especially unexpected, as low parental warmth has been repeatedly associated with depression and anxiety among both general and high-risk pediatric populations (for meta-analyses, see McLeod, Weisz, & Wood, 2007; and McLeod, Wood, & Weisz, 2007). This pattern of findings suggests that the OBD may be more susceptible to the adverse effects of disrupted parental structure and control than support. For example, when compared to the ONAD, the OBD display dysregulations in the functioning of the hypothalamus–pituitary–adrenal axis, a neuroendocrine system responsible for the secretion of cortisol in response to environmental stressors (Ostiguy et al., 2011). In turn, hypothalamus–pituitary–adrenal axis dysfunction may represent a biological marker of vulnerability to psychopathology in the OBD (Ellenbogen et al., 2011) that, in one study, was strongly predicted by disruptions in predictable and consistent caregiving 10 years earlier, but not supportive parenting (Ellenbogen & Hodgins, 2009).

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Thus, the robust effects of parenting structure, rather than support, on mental health in the OBD highlights how the impact of different parenting factors on risk for mental illness may be partly determined by the nature of the vulnerabilities inherent to the population of high-risk youth being studied. It is worth noting that this finding stands in contrast to results reported by Meyer et al. (2006), who found that negative affect and attitudes in mothers with BD during early childhood were associated with greater odds of their offspring developing BD in early adulthood. However, important differences exist with regard to the parenting constructs (i.e., parental support vs. maternal negativity) and mental health outcomes (internalizing and externalizing problems vs. BD) assessed in each study, suggesting that the two studies are far too different to be compared with one another.

Likewise, parenting practices in middle childhood did not predict symptoms of anxiety in late adolescence and early adulthood, despite anxiety being among the most common psychiatric disorder developed by the OBD. This is in line with the findings from a recent systematic review (Wood, McLeod, Sigman, Hwang, & Chu, 2003) and meta-analysis (Yap & Jorm, 2015) demonstrating little evidence for an association between parenting styles and pediatric anxiety. McLeod, Weisz, et al. (2007) and Mcleod, Wood, et al. (2007) conducted two separate meta-analyses into the role of parenting in determining child and adolescent internalizing difficulties. In aggregate, parenting accounted for less than 4% of the total variance in offspring anxiety relative to 8% with regard to depression. This suggests that factors other than parenting may be especially important to consider in the pathogenesis of pediatric anxiety disorders, which may account for the present null result.

Insufficient parental control is particularly salient in the context of families that include a parent having BD and may have important implications for models of abnormal development among the OBD. Externalizing problems are elevated in the OBD compared to age-matched controls (Klimes-Dougan, Ronsaville, Wiggs, & Martinez, 2006; Linnen, aan het Rot, Ellenbogen, & Young, 2009). Moreover, among high-risk offspring who develop BD, there is evidence of a subgroup characterized by antisocial behavior during childhood and adolescence who exhibit a more severe course of BD, including more hospitalizations and episodes, and a higher prevalence of psychotic symptoms during manic episodes (Carlson, Bromet, & Sievers, 2000; Carlson & Weintraub, 1993). Similarly, OBD who exhibit high levels of quarrelsome behavior in their daily social interactions, relative to those with low levels and to the ONAD, show blunted daytime cortisol levels (Ellenbogen et al., 2011), a frequent biological correlate of antisocial behavior (Alink et al., 2008). Externalizing problems, therefore, represent a core feature of premorbid risk among the OBD. As such, parenting behaviors, such as effective disciplinary strategies, that decrease externalizing behaviors in childhood, may have beneficial long-term effects for the OBD. In addition, the caregiving environment, specifically the relation between parent and child, interacts with specific genetic variants to modify the risk of delinquency (Nilsson et al., 2015) and depression (Comasco, Aslund, Oreland, & Nilsson, 2013).

It was unexpected that high levels of structure in middle childhood were significantly related to elevated rates of depressive symptoms in offspring 12 years later. Although a small statistical effect relative to the other reported findings, the direction of the mediating effect was contrary to the cross-sectional results, where high levels of structure were associated with fewer symptoms. Within community samples, a lack of age-appropriate autonomy granting and enmeshed family interactions have been associated with increased risk for adolescent depression (Jewell & Stark, 2003; Noom, Dekovic, & Meeus, 1999; Yap et al., 2014), both of which could conceivably be a consequence of excessive parental structuring in later developmental stages. In the current study, this finding may be best understood within the context of co-occurring low levels of parental control, which also significantly predicted elevated symptoms of depression 12 years later. Namely, the establishment of rules, schedules, and order in the absence of parental monitoring, limit setting, and consequences may have created erratic, ambiguous, and confusing environments that further contributed to depressive symptomology in the OBD. Possible interpretations remain limited, however, as there are few studies to guide our understanding of how high parental structure might have a unique impact within the context of growing up with a parent with BD and a heightened familial risk for adolescent depression. Additional research is needed of combinations of the different aspects of parenting and outcomes for the OBD.

Strengths and limitations

To the best of our knowledge, this is the first study to investigate the mediating role of parenting practices in middle childhood on the mental health outcomes of the OBD measured concurrently and approximately 12 years. Thus, these data increase understanding of a putative mechanism that may be causally related to externalizing and internalizing symptoms among the OBD. Offspring psychopathology was independently rated by parents, teachers, and experienced clinicians. Three dimensions of parenting practices were examined simultaneously, allowing for comparisons of relative strength in predicting outcomes in the OBD. Another strength of the study was the estimations of associations of parenting and psychopathology in late adolescence and early adulthood while statistically controlling for offspring psychopathology in middle childhood. This reduced the likelihood that symptoms of depression and substance use disorders in late adolescence and early adulthood simply represented continuity from internalizing and externalizing problems observed in middle childhood, further supporting our finding of the enduring association between parenting practices and offspring psychopathology.

A number of study limitations should be considered when interpreting the results. Parenting practices were only mea-

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sured when offspring were in middle childhood, limiting knowledge of parenting into late adolescence and early adulthood when the follow-up assessment of offspring mental health was undertaken. In particular, the absence of parenting measures during adolescence precludes the possibility of assessing how changing parenting practices over time may have influenced outcomes. Another limitation is the large age range of the participants. While parenting was measured during the developmental stage that is commonly described as middle childhood, certain practices will have a differential impact on offspring adjustment based on age (Frick et al., 1999). Parenting practices were assessed using a single self-report measure, which could have been influenced by parents' mental health status (De Los Reyes & Kazdin, 2005). However, parents with BD were in a euthymic state when completing the PDI. The use of an average score that represented levels of support, structure, or control across all parents within a family likely further helped reduce bias and error commonly associated with the use of self-report data derived from a single informant (Kroes, Veerman, & De Bruyn, 2003). However, this approach prevented us from gaining a more comprehensive understanding of the ways in which the specific parenting practices adopted by each caregiver may have interacted to determine psychiatric outcomes in the OBD. There is a growing area of research attesting to the differential impact of mothers' and fathers' parenting strategies on offspring clinical outcomes (McKinney & Renk, 2008; Milevsky, Schlechter, Netter, & Keehn, 2007) and that exposure to competent caregiving from at least one parent provides a buffer against emotional maladjustment in adolescents (Simons & Conger, 2007). Given the elevated rates of assortative mating among patients with BD (Mathews & Reus, 2001), the implications with regard to parenting competence may be especially disadvantageous for the OBD. The unique contribution of parental support to offspring mental health may have been more apparent if considered in interaction with, rather than simultaneously to, the other components of parenting. For example, an authoritative parenting style, which combines parenting practices designed to optimize levels of both parental responsiveness and demandingness, has been consistently linked to psychological well-being in youth (Larzelere, Morris, & Harrist, 2013; Piko & Balazs, 2012). Ultimately, the use of both multiple informants and observation to assess parenting practices should be considered in future studies.

Conclusions

Low levels of structure provided by parents in middle childhood mediated the relation between having a parent with BD and elevated rates of internalizing and externalizing difficulties in the OBD during middle childhood. For the longitudinal outcomes, parental control in middle childhood emerged as the strongest mediator of the relation between having a parent with BD and offspring psychopathology 12 years later, in late adolescence and early adulthood. These findings support the usefulness of parent training prevention programs targeting the caregiving environment to reduce risk of psychopathology in the OBD. Specifically, the present findings not only emphasize the promotion of parental monitoring and adaptive disciplinary practices as a means of mental illness prevention but also highlight the importance of addressing issues of predictability, consistency, and organization in the home environment of the OBD. This is consistent with current trends in the treatment of adult and pediatric BD (Miklowitz, 2010). Further dismantling of parenting constructs is needed to help clarify some of the discrepant and unexpected findings that emerged in the current study. More multimethod, longitudinal research in populations at risk for affective disorders would also be of benefit.

References

Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the ASEBA School-Age Forms & Profiles*. Burlington, VT: University of Vermont, Department of Psychiatry.

Alink, L. R., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Mesman, J., Juffer, F., & Koot, H. M. (2008). Cortisol and externalizing behavior in children and adolescents: Mixed meta-analytic evidence for the inverse relation of basal cortisol and cortisol reactivity with externalizing behavior. *Developmental Psychobiology*, *50*, 427–450.

Alloy, L. B., Abramson, L. Y., Smith, J. M., Gibb, B. E., & Neeren, A. M. (2006). Role of parenting and maltreatment histories in unipolar and bipolar mood disorders: Mediation by cognitive vulnerability to depression. *Clinical Child and Family Psychology Review*, *9*, 23–64. doi:10.1007/s10567-006-0002-4

Alloy, L. B., Abramson, L. Y., Urosevic, S., Walshaw, P. D., Nusslock, R., & Neeren, A. M. (2005). The psychosocial context of bipolar disorder: Environmental, cognitive, and developmental risk factors. *Clinical Psychology Review*, *25*, 1043–1075. doi:10.1016/j.cpr.2005.06.006

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Angold, A., Erkanli, A., Silberg, J., Eaves, L., & Costello, E. J. (2002). Depression scale scores in 8–17-year-olds: Effects of age and gender. *Journal of Child Psychology and Psychiatry*, *43*, 1052–1063. doi:10.1111/1469-7610.00232

Bacchini, D., Miranda, M. C., & Affuso, G. (2011). Effects of parental monitoring and exposure to community violence on antisocial behavior and anxiety/depression among adolescents. *Journal of Interpersonal Violence*, *26*, 269–292. doi:10.1177/0886260510362879

Barkley, R. A. (1988). Child behavior rating scales and checklists. In M. Rutter & A. H. Tuma (Eds.), *Assessment and diagnosis in child psychopathology* (pp. 113–155). New York: Guilford Press.

Barron, E., Sharma, A., Le, C. J., Rushton, S., Close, A., Kelly, T., . . . Le, C. A. (2014). Family environment of bipolar families: A UK study. *Journal of Affective Disorders*, *152–154*, 522–525. doi:10.1016/j.jad.2013.08.016

Basco, M. R., Bostic, J. Q., Davies, D., Rush, A. J., Witte, B., Hendrickse, W., & Barnett, V. (2000). Methods to improve diagnostic accuracy in a community mental health setting. *American Journal of Psychiatry*, *157*, 1599–1605.

Beardslee, W. R., Gladstone, T. R., & O'Connor, E. E. (2011). Transmission and prevention of mood disorders among children of affectively ill parents: A review. *Journal of the American Academy of Child & Adolescent Psychiatry*, *50*, 1098–1109. doi:10.1016/j.jaac.2011.07.020

Begley, C. E., Annegers, J. F., Swann, A. C., Lewis, C., Coan, S., Schnapp, W. B., & Bryant-Comstock, L. (2001). The lifetime cost of bipolar disorder in the US: An estimate for new cases in 1998. *Pharmacoeconomics*, *19*, 483–495.

https://doi.org/10.1017/S095457941700116X Published online by Cambridge University Press

Birmaher, B., Axelson, D., Monk, K., Kalas, C., Goldstein, B., Hickey, M. B., . . . Brent, D. (2009). Lifetime psychiatric disorders in school-aged offspring of parents with bipolar disorder. *Archives of General Psychiatry*, *66*, 287–296. doi:10.1001/archgenpsychiatry.2008.546

Bradley, R. H., & Corwyn, R. F. (2002). Socioeconomic status and child development. *Annual Review of Psychology*, *53*, 371–399. doi:0084-6570/02/0201-0371$14.00

Bridley, A., & Jordan, S. S. (2012). Child routines moderate daily hassles and children's psychological adjustment. *Children's Health Care*, *41*, 129–144. doi:10.1080/02739615.2012.657040

Brietzke, E., Mansur, R. B., Soczynska, J. K., Kapczinski, F., Bressan, R. A., & McIntyre, R. S. (2012). Towards a multifactorial approach for prediction of bipolar disorder in at risk populations. *Journal of Affective Disorders*, *140*, 82–91. doi:10.1016/j.jad.2012.02.016

Broidy, L. M., Nagin, D. S., Tremblay, R. E., Bates, J. E., Brame, B., Dodge, K. A., . . . Vitaro, F. (2003). Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: A six-site, cross-national study. *Developmental Psychology*, *39*, 222–245. doi:10.1037//0012-1649.39.2.222

Calam, R., Jones, S., Sanders, M. R., Dempsey, R., & Sadhnani, V. (2012). Parenting and the emotional and behavioural adjustment of young children in families with a parent with bipolar disorder. *Behavioural and Cognitive Psychotherapy*, *40*, 425–437. doi:10.1017/S1352465812000094

Carlson, G. A., Bromet, E. J., & Sievers, S. (2000). Phenomenology and outcome of subjects with early- and adult-onset psychotic mania. *American Journal of Psychiatry*, *157*, 213–219.

Carlson, G. A., & Weintraub, S. (1993). Childhood behavior problems and bipolar disorder—Relationship or coincidence? *Journal of Affective Disorders*, *28*, 143–153.

Carrasco, M. A., Rodriguez, M. A., Del Barrio, M. V., & Holgado, F. P. (2011). Relative and absolute stability in perceived parenting behaviour: A longitudinal study with children and adolescents. *Psychological Reports*, *108*, 149–166. doi:10.2466/02.09.10.21.PR0.108.1.149-166

Chang, K. D., Blasey, C., Ketter, T. A., & Steiner, H. (2001). Family environment of children and adolescents with bipolar parents. *Bipolar Disorders*, *3*, 73–78. doi:10.1034/j.13995618.2001.030205.x

Coldwell, J., Pike, A., & Dunn, J. (2006). Household chaos—Links with parenting and child behaviour. *Journal of Child Psychology and Psychiatry*, *47*, 1116–1122. doi:10.1111/j.1469-7610.2006.01655.x

Comasco, E., Åslund, C., Oreland, L., & Nilsson, K. W. (2013). Three-way interaction effect of 5-HTTLPR, BDNF Val66Met, and childhood adversity on depression: A replication study. *European Neuropsychopharmacology*, *23*, 1300–1306. doi:10.1016/j.euroneuro.2013.01.010

Das Gupta, R., & Guest, J. F. (2002). Annual cost of bipolar disorder to UK society. *British Journal of Psychiatry*, *180*, 227–233. doi:10.1192/bjp.180.3.227

Deater-Deckard, K., Mullineaux, P. Y., Beekman, C., Petrill, S. A., Schatschneider, C., & Thompson, L. A. (2009). Conduct problems, IQ, and household chaos: A longitudinal multi-informant study. *Journal of Child Psychology and Psychiatry*, *50*, 1301–1308. doi:10.1111/j.1469-7610.2009.02108.x

De Los Reyes, A., & Kazdin, A. E. (2005). Informant discrepancies in the assessment of childhood psychopathology: A critical review, theoretical framework, and recommendations for further study. *Psychological Bulletin*, *131*, 483. doi:10.1037/0033-2909.131.4.483

DeMulder, E. K., & Radke-Yarrow, M. (1991). Attachment with affectively ill and well mothers: Concurrent behavioral correlates. *Development and Psychopathology*, *3*, 227–242. doi:10.1017/S0954579400005277

DeVore, E. R., & Ginsburg, K. R. (2005). The protective effects of good parenting on adolescents. *Current Opinion in Pediatrics*, *17*, 460–465.

Dix, T., & Meunier, L. N. (2009). Depressive symptoms and parenting competence: An analysis of 13 regulatory processes. *Developmental Review*, *29*, 45–68. doi:10.1016/j.dr.2008.11.002

Dore, G., & Romans, S. E. (2001). Impact of bipolar affective disorder on family and partners. *Journal of Affective Disorders*, *67*, 147–158. doi:10.1016/S0165-0327(01)00450-5

Duffy, A., Alda, M., Hajek, T., Sherry, S. B., & Grof, P. (2010). Early stages in the development of bipolar disorder. *Journal of Affective Disorders*, *42*, 127. doi:10.1016/j.jad.2009.05.022.

Duffy, A., Horrocks, J., Doucette, S., Keown-Stoneman, C., McCloskey, S., & Grof, P. (2014). The developmental trajectory of bipolar disorder. *British Journal of Psychiatry*, *204*, 122–128. doi:10.1192/bjp.bp.113.126706

Ellenbogen, M. A., & Hodgins, S. (2004). The impact of high neuroticism in parents on children's psychosocial functioning in a population at high risk for major affective disorder: A family environmental pathway of intergenerational risk. *Development and Psychopathology*, *16*, 113–136. doi:10.1017/S0954579404044438

Ellenbogen, M. A., & Hodgins, S. (2009). Structure provided by parents in middle childhood predicts cortisol reactivity in adolescence among the offspring of parents with bipolar disorder and controls. *Psychoneuroendocrinology*, *34*, 773–785. doi:10.1016/j.psyneuen.2008.12.011

Ellenbogen, M. A., Hodgins, S., Linnen, A. M., & Ostiguy, C. S. (2011). Elevated daytime cortisol levels: A biomarker of subsequent major affective disorder? *Journal of Affective Disorders*, *132*, 265–269. doi:10.1016/j.jad.2011.01.007

Ellenbogen, M. A., Hodgins, S., & Walker, C.-D. (2004). High levels of cortisol among adolescent offspring of parents with bipolar disorder: A pilot study. *Psychoneuroendocrinology*, *29*, 99–106. doi:10.1016/S0306-4530(02)00135-X

Else-Quest, N. M., Clark, R., & Tresch Owen, M. (2011). Stability in mother–child interactions from infancy through adolescence. *Parenting*, *11*, 280–287. doi:10.1080/15295192.2011.613724

Ferreira, G. S., Moreira, C. R., Kleinman, A., Nader, E. C., Gomes, B. C., Teixeira, A. M., . . . Caetano, S. C. (2013). Dysfunctional family environment in affected versus unaffected offspring of parents with bipolar disorder. *Australian and New Zealand Journal of Psychiatry*, *47*, 1051–1057. doi:10.1177/0004867413506754

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, research version, non-patient edition* (SCID-I/NP). New York: New York State Psychiatric Institute, Biometrics Research.

Freed, R. D., Tompson, M. C., Wang, C. H., Otto, M. W., Hirshfeld-Becker, D. R., Nierenberg, A. A., & Henin, A. (2015). Family functioning in the context of parental bipolar disorder: Associations with offspring age, sex, and psychopathology. *Journal of Family Psychology*, *29*, 108–118. doi:10.1037/fam0000048

Frick, P. J., Christian, R. E., & Wootton, J. M. (1999). Age trends in the association between parenting practices and conduct problems. *Behavior Modification*, *23*, 106–128. doi:10.1177/0145445599231005

Galambos, N. L., Barker, E. T., & Almeida, D. M. (2003). Parents do matter: Trajectories of change in externalizing and internalizing problems in early adolescence. *Child Development*, *74*, 578–594. doi:0009-3920/2003/7402-0017

Goodyer, I. M., Bacon, A., Ban, M., Croudace, T., & Herbert, J. (2009). Serotonin transporter genotype, morning cortisol and subsequent depression in adolescents. *British Journal of Psychiatry*, *195*, 39–45. doi:10.1192/bjp.bp.108.054775

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. New York: Guilford Press.

Hillegers, M. H., Burger, H., Wals, M., Reichart, C. G., Verhulst, F. C., Nolen, W. A., & Ormel, J. (2004). Impact of stressful life events, familial loading and their interaction on the onset of mood disorders: Study in a high-risk cohort of adolescent offspring of parents with bipolar disorder. *British Journal of Psychiatry*, *185*, 97–101. doi:10.1192/bjp.185. 2.97

Hodges, K. (1981). *Catalog of selected documents in psychology series: Vol. 11. The Child Assessment Schedule: A diagnostic interview for research and clinical use*. Washington, DC: American Psychological Association.

Hodges, K., McKnew, D., Cytryn, L., Stern, L., & Kline, J. (1982). The Child Assessment Schedule (CAS) Diagnostic Interview: A report on reliability and validity. *Journal of the American Academy of Child & Adolescent Psychiatry*, *21*, 468–473. doi:10.1016/S0002-7138(09)60796-2

Hodgins, S., Faucher, B., Zarac, A., & Ellenbogen, M. (2002). Children of parents with bipolar disorder: A population at high risk for major affective disorders. *Child and Adolescent Psychiatric Clinics of North America*, *11*, 533–553. doi:10.1016/S1056-4993(02)00002-0

Hoeve, M., Dubas, J. S., Eichelsheim, V. I., Van der Laan, P. H., Smeenk, W., & Gerris, J. R. (2009). The relationship between parenting and delinquency: A meta-analysis. *Journal of Abnormal Child Psychology*, *37*, 749–775. doi:10.1007/s10802-009-9310-8

Inoff-Germain, G., Nottelmann, E. D., & Radke-Yarrow, M. (1992). Evaluative communications between affectively ill and well mothers and their children. *Journal of Abnormal Child Psychology*, *20*, 189–212. doi:10.1007/BF00916548

Jewell, J. D., & Stark, K. D. (2003). Comparing the family environments of adolescents with conduct disorder or depression. *Journal of Child and Family Studies*, *12*, 77–89. doi:10.1023/A:1021310226400

Kaufman, J., & Schweder, A. E. (2004). The Schedule for Affective Disorders and Schizophrenia for School-Age Children: Present and Lifetime

https://doi.org/10.1017/S095457941700116X Published online by Cambridge University Press

version (K-SADS-PL). In M. J. Hilsenroth & D. L. Segal (Eds.), *Comprehensive handbook of psychological assessment: Vol. 2. Personality assessment* (pp. 247–255). New York: Wiley.
Kieseppä, T., Partonen, T., Haukka, J., Kaprio, J., & Lönnqvist, J. (2014). High concordance of bipolar I disorder in a nationwide sample of twins. *American Journal of Psychiatry*, *161*, 1814–1821. doi:10.1176/appi.ajp.161.10.1814
Kim-Cohen, J., Moffitt, T. E., Taylor, A., Pawlby, S. J., & Caspi, A. (2005). Maternal depression and children's antisocial behavior: Nature and nurture effects. *Archives of General Psychiatry*, *62*, 173–181. doi:10.1001/archpsyc.62.2.173
Klimes-Dougan, B., Ronsaville, D., Wiggs, E. A., & Martinez, P. E. (2006). Neuropsychological functioning in adolescent children of mothers with a history of bipolar or major depressive disorders. *Biological Psychiatry*, *60*, 957–965. doi:10.1016/j.biopsych.2006.03.031
Kroes, G., Veerman, J. W., & De Bruyn, E. E. (2003). Bias in parental reports? Maternal psychopathology and the reporting of problem behavior in clinic-referred children. *European Journal of Psychological Assessment*, *19*, 195. doi:10.1027//1015-5759.19.3.195
Lam, D., Donaldson, C., Brown, Y., & Malliaris, Y. (2005). Burden and marital and sexual satisfaction in the partners of bipolar patients. *Bipolar Disorders*, *7*, 431–440. doi:10.1111/j.1399-5618.2005.00240.x
Lanza, H. I., & Drabick, D. A. (2011). Family routine moderates the relation between child impulsivity and oppositional defiant disorder symptoms. *Journal of Abnormal Child Psychology*, *39*, 83–94. doi:10.1007/s10802-010-9447-5
Larsson, H., Viding, E., Rijsdijk, F., & Plomin, R. (2008). Relationships between parental negativity and childhood antisocial behavior over time: A bidirectional effects model in a longitudinal genetically informative design. *Journal of Abnormal Child Psychology*, *36*, 633–645. doi:10.1007/s10802-007-9151-2
Larzelere, R. E., Morris, A. S. E., & Harrist, A. W. (Eds.). (2013). *Authoritative parenting: Synthesizing nurturance and discipline for optimal child development*. Washington, DC: American Psychological Association.
Linnen, A. M., aan het Rot, M., Ellenbogen, M. A., & Young, S. N. (2009). Interpersonal functioning in adolescent offspring of parents with bipolar disorder. *Journal of Affective Disorders*, *114*, 122–130.
Mathews, C. A., & Reus, V. I. (2001). Assortative mating in the affective disorders: A systematic review and meta-analysis. *Comprehensive Psychiatry*, *42*, 257–262. doi:10.1053/comp.2001.24575
McGuffin, P., Rijsdijk, F., Andrew, M., Sham, P., Katz, R., & Cardno, A. (2003). The heritability of bipolar affective disorder and the genetic relationship to unipolar depression. *Archives of General Psychiatry*, *60*, 497–502. doi:10.1001/archpsyc.60.5.497
McKinney, C., & Renk, K. (2008). Differential parenting between mothers and fathers: Implications for late adolescents. *Journal of Family Issues*, *29*, 806–827. doi:10.1177/0192513X07311222
McLeod, B. D., Weisz, J. R., & Wood, J. J. (2007). Examining the association between parenting and childhood depression: A meta-analysis. *Clinical Psychology Review*, *27*, 986–1003. doi:10.1016/j.cpr.2007.03.001
McLeod, B. D., Wood, J. J., & Weisz, J. R. (2007). Examining the association between parenting and childhood anxiety: A meta-analysis. *Clinical Psychology Review*, *27*, 155–172. doi:10.1016/j.cpr.2006.09.002
Mesman, E., Nolen, W. A., Reichart, C. G., Wals, M., & Hillegers, M. H. (2013). The Dutch bipolar offspring study: 12-year follow-up. *American Journal of Psychiatry*, *170*, 542–549. doi:10.1176/appi.ajp.2012.12030401
Meyer, S. E., Carlson, G. A., Wiggs, E. A., Ronsaville, D. S., Martinez, P. E., Klimes-Dougan, B., . . . Radke-Yarrow, M. (2006). A prospective high-risk study of the association among maternal negativity, apparent frontal lobe dysfunction, and the development of bipolar disorder. *Development and Psychopathology*, *18*, 573–589. doi:10.10170S0954579406060299
Miklowitz, D. J. (2007). The role of the family in the course and treatment of bipolar disorder. *Current Directions in Psychological Science*, *16*, 192–196. doi:10.1111/j.1467-8721.2007.00502.x
Miklowitz, D. J. (2010). *Bipolar disorder: A family-focused treatment approach*. New York: Guilford Press.
Miklowitz, D. J., & Cicchetti, D. (Eds.). (2006). A developmental perspective on bipolar disorder [Special Issue]. *Development and Psychopathology*, *18*, 935–1317. doi:10.10170S0954579406060469
Miklowitz, D. J., & Cicchetti, D. (Eds.) (2010). *Bipolar disorder: A developmental psychopathology approach*. New York: Guilford Press.
Miklowitz, D. J., Goldstein, M. J., Nuechterlein, K. H., Snyder, K. S., & Mintz, J. (1988). Family factors and the course of bipolar affective disorder. *Archives of General Psychiatry*, *45*, 225–231. doi:10.1001/archpsyc.1988.01800270033004
Miklowitz, D. J., Wisniewski, S. R., Miyahara, S., Otto, M. W., & Sachs, G. S. (2005). Perceived criticism from family members as a predictor of the one-year course of bipolar disorder. *Psychiatry Research*, *136*, 101–111. doi:10.1016/j.psychres.2005.04.005
Milevsky, A., Schlechter, M., Netter, S., & Keehn, D. (2007). Maternal and paternal parenting styles in adolescents: Associations with self-esteem, depression and life-satisfaction. *Journal of Child and Family Studies*, *16*, 39–47. doi:10.1007/s10826-006-9066-5
Morgan, Z., Brugha, T., Fryers, T., & Stewart-Brown, S. (2012). The effects of parent–child relationships on later life mental health status in two national birth cohorts. *Social Psychiatry and Psychiatric Epidemiology*, *47*, 1707–1715. doi:10.1007/s00127-012-0481-1
Nijjar, R., Ellenbogen, M. A., & Hodgins, S. (2014). Personality, coping, risky behavior, and mental disorders in the offspring of parents with bipolar disorder: A comprehensive psychosocial assessment. *Journal of Affective Disorders*, *166*, 315–323. doi:10.1016/j.jad.2014.04.047
Nijjar, R., Ellenbogen, M. A., & Hodgins, S. (2016). Sexual risk behaviors in the adolescent offspring of parents with bipolar disorder: Prospective associations with parents' personality and externalizing behavior in childhood. *Journal of Abnormal Child Psychology*. Advance online publication. doi:10.1007/s10802-015-0112-x
Nilsson, K. W., Comasco, E., Hodgins, S., Oreland, L., & Åslund, C. (2015). Genotypes do not confer risk for delinquency but rather alter susceptibility to positive and negative environmental factors: Gene–environment interactions of BDNF Val66Met, 5-HTTLPR, and MAOA-uVNTR. *International Journal of Neuropsychopharmacology*, *18*, 1–10. doi:10.1093/ijnp/pyu107
Noom, M. J., Deković, M., & Meeus, W. H. (1999). Autonomy, attachment and psychosocial adjustment during adolescence: a double-edged sword? *Journal of Adolescence*, *22*, 771–783. doi:10.1006/jado.1999.0269
Ostiguy, C. S., Ellenbogen, M. A., & Hodgins, S. (2012). Personality of parents with bipolar disorder and interpersonal functioning among their offspring: A prospective 10-year study. *Development and Psychopathology*, *24*, 573–587. doi:10.1017/S095457941200017X
Ostiguy, C. S., Ellenbogen, M. A., Linnen, A. M., Walker, E. F., Hammen, C., & Hodgins, S. (2009). Chronic stress and stressful life events in the offspring of parents with bipolar disorder. *Journal of Affective Disorders*, *114*, 74–84. doi:10.1016/j.jad.2008.08.006
Ostiguy, C. S., Ellenbogen, M. A., Walker, C. D., Walker, E. F., & Hodgins, S. (2011). Sensitivity to stress among the offspring of parents with bipolar disorder: A study of daytime cortisol levels. *Psychological Medicine*, *41*, 2447–2457. doi:10.1017/S0033291711000523
Piko, B. F., & Balázs, M. Á. (2012). Authoritative parenting style and adolescent smoking and drinking. *Addictive Behaviors*, *37*, 353–356. doi:10.1016/j.addbeh.2011.11.022
Pini, S., de Queiroz, V., Pagnin, D., Pezawas, L., Angst, J., Cassano, G. B., & Wittchen, H.-U. (2005). Prevalence and burden of bipolar disorders in European countries. *European Neuropsychopharmacology*, *15*, 425–434. doi:10.1016/j.euroneuro.2005.04.011
Post, R. M., Leverich, G. S., Xing, G., & Weiss, S. R. (2001). Developmental vulnerabilities to the onset and course of bipolar disorder. *Development and Psychopathology*, *13*, 581–598.
Radke-Yarrow, M. (1998). *Children of depressed mothers: From early childhood to maturity*. New York: Cambridge University Press.
Rasic, D., Hajek, T., Alda, M., & Uher, R. (2013). Risk of mental illness in offspring of parents with schizophrenia, bipolar disorder, and major depressive disorder: A meta-analysis of family high-risk studies. *Schizophrenia Bulletin*. Advance online publication. doi:10.1093/schbul/sbt114
Romero, S., DelBello, M. P., Soutullo, C. A., Stanford, K., & Strakowski, S. M. (2005). Family environment in families with versus families without parental bipolar disorder: A preliminary comparison study. *Bipolar Disorders*, *7*, 617–622. doi:10.1111/j.1399-5618.2005.00270.x
Rucker, D. D., Preacher, K. J., Tormala, Z. L., & Petty, R. E. (2011). Mediation analysis in social psychology: Current practices and new recommendations. *Social and Personality Psychology Compass*, *5*, 359–371. doi:10.1111/j.1751-9004.2011.00355.x
Schaffer, A., Cairney, J., Cheung, A., Veldhuizen, S., & Levitt, A. (2006). Community survey of bipolar disorder in Canada: Lifetime prevalence and illness characteristics. *Canadian Journal of Psychiatry*, *51*, 9–16.
Schudlich, T. D. D. R., Youngstrom, E. A., Calabrese, J. R., & Findling, R. L. (2008). The role of family functioning in bipolar disorder in families. *Journal of Abnormal Child Psychology*, *36*, 849–863.
Schultz, P. W., & Searleman, A. (2002). Rigidity of thought and behavior: 100 years of research. *Genetic, Social, and General Psychology Monographs*, *128*, 165.

https://doi.org/10.1017/S095457941700116X Published online by Cambridge University Press

Simons, L. G., & Conger, R. D. (2007). Linking mother–father differences in parenting to a typology of family parenting styles and adolescent outcomes. *Journal of Family Issues*, *28*, 212–241. doi:10.1177/0192513X06294593

Singh, M. K., Delbello, M. P., Stanford, K. E., Soutullo, C., Mcdonough-Ryan, P., McElroy, S. L., & Strakowski, S. M. (2007). Psychopathology in children of bipolar parents. *Journal of Affective Disorders*, *102*, 131–136. doi:10.1016/j.jad.2007.01.004

Slater, M. A., & Power, T. G. (1987). Multidimensional assessment of parenting in single-parent families. In J. P. Vincent (Ed.), *Advances in family intervention, assessment and theory*. Greenwich, CT: JAI.

Spitzer, R. L., Williams, J. B., Gibbon, M., & First, M. B. (1992). The Structured Clinical Interview for DSM-III-R (SCID): I. History, rationale, and description. *Archives of General Psychiatry*, *49*, 624–629.

Vance, Y. H., Huntley, J. S., Espie, J., Bentall, R., & Tai, S. (2008). Parental communication style and family relationships in children of bipolar parents. *British Journal of Clinical Psychology*, *47*, 355–359. doi:10.1348/014466508X282824

Verhulst, F. C., Althaus, M., & Berden, G. (1987). The child assessment schedule: Parent-child agreement and validity measures. *Journal of Child Psychology and Psychiatry*, *28*, 455–466. doi:10.1111/j.1469-7610.1987.tb01766.x

Wals, M., Hillegers, M. H., Reichart, C. G., Verhulst, F. C., Nolen, W. A., & Ormel, J. (2005). Stressful life events and onset of mood disorders in children of bipolar parents during 14-month follow-up. *Journal of Affective Disorders*, *87*, 253–263. doi:10.1016/j.jad.2005.04.006

Weich, S., Patterson, J., Shaw, R., & Stewart-Brown, S. (2009). Family relationships in childhood and common psychiatric disorders in later life: Systematic review of prospective studies. *British Journal of Psychiatry*, *194*, 392–398. doi:10.1192/bjp.bp.107.042515

Wilson, S., & Durbin, C. E. (2010). Effects of paternal depression on fathers' parenting behaviors: A meta-analytic review. *Clinical Psychology Review*, *30*, 167–180. doi:10.1016/j.cpr.2009.10.007

Wood, J. J., McLeod, B. D., Sigman, M., Hwang, W. C., & Chu, B. C. (2003). Parenting and childhood anxiety: Theory, empirical findings, and future directions. *Journal of Child Psychology and Psychiatry*, *44*, 134–151. doi:10.1111/1469-7610.00106

World Health Organization. (2001). *The World Health Report 2001—Mental health: New understandings, new hope*. Geneva: Author.

Yap, M. B. H., & Jorm, A. F. (2015). Parental factors associated with childhood anxiety, depression, and internalizing problems: A systematic review and meta-analysis. *Journal of Affective Disorders*, *175*, 424–440. doi:10.1016/j.jad.2015.01.050

Yap, M. B. H., Pilkington, P. D., Ryan, S. M., & Jorm, A. F. (2014). Parental factors associated with depression and anxiety in young people: A systematic review and meta-analysis. *Journal of Affective Disorders*, *156*, 8–23. doi:10.1016/j.jad.2013.11.007

https://doi.org/10.1017/S095457941700116X Published online by Cambridge University Press