

CHILDREN'S EMOTION REGULATION DIFFICULTIES MEDIATE THE ASSOCIATION BETWEEN MATERNAL BORDERLINE AND ANTISOCIAL SYMPTOMS AND YOUTH BEHAVIOR PROBLEMS OVER 1 YEAR

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Borderline personality disorder (BPD) and antisocial personality disorder (ASPD) are among the most debilitating psychiatric conditions. Behaviors and traits associated with these disorders can have profound influences on those surrounding the affected individual. Accordingly, researchers have begun to examine effects of these symptoms on parent-child relationships. Theoretical and empirical work suggests that one mechanism linking maternal psychopathology to child symptoms is familial transmission of emotion dysregulation. The authors examined children's emotion regulation difficulties as a mediator between maternal BPD/ASPD symptoms and child behavior problems 1 year later. Analyses revealed that a composite of maternal BPD/ASPD symptoms had a direct effect on child internalizing, externalizing, and total symptoms. Associations between maternal BPD/ASPD symptoms and youth problems were partially mediated by child emotion regulation difficulties, even with maternal depression and other relevant covariates included in the models. Thus, maternal BPD/ASPD symptoms and child emotion regulation difficulties represent potential targets for prevention of psychopathology among youth.

Borderline personality disorder (BPD) and antisocial personality disorder (ASPD) are two of the most impairing mental health conditions. Each is associated with tremendous financial and emotional costs for affected individuals and society more broadly (American Psychiatric Association [APA], 2013; Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Trull, Distel,

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& Carpenter, 2011). Although ASPD is more commonly diagnosed among males and BPD is more commonly diagnosed among females, the two disorders share common features, stem from similar etiological factors, and are comorbid at relatively high rates (Beauchaine et al., 2009; Paris, Chenard-Poirier, & Biskin, 2013). Psychosocial risk factors for ASPD and BPD overlap substantially (e.g., parental psychopathology, family dysfunction, coercive family processes), and shared biological vulnerabilities have also been identified (e.g., serotonergic dysfunction, trait impulsivity; see Beauchaine et al., 2009; Kaufman, Crowell, & Stepp, 2015; Zanarini, 2000). In fact, recent longitudinal twin research indicates that genetic liability for ASPD and BPD is largely shared (Reichborn-Kjennerud et al., 2015). BPD and ASPD are each characterized by persistent and pervasive negative affectivity, interpersonal problems, and disinhibition (APA, 2013; Trull et al., 2011).

Epidemiological surveys indicate that BPD affects between 1.2% and 5.9% of community adults, with studies converging on a prevalence rate around 2% (Grant et al., 2008; Lenzenweger, Lane, Loranger, & Kessler, 2007; Trull, Jahng, Tomko, Wood, & Sher, 2010). Although studies with community samples suggest that prevalence rates are comparable across sexes, BPD is associated with more severe psychological and physical impairment among women (Grant et al., 2008). Furthermore, in clinical samples, approximately 70%–80% of those who meet criteria for BPD are female (Skodol & Bender, 2003; Swartz, Blazer, George, & Winfield, 1990). Lifetime prevalence estimates indicate that ASPD affects approximately 1.2% of community women (Kessler, Chiu, Demler, Merikangas, & Walters, 2005), although rates vary notably by study (see Paris, 2010). Some research suggests that antisocial features are also more impairing among women compared with men (Alegria et al., 2013). Thus, millions of women are suffering from one or both of these personality disorders in the United States, and many affected individuals are likely to become mothers.

Children of mothers with BPD and ASPD are at risk for a host of negative outcomes. Offspring of mothers with BPD have higher rates of internalizing and externalizing psychopathology and poorer psychosocial functioning compared to children of mothers without BPD (Barnow, Spitzer, Grabe, Kessler, & Freyberger, 2006; Herr, Hammen, & Brennan, 2008; Macfie, 2009; Stepp, Whalen, Pilkonis, Hipwell, & Levine, 2012; Weiss et al., 1996). Maternal antisocial behavior is also associated with increased risk for offspring psychopathology, particularly disruptive behavior (Harold et al., 2010; Harold, Elam, Lewis, Rice, & Thapar, 2012; Kim-Cohen, Caspi, Rutter, Tomás, & Moffitt, 2006; Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003). Longitudinal twin research indicates that children of mothers with comorbid antisocial and depressive features have significantly higher levels of antisocial behavior and rates of conduct disorder compared with children of mothers with depression alone (Kim-Cohen et al., 2006).

Although maternal BPD and ASPD have been linked consistently to poor developmental outcomes, there is insufficient research examining *why* these children may be at greater risk (Barnow et al., 2006). Although a number of factors contribute to intergenerational transmission of psychopathology (e.g., genetic transmission, shared neurological and physiological vulner-

abilities, ineffective parenting, poverty, family conflict; Barnow et al., 2006; Feldman, Zerkowitz, Weiss, & Vogel, 1995; Golomb et al., 1994; Gunderson et al., 2011; Stepp et al., 2012; Torgersen, 2000), it is especially important to consider the influence of maternal symptoms on children's key developmental tasks (Gratz et al., 2014). Child behavior problems often emerge when stage-salient developmental tasks are not navigated effectively, thereby delaying or preventing acquisition of later skills (Sroufe & Rutter, 1984).

Emotion regulation is one developmental task that warrants attention when examining familial transmission of psychopathology (Gratz et al., 2014; Mazursky-Horowitz et al., 2015; Suveg, Shaffer, Morelen, & Thomassin, 2011). Emotion regulation is essential to normative development and encompasses automatic and volitional behaviors, skills, and strategies, which modulate emotional experiences and expressions (Beauchaine, 2015; Calkins & Marcovitch, 2010; Thompson, 1990). Successful emotion regulation abilities build upon a foundation of early coregulation by parents and stable interpersonal relationships (e.g., Diamond & Fagundes, 2008; Hughes, Crowell, Uyeji, & Coan, 2012). Prospective longitudinal research shows that children with emotion regulation skills have fewer negative outcomes, even when they display high levels of negative affect, whereas children with high levels of negative affect and difficulties with emotion regulation display lower social competence and greater behavior problems (Eisenberg et al., 1997; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011). Importantly, emotion regulation and dysregulation are often socialized within families (see Beauchaine, 2015; Beauchaine & Zalewski, 2015; Crowell, Beauchaine, & Linehan, 2009; McLaughlin et al., 2011; Suveg et al., 2011).

Disrupted affect and problems with emotion regulation appear as early as infancy among children of mothers with personality pathology (Crandell, Patrick, & Hobson, 2003; Gratz et al., 2014; Hobson, Patrick, Crandell, Garcia-Perez, & Lee, 2005; Newman, Stevenson, Bergman, & Boyce, 2007). This is perhaps unsurprising, given that mothers with BPD and ASPD are likely to pass down biological vulnerabilities to their offspring and also rear their children in environments that may compromise development of regulatory skills (Goldsmith, Pollak, & Davidson, 2008; Kim-Cohen et al., 2006; Morris, Silk, Steinberg, Myers, & Robinson, 2007; Sellers et al., 2014). Research on mothers with BPD, ASPD, and their families reveals marked discord and conflict in the home, low family cohesiveness, high divorce rates, and financial hardships (Feldman et al., 1995; Golomb et al., 1994; Kim-Cohen et al., 2006). Furthermore, preschoolers of mothers with a BPD diagnosis experience more difficulties with emotion regulation compared to control families (See Macfie, 2009, for a review; Macfie & Swann, 2009). Theoretical and empirical work implicates intergenerational transmission of emotion dysregulation as a mechanism explaining links between maternal BP and child psychopathology (see, e.g., Gratz et al., 2014; Stepp et al., 2012). However, no studies have examined the combined influence of borderline and antisocial symptoms on child emotion regulation and behavior problems, in spite of broad evidence that these diagnoses co-occur at high rates and are likely to affect child outcomes.

At present, it is unclear whether maternal BPD/ASPD symptoms contribute to child emotion regulation difficulties or later behavioral problems above and beyond comorbid diagnoses or relevant contextual stressors. Major depressive disorder (MDD) commonly co-occurs with ASPD, and approximately 80% of those with BPD also meet criteria for MDD in their lifetime (Gunderson, 2011; Lenzenweger et al., 2007; Zanarini et al., 1998). A large literature examines the relation between maternal depression and child behavior problems (e.g., Downey & Coyne, 1990; Seifer, Dickstein, Sameroff, Magee, & Hayden, 2001; Tompson et al., 2010). Similar to children of mothers with BPD and ASPD, children of depressed mothers often develop a wide range of internalizing and externalizing symptoms, including emotion regulation difficulties (e.g., Herwig, Wirtz, & Bengel, 2004; Hoffman, Crnic, & Baker, 2006; Maughan, Cicchetti, Toth, & Rogosch, 2007). Models of maternal ASPD often account for maternal depression and still find unique contributions to child psychopathology (e.g., Harold et al., 2010, 2012; Kim-Cohen et al., 2006). Yet many studies have not included depression or ASPD in models of maternal BPD, and a majority of maternal depression studies fail to assess for mothers' borderline or antisocial symptoms. This is a significant limitation. Because ASPD is more commonly diagnosed in males, we may be overlooking important antisocial features among women and mothers.

In the current study, we hypothesize that a composite maternal BPD/ASPD symptom score will predict youth internalizing and externalizing psychopathology, and that this relation will be mediated by child emotion regulation difficulties. We also examine whether this composite of maternal BPD/ASPD symptoms is associated with child behavior problems 1 year later, even with maternal depression and other relevant contextual factors (maternal marital status, maternal education, and annual household income) included in the models. Each criterion for BPD and ASPD is impairing and has the potential to affect child development negatively. Thus, we chose to examine continuous symptom scores rather than diagnostic status as our independent variable.

METHOD

PARTICIPANTS

After obtaining institutional review board approval, participants were recruited from predominantly lower socioeconomic status (SES) neighborhoods in the greater Seattle metropolitan area through King County bus and local newspaper advertisements, community publications, direct mailings, flyers placed in community centers, and radio advertisements. Separate advertisements targeted children who were either well adjusted (controls), down or depressed (depressed), and/or experiencing behavior problems (conduct disorder [CD]/comorbid depression and CD). Each advertisement stated that parents and their child could earn up to \$175 by participating in a study about children's emotional adjustment. Median family income was

\$47,000, well below the median family income for Seattle at the time of enrollment (United States Census Bureau, 2005).

Parents who responded to advertisements completed a 20- to 30-minute telephone screening interview with a trained research assistant. Interviewers administered a computerized diagnostic interview that included portions of the Child Symptom Inventory (CSI; Gadow & Sprafkin, 1997) and the Child Behavior Checklist (CBCL; Achenbach, 1991). The CSI yields both dimensional scores and diagnostic cutoffs for many *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (*DSM-IV*) disorders. Eligibility was determined based on the conduct disorder (CD), attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), major depressive disorder (MDD), and dysthymia (DYS) subscales of the CSI and the aggression, attention problems, and anxious/depressed subscales of the CBCL. Of 445 parents who completed the phone interview, 206 (46.3%) were accepted based on their CSI and CBCL profiles and completed some portion of the study. Those who were not accepted failed to meet required cutoffs for the study groups.

Children were admitted based on meeting criteria for one of four groups, as assessed during the structured phone interview and later confirmed with the Diagnostic Interview Schedule for Children and Adolescents (NIMH DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). At baseline, children in the CD group were required to meet CSI criteria for CD and/or ODD, but not for DYS or MDD ($n = 28$, 14% of sample; 79% male). Children in the DEP group were required to meet CSI criteria for DYS and/or MDD, but not CD or ODD ($n = 27$, 13% of sample; 67% male). Children in the comorbid group were required to meet criteria for both the conduct problems and DEP groups ($n = 81$, 39% of sample; 75% male), or no diagnostic criteria ($n = 70$, 40% of sample; 47% male). Control participants could not meet criteria for any disorder on the CSI and did not exceed a T score > 59 on any CBCL scale. As discussed below, all analyses were conducted using continuous scores rather than categorical diagnoses. Parental willingness to participate with an eligible child was the only inclusion criterion for parents.

We analyzed data from participants who completed the second (Year 2) and third (Year 3) time points of the project, which occurred 12 and 24 months after the baseline (Year 1) assessment. This approach was necessary because maternal borderline and antisocial symptoms were not assessed at Year 1. We retained 92% (196 parent-child dyads) and 77% (164 parent-child dyads) of the initial sample at the Year 2 and Year 3 assessments, respectively. For the present analyses, complete data were available for 119 dyads. Demographic information for the sample is presented in Table 1. At Year 2, children were 56.3% male and 9–14 years old ($M = 11.0$, $SD = 1.5$). Consistent with the ethnic representation of the study location, a majority of parents identified their children as White (63.9%). The remaining 36% identified their children's ethnicity as African American (9.2%), mixed or unspecified heritage (3.4%), Latino (12.6%), Asian American/Pacific Islander (9.6%), and Native American (1.7%). Parents were 27–60 years old ($M = 41.3$, $SD = 7.6$). Because so few fathers participated ($n = 4$ at Year 1, $n = 5$ at Year 2), their data are omitted.

TABLE 1. Demographic Characteristics of the Sample

Variable	Mean (<i>SD</i>)	Frequency (%)
Child age		
Year 2	10.95 (1.49)	—
Year 3	12.03 (1.48)	—
Child sex		
Male	—	67 (56.3)
Female	—	52 (43.7)
Child race		
Caucasian	—	76 (63.9)
African American	—	11 (9.2)
Asian American/Pacific Islander	—	11 (9.2)
Hispanic/Latino	—	15 (12.6)
Native American	—	2 (1.7)
Other/Mixed Ethnicity	—	4 (3.4)
Mother age		
Year 2	41.33 (7.65)	—
Mother marital status		
Married or living with someone	—	76 (63.9)
Separated	—	10 (8.4)
Divorced or annulled	—	22 (18.5)
Widowed	—	1 (0.8)
Never married	—	10 (8.4)
Mother education		
Grade 6 or less	—	4 (3.4)
Grade 7–12	—	3 (2.5)
High school	—	5 (4.2)
Partial college	—	42 (35.3)
Graduated 2-year college	—	9 (7.6)
Graduated 4-year college	—	27 (22.7)
Partial postgraduate studies	—	11 (9.2)
Completed postgraduate studies	—	18 (15.1)
Annual household income	\$54,660 (\$33,560)	—

PROCEDURE

We collected informed parental consent and child assent at each assessment. Details of the baseline assessment can be found elsewhere (e.g., Gatzke-Kopp & Beauchaine, 2007). At follow-up Years 1 and 2, children and their mothers returned to the lab for further assessments. Graduate research assistants conducted a clinical interview with parents using the nonpatient version of the Structured Clinical Interview for the *DSM-IV-TR* (SCID; First, Spitzer, Gibbon, & Williams, 2002) and the SCID-II (First, Spitzer, Gibbon, & Williams, 1997). Among other modules, we included the MDD section of the SCID I, and the BPD and ASPD sections of the SCID-II. Research indicates excellent interrater reliability (intraclass correlation coefficient [ICC] = .96) for the MDD module (Mitchell, Wolf, Reardon, & Miller, 2014), good to excellent reliability for criteria in the BPD module (ICC values for each criterion range from .75 to .95 and average .86; Huprich, Paggeot, & Samuel, 2015), and good to excellent reliability for the ASPD module (kappas ranging from .74 to 1.0; Guy, Poythress, Douglas, Skeem, & Edens, 2008; Maffei et al., 1997; Zanarini et al., 2000).

Parents also completed the CBCL, which has well-established reliability and validity (Achenbach, 1991; Achenbach & Rescorla, 2001). The CBCL

includes a broadband externalizing factor (composed of aggressive behavior and delinquency subscales), a broadband internalizing factor (composed of anxious/depressed, somatic complaints, and withdrawn subscales), and other subscales that do not load onto either externalizing or internalizing (thought problems, social problems, attention problems).

Youth also completed the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). This measure was developed for adults to capture multiple aspects of emotion dysregulation. The initial factor analysis revealed six scales: nonacceptance of emotional responses (nonacceptance), difficulties engaging in goal-directed behavior (goals), impulse control difficulties (impulse), lack of emotional awareness (awareness), limited access to emotion regulation strategies (strategies), and lack of emotional clarity (clarity). The factor structure has since been replicated with youth, and the DERS yields good reliability and validity with adolescents ages 11–17 (Neumann, van Lier, Gratz, & Koot, 2010; Weinberg & Klonsky, 2009). In another report with the present sample (Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009), Cronbach's α s ranged from .72 to .87 across subscales (reliability), and psychophysiological profiles predicted self-reported emotion dysregulation (predictive validity).

ANALYTIC PLAN

We first assessed the independent effects of maternal ASPD and BPD symptoms on child emotion regulation difficulties and psychopathology. Next, to determine their additive effect on child outcomes, scores from the BPD and ASPD modules of the SCID-II were standardized and combined as a unit-weighted BPD/ASPD personality pathology composite variable, such that each subscale contributed equally to the composite. Specifically, BPD and ASPD items were each summed into total scores from their respective sections of the SCID interview before being combined into a composite score. Although two criteria for BPD and ASPD diagnoses are highly similar (i.e., each assesses impulsivity and anger/irritability), each diagnosis requires the symptoms to manifest in distinct ways. Furthermore, the items from the SCID interview that assess these criteria are worded differently in the ASPD and BPD modules (First et al., 1997). For example, impulsivity is assessed within the BPD interview specifically in regard to self-damaging behaviors, whereas criteria for ASPD are broader, assessing for impulsivity generally and failure to plan ahead (APA, 2000; First et al., 1997). Similarly, the DSM presents irritability/anger quite broadly in the context of BPD (i.e., “inappropriate, intense anger or difficulty controlling anger” [APA, 2013, p. 663]), but specifically as aggression and physical fighting in ASPD (APA, 2000; First et al., 1997). Therefore, we chose to include these criteria in the total scores for each disorder before creating a composite. For purposes of this study, all data were analyzed using continuous scores of symptom severity rather than diagnoses or groups. There are statistical advantages of analyzing continuous variables, including greater power, decreased risk of Type I error, and the retention of more nuanced information about variation in both symptoms

TABLE 2. Descriptive Statistics and Correlations for SCID-II Maternal BPD (Year 2), SCID-I Maternal Depression (Year 2), Child-Reported DERS Subscales (Year 3), and CBCL Subscales (Year 3)

	M (SD)	1.	2.	3.	4.	5.	6.	7.	8.
SCID symptom scores †									
1. Maternal BPD	10.8 (2.8)	—	.65**	.91**	.35**	.15	.29**	.42**	.38**
2. Maternal ASPD	8.5 (2.3)	.65**	—	.91**	.13	.21*	.22*	.36**	.34**
3. Maternal ASPD/BPD	— §	.91**	.91**	—	.27**	.19*	.28**	.42**	.39**
4. Maternal Depression	12.0 (5.3)	.35**	.13	.27**	—	-.12	.17*	.18*	.21*
DERS scores ‡									
5. Child total	76.0 (22.4)	.15	.21*	.19*	-.12	—	.31**	.38**	.37**
CBCL <i>t</i> scores ‡									
6. Child Internalizing	53.9 (13.0)	.29**	.22*	.28**	.17*	.31**	—	.68**	.89**
7. Child Externalizing	51.7 (12.2)	.42**	.36**	.42**	.18*	.38**	.68**	—	.88**
8. Child Total Problems	51.9 (12.4)	.38**	.34**	.39**	.21*	.37**	.89**	.88**	—

Note. ASPD = antisocial personality disorder; BPD = borderline personality disorder; CBCL = Child Behavior Checklist; DERS = Difficulties with Emotion Regulation Scale; SCID = Structured Clinical Interview for *DSM-IV-TR*. †From Year 2 visit; ‡from Year 3 visit; §due to standardization of BPD and ASPD symptom variables prior to the creation of the composite, the mean and standard deviation of the ASPD/BPD composite variable are not readily interpretable and therefore are not reported. * $p < .01$. ** $p < .001$.

and outcomes (see, e.g., Beauchaine, 2003; MacCallum, Zhang, Preacher, & Rucker, 2002).

We used Hayes's (2012, 2013) PROCESS computational macro in SPSS version 22.0 to test three models in which total scores on the DERS mediated the relation between maternal BPD/ASPD personality symptoms and child CBCL *t* scores. Although internalizing and externalizing psychopathology are often conceptualized as developing through independent processes, recent evidence suggests that common risk and vulnerability factors may also apply to the development of psychopathology more broadly (Caspi et al., 2014). Therefore, we tested three mediation models with *t* scores for internalizing problems, externalizing problems, and total problems each considered as outcome variables. Given the relatively small sample size and because we expected the distribution of mothers' personality disorder symptoms to be positively skewed, we used nonparametric bootstrapping analyses based on 1,000 bootstrapped samples to test the mediational models (Preacher & Hayes, 2004; Preacher, Rucker, & Hayes, 2007).

RESULTS

DESCRIPTIVES

Means, standard deviations, and bivariate correlations are reported in Table 2. Distributions of all reported scales were relatively normal (i.e., skew and kurtosis ≤ 2 or > 2), with the exception of BPD and ASPD symptoms on the SCID-II (BPD: skew = 2.4; kurtosis = 6.2; ASPD: skew = 2.1, kurtosis = 4.7) and the composite BPD/ASPD personality symptom variable (skew = 2.4, kurtosis = 6.9), which were positively skewed and leptokurtotic. Given that

TABLE 3. Results of Test for Child Emotion Regulation Difficulties as Mediator of the Relation Between Maternal BPD/ASPD Symptoms and Child Externalizing Problems

	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>	<i>F</i> (<i>df</i> ₁ , <i>df</i> ₂)	<i>R</i>	<i>R</i> ²
<i>Model summary: ASPD symptoms</i>	—	—	—	< .001	<i>F</i> (6, 112) = 6.65	.52	.27
Direct Effects							
Maternal ASPD symptoms	1.23	0.49	2.52	.01	—	—	—
Child emotion regulation difficulties	0.19	0.05	4.13	< .001	—	—	—
Indirect Effect							
Child emotion regulation difficulties	0.44	0.23	—	[0.08, 0.99]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.08	0.04†	—	[0.01, 0.16]‡	—	—	—
<i>Model summary: BPD symptoms</i>	—	—	—	< .001	<i>F</i> (6, 112) = 7.71	.54	.29
Direct Effects							
Maternal BPD symptoms	1.27	0.39	3.27	.001	—	—	—
Child emotion regulation difficulties	0.19	0.04	4.14	< .001	—	—	—
Indirect Effect							
Child emotion regulation difficulties	0.33	0.21†	—	[0.00, 0.86]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.07	0.04†	—	[0.00, 0.18]‡	—	—	—
<i>Model summary: BPD/ASPD symptoms</i>	—	—	—	< .001	<i>F</i> (6, 112) = 7.70	.54	.29
Direct Effects							
Maternal BPD/ASPD symptoms	3.99	1.22	3.26	.002	—	—	—
Child emotion regulation difficulties	0.18	0.05	4.03	< .001	—	—	—
Indirect Effect							
Child emotion regulation difficulties	1.13	0.62†	—	[0.20, 2.76]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.08	0.04†	—	[0.01, 0.18]‡	—	—	—

Note. ASPD = antisocial personality disorder; BPD = borderline personality disorder. †Bootstrap-derived estimate of standard error of indirect effect, 1,000 bootstrapped samples. ‡95% bias-corrected lower-level and upper-level confidence interval, 1,000 bootstrapped samples.

the present study did not recruit participants based on maternal personality psychopathology, such distributional properties are not unexpected; additionally, bootstrapping is advantageous in that it does not require that the data conform to particular distributional assumptions (Hayes & Preacher, 2010).

MEDIATION OF CHILD OUTCOMES

To establish the first step of mediation, we examined unmediated models of child outcome variables (i.e., externalizing problems, internalizing problems, and total problems) regressed onto maternal BPD/ASPD symptoms and child emotion regulation difficulties. Maternal depression, maternal marital status, maternal education, and annual household income were included as covariates. Results are reported separately for each outcome variable below (also, see Tables 3–5.) Second, a model regressing child emotion regulation difficulties onto maternal BPD/ASPD symptoms yielded a positive associa-

TABLE 4. Results of Test for Child Emotion Regulation Difficulties as Mediator of the Relation Between Maternal BPD/ASPD Symptoms and Child Internalizing Problems

	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>	<i>F</i> (<i>df</i> ₁ , <i>df</i> ₂)	<i>R</i>	<i>R</i> ²
<i>Model summary: ASPD symptoms</i>	—	—	—	.002	<i>F</i> (6, 112) = 3.72	.41	.17
Direct Effects							
Maternal ASPD symptoms	0.63	0.55	1.14	.26	—	—	—
Child emotion regulation difficulties	0.18	0.05	3.43	< .001	—	—	—
Indirect Effect							
Child emotion regulation difficulties	0.41	0.22†	—	[0.06, 0.94]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.07	0.04†	—	[0.01, 0.15]‡	—	—	—
<i>Model summary: BPD symptoms</i>	—	—	—	< .001	<i>F</i> (6, 12) = 4.14	.43	.18
Direct Effects							
Maternal BPD symptoms	0.88	0.44	1.99	.049	—	—	—
Child emotion regulation difficulties	0.17	0.05	3.25	.002	—	—	—
Indirect Effect							
Child emotion regulation difficulties	0.29	0.20†	—	[0.00, 0.78]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.06	0.04†	—	[0.00, 0.16]‡	—	—	—
<i>Model summary: BPD/ASPD symptoms</i>	—	—	—	.001	<i>F</i> (6, 112) = 3.97	.42	.18
Direct Effects							
Maternal BPD/ASPD symptoms	2.46	1.41	1.75	.08	—	—	—
Child emotion regulation difficulties	0.17	0.05	3.22	.002	—	—	—
Indirect Effect							
Child emotion regulation difficulties	1.03	0.62†	—	[0.15, 2.63]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.07	0.04†	—	[0.01, 0.16]‡	—	—	—

Note. ASPD = antisocial personality disorder; BPD = borderline personality disorder. †Bootstrap-derived estimate of standard error of indirect effect, 1,000 bootstrapped samples. ‡95% bias-corrected lower-level and upper-level confidence interval, 1,000 bootstrapped samples.

tion when accounting for the same covariates (ASPD alone: $B = 2.27$, $SE = 0.98$, $p = .02$; BPD alone: $B = 1.76$, $SE = 0.80$, $p = .03$; BPD/ASPD composite: $B = 6.19$, $SE = 2.48$, $p = .01$). Next, the models were modified such that difficulties with emotion regulation served as a mediator between maternal personality disorder symptoms and child outcomes. The presence of a significant indirect effect indicated partial mediation of the relation between maternal BPD/ASPD symptoms and child outcomes by child emotion regulation difficulties.¹

1. Because child emotion dysregulation and child outcomes were measured concurrently, significant indirect effects do not preclude the possibility that causality actually flows in the opposite direction. To assess this possibility, we reran each model, switching the mediator and outcome variables (i.e., allowing child problems to mediate the relationship between maternal BPD/ASPD and child emotion regulation difficulties). These analyses produced a comparable pattern of significance. Results indicated significant indirect effects of child internalizing (1.77, 95% CI [0.14, 4.66]), externalizing (3.56, 95% CI [1.49, 7.29]), and total problems (3.23, 95% CI [1.24, 6.53]) on emotion regulation difficulties. Therefore, we cannot disprove the counterhypothesis that child behavior problems precede and lead to difficulties in emotion regulation.

TABLE 5. Results of Test for Child Emotion Regulation Difficulties as Mediator of the Relation Between Maternal BPD/ASPD Symptoms and Child Total Problems

	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>	<i>F</i> (<i>df</i> , <i>df</i>)	<i>R</i>	<i>R</i> ²
<i>Model summary: ASPD symptoms</i>							
Direct Effects							
Maternal ASPD symptoms	1.14	0.49	2.33	.02	—	—	—
Child emotion regulation difficulties	.20	0.05	4.24	< .001	—	—	—
Indirect Effect							
Child emotion regulation difficulties	0.45	0.23†	—	[0.08, 1.00]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.08	0.04†	—	[0.01, 0.16]‡	—	—	—
<i>Model summary: BPD symptoms</i>							
Direct Effects							
Maternal BPD symptoms	1.12	0.40	2.81	.006	—	—	—
Child emotion regulation difficulties	0.19	0.05	4.13	< .001	—	—	—
Indirect Effect							
Child emotion regulation difficulties	0.33	0.21†	—	[0.00, 0.86]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.07	0.04†	—	[0.00, 0.17]‡	—	—	—
<i>Model summary: BPD/ASPD symptoms</i>							
Direct Effects							
Maternal BPD/ASPD symptoms	3.62	1.26	2.87	.005	—	—	—
Child emotion regulation difficulties	0.19	0.05	4.02	<.001	—	—	—
Indirect Effect							
Child emotion regulation difficulties	1.15	0.63†	—	[0.17, 2.77]‡	—	—	—
Completely Standardized Indirect Effect							
Child emotion regulation difficulties	0.08	0.04†	—	[0.01, 0.17]‡	—	—	—

Note. ASPD = antisocial personality disorder; BPD = borderline personality disorder. †Bootstrap-derived estimate of standard error of indirect effect, 1,000 bootstrapped samples. ‡95% bias-corrected lower-level and upper-level confidence interval, 1,000 bootstrapped samples.

Child Externalizing Problems. Main effects models demonstrated significant positive relations between child externalizing problems and maternal BPD/ASPD personality symptoms, and between child externalizing problems and emotion regulation difficulties (see Table 3 and Figure 1). Maternal BPD/ASPD and depression symptoms were positively and significantly correlated (see Table 2), yet maternal depression was unrelated to child externalizing problems in the BPD alone and BPD/ASPD composite models. Maternal depression did predict child externalizing problems in the ASPD alone model ($B = 0.42$, $SE = 0.21$, $p = .046$). When child externalizing problems were regressed on maternal BPD/ASPD personality symptoms, maternal personality symptoms were related positively to child externalizing problems both directly and indirectly through child emotion regulation difficulties (see Table 3). These results are consistent with partial mediation. The model using the BPD/ASPD composite variable explained the most variance in child externalizing problems ($R^2 = .29$). The completely standardized indirect effect for this model was 0.08 (95% CI [0.01, 0.18]), which indicates that child emotion

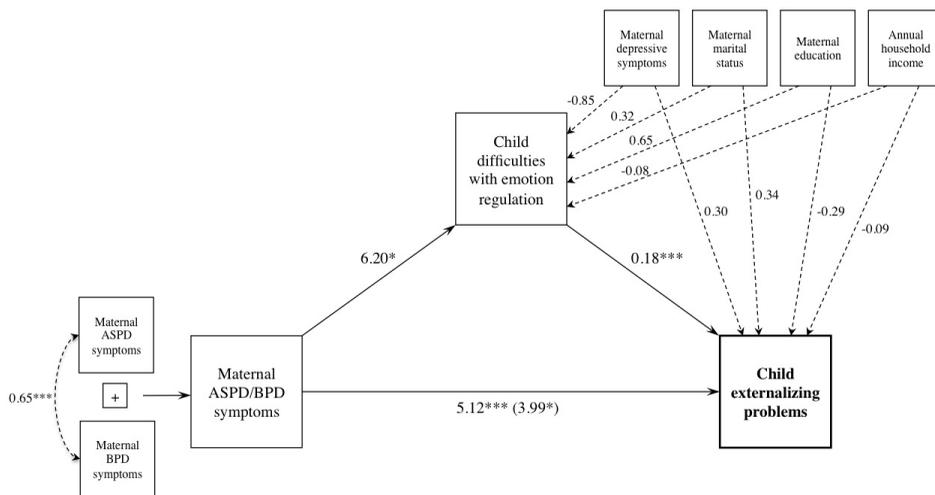


FIGURE 1. Results of regression analyses for mediation of maternal BPD symptoms and child externalizing problems by child emotion dysregulation. Path values represent unstandardized regression coefficients. The value outside of the parentheses represents the total effect of Year 2 maternal BPD symptoms on Year 3 child externalizing problems prior to the inclusion of Year 3 child emotion dysregulation as a mediating variable; the parenthetical value represents the direct effect, from bootstrapping analyses, after mediation. * $p < .05$. *** $p < .001$.

regulation difficulties indirectly confer an expected 0.07 standard deviation increase in child externalizing problems for each one standard deviation increase in maternal BPD/ASPD symptoms.

Child Internalizing Problems. Unmediated models indicated that child internalizing problems were significantly and positively associated with child emotion regulation difficulties (see Table 4 and Figure 2). Internalizing problems were also associated positively with maternal BPD personality symptoms and with the BPD/ASPD symptom composite variable. However, ASPD symptoms alone did not predict child internalizing problems ($B = 0.63$, $SE = 0.55$, $p = .26$). The relation between maternal depressive symptoms and child internalizing problems was not significant in any model. In all mediational models, child emotion regulation difficulties mediated the relation between maternal BPD/ASPD symptoms and child internalizing problems. When accounting for mediation by child emotion regulation difficulties, the direct effect of maternal personality symptoms on child internalizing problems no longer reached significance in the ASPD alone and BPD/ASPD composite models. These results suggest that child emotion regulation difficulties mediate the relation between maternal personality disorder symptoms and child internalizing problems. However, the direct effect of BPD symptoms alone remained significant ($B = 0.88$, $SE = 0.44$, $p = .049$), such that child emotion regulation difficulties only partially mediated the relation between maternal BPD symptoms and child internalizing problems. Overall, the BPD/ASPD

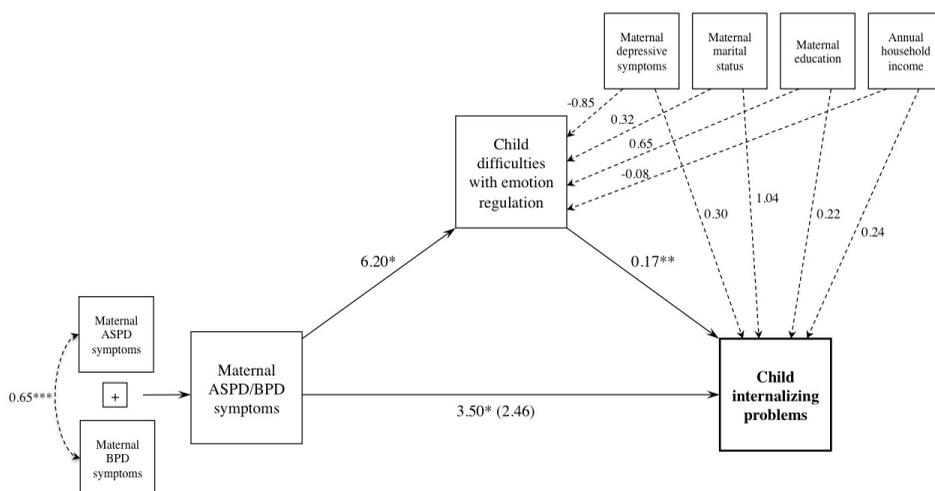


FIGURE 2. Results of regression analyses for mediation of maternal BPD symptoms and child internalizing problems by child emotion dysregulation. Path values represent unstandardized regression coefficients. The value outside of the parentheses represents the total effect of Year 2 maternal BPD symptoms on Year 3 child internalizing problems prior to the inclusion of Year 3 child emotion dysregulation as a mediating variable; the parenthetical value represents the direct effect, from bootstrapping analyses, after mediation. * $p < .05$. ** $p < .01$. *** $p < .001$.

composite model explained the most variance in child internalizing problems ($R^2 = .18$). The completely standardized indirect effect of maternal BPD/ASPD on child internalizing problems via child emotion regulation difficulties was 0.04 (95% CI [0.01, 0.16]).

Child Total Problems. Finally, we examined effects of maternal BPD/ASPD personality symptoms and child emotion regulation difficulties on child total problems (see Table 5 and Figure 3). As in previous analyses, maternal BPD/ASPD symptoms and child emotion regulation difficulties were both significantly and positively related to child total problems in unmediated models. Again, when considered with the other predictors in the model, we did not observe a significant association between maternal depression and child problems in any model. When child emotion regulation difficulties were included as a mediator, the effect of maternal personality symptoms on child problems, significant direct and indirect effects both emerged for all models, indicating partial mediation. The BPD/ASPD composite model explained the most variance in child total problems ($R^2 = .27$). The completely standardized indirect effect of maternal BPD/ASPD symptoms on child total problems via child emotion regulation difficulties was 0.04 (95% CI [0.01, 0.17]).

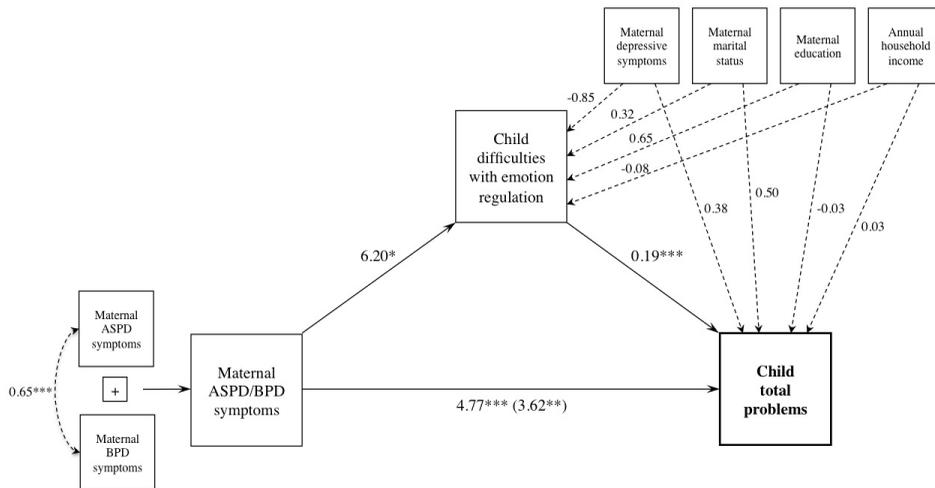


FIGURE 3. Results of regression analyses for mediation of maternal BPD symptoms and child total problems by child emotion dysregulation. Path values represent unstandardized regression coefficients. The value outside of the parentheses represents the total effect of Year 2 maternal BPD symptoms on Year 3 child problems prior to the inclusion of Year 3 child emotion dysregulation as a mediating variable; the parenthetical value represents the direct effect, from bootstrapping analyses, after mediation. * $p < .05$. ** $p < .01$. *** $p < .001$.

DISCUSSION

There is growing interest in understanding mechanisms through which parental psychopathology affects developmental outcomes of their children. In this study, we investigated the association between maternal BPD/ASPD symptoms and youth psychopathology through a proposed mediational pathway of child emotion regulation difficulties. This study was conducted with a relatively large, high-risk community sample. We found that maternal BPD/ASPD symptoms predicted child behavior problems assessed 1 year later both directly and indirectly through child emotion regulation difficulties (see Tables 3–5). Results are consistent with mediation between maternal BPD/ASPD symptoms and child internalizing problems and partial mediation between maternal BPD/ASPD symptoms and externalizing as well as total child psychopathology scores. These relations held even with maternal depression and other important contextual variables included as covariates. Furthermore, our composite score of maternal BPD/ASPD symptoms often accounted for more variance in child outcomes than BPD or ASPD alone. Our results highlight emotion regulation difficulties as one pathway through which mothers' psychopathology affects child outcomes. The results also demonstrate the importance of assessing maternal BPD/ASPD *symptoms* and not only their full diagnoses.

The association between maternal BPD symptoms and child emotion regulation is unsurprising given that (a) emotion dysregulation plays an inte-

gral role in the development and maintenance of BPD and is a hallmark feature of the disorder (Beauchaine et al., 2009; Linehan, 1993), and (b) studies find significant direct relations between mother and offspring emotion regulation difficulties (e.g., Kim, Pears, Capaldi, & Owen, 2009). Some have argued that emotion regulation difficulties are a key risk factor for antisocial behavior (e.g., Beauchaine & McNulty, 2013; Beauchaine, Gatzke-Kopp, & Mead, 2007); however, the role of these difficulties in the intergenerational transmission of externalizing symptoms is understudied. Based upon developmental theories, parents with poor emotion regulation, such as those with borderline and antisocial symptoms, may socialize similar problems in their children (Stepp et al., 2012) and/or pass down heritable vulnerabilities for psychopathology (see Beauchaine et al., 2009).

Several studies demonstrate that maternal BPD/ASPD symptoms are associated with compromised parenting (e.g., Hobson et al., 2009; Kiel, Gratz, Moore, Latzman, & Tull, 2011; Kim-Cohen et al., 2006; Newman et al., 2007; Zalewski et al., 2014). Poor parenting is one factor that contributes to emerging child emotion regulation difficulties (Eisenberg & Fabes, 1994; Eisenberg, Fabes, Carlo, & Karbon, 1992; Eisenberg, Fabes, & Murphy, 1996; Lunkenheimer, Shields, & Cortina, 2007), which may then contribute to child behavior problems (see, e.g., Beauchaine et al., 2009). This relatively straightforward trajectory may represent one pathway by which mothers with BPD/ASPD symptoms affect child outcomes.

Alternatively, child symptoms may be influenced more by heritable characteristics that also affect mothers' functioning negatively. Impulsivity and affective instability are two highly heritable vulnerabilities to BPD/ASPD, and to psychopathology more generally, with heritability coefficients around 80% and 50%, respectively (Krueger et al., 2002; Livesley & Jang, 2008; Livesley, Jang, & Vernon, 1998; Price, Simonoff, Waldman, Asherson, & Plomin, 2001; Widiger & Simonson, 2005). Twin research provides heritability estimates of around 40% for BPD (see Bornovalova, Hicks, Iacono, & McGue, 2009; Distel et al., 2008; Torgersen et al., 2008) and 80% for a general vulnerability to all externalizing disorders (Hicks, Krueger, Iacono, McGue, & Patrick, 2004). Furthermore, longitudinal twin research finds genetic effects of 71% and 72% of the stability of ASPD and BPD traits, respectively, from young through middle adulthood (Reichborn-Kjennerud et al., 2015). In our sample, shared genes may have influenced emotion regulation difficulties and behavioral problems observed across mothers and their offspring.

It is unlikely that any single genetic or environmental mechanism is wholly responsible for child psychopathology. Rather, the combination of causal influences, gene-environment correlations, and gene-environment interactions are probably functioning in tandem to produce child behaviors. Children's heritable characteristics or behaviors may evoke environmental risk. For example, sensitive, impulsive, and/or aggressive children may place strain on parents' emotional and financial resources, resulting in harsher treatment and/or neglect (Evans, Sibley, & Serpell, 2009). Gene-environment interactions such as poor mother-child match on important characteristics could result in mothers being unable to meet developmental needs specific to their offspring, and subsequent poor outcomes. In this case, child char-

acteristics (i.e., impulsivity, emotional sensitivity) could exacerbate maternal borderline symptoms or evoke the use of less optimal parenting behaviors. Interestingly, Zalewski and colleagues (2014) investigated whether adolescent negative emotionality and low self-control interacted with maternal BPD symptoms to predict parenting. Although indices of youth temperament were related to compromised parenting outcomes, adolescent temperament and maternal BPD symptoms did not interact to predict parenting. These findings suggest that the influence of maternal BPD symptoms on parenting was not exacerbated by adolescent daughters' low self-control and negative emotionality. Rather, maternal emotion dysregulation was the best predictor of poor parenting outcomes. Twin research and gene-environment studies of BPD, impulsivity, emotional sensitivity, aggression, and anger expression each highlight joint roles of genetic and environmental contributions (see Carpenter, Tomko, Trull, & Boomsma, 2013, for a review). Further research is needed to tease apart these potential pathways.

There are several limitations to the current study. First, although the study was conducted longitudinally and mothers' BPD/ASPD symptoms explained a portion of the variance in child outcomes 1 year later, we cannot establish precedence of mothers' BPD/ASPD symptoms prior to child emotion regulation difficulties or the emergence of poor behavioral outcomes. Child emotion regulation difficulties and behavior problems were also both assessed at the same time point, although by different reporters (self-report for the DERS and mother report on the CBCL). This limits the interpretability of our results and prohibits any claims regarding directions of observed effects. It is impossible to be certain that child emotion regulation difficulties are truly the mediator, rather than an outcome, of child behavior problems. Indeed, results were still statistically significant when flipping the mediator and the dependent variable (see footnote 1), suggesting that only additional longitudinal research can address questions of causality. However, substantial evidence has accumulated showing that child emotion regulation difficulties are a developmental precursor to many behavior problems (see, e.g., Crowell, Puzia, & Yaptangco, 2015; De Caluwé, Decuyper, & De Clercq, 2013). Therefore, others' theoretical and empirical work supports our designation of emotion regulation difficulties as the mediator in our models.

Second, although we accounted for maternal depression, education level, marital status, and annual income in our models, many other potential influences on child emotion regulation and psychopathology were not included for both statistical and theoretical reasons. For example, we did not include every potential diagnostic comorbidity as a covariate in our mediational models, in spite of evidence that many diverse forms of maternal psychopathology affect child emotion regulation (Beauchaine et al., 2009; Kober, 2014; Shaw, Stringaris, Nigg, & Leibenluft, 2014). Including maternal major depressive, BPD, and ASPD symptoms likely captures both internalizing and externalizing risk without the statistical limitations inherent in including numerous covariates. Measuring key transdiagnostic symptoms such as those captured by the MDD, BPD, and ASPD interviews (e.g., emotional lability, impulsivity, aggression, difficulty concentrating) is more consistent with the National Institute of Mental Health's (2011) research domain criteria

(RDoC) than measuring every internalizing/externalizing diagnosis individually. A third limitation is the potential influence on our results of having the same reporter for multiple measures. Mothers reported on their own symptoms via clinical interview as well as their children's symptoms on the CBCL 1 year later. Although maternal reports informed measures for our independent and dependent variables, the methodology by which the data were collected and the timing were distinct. Thus, the effect of shared reporter variance is less likely to have significantly influenced our results.

Finally, paternal data were largely unavailable and therefore not assessed. Much of the research on parental BPD focuses on mothers rather than fathers (Macfie, 2009), despite evidence that the sex distribution of this disorder is more equal than was once thought (Grant et al., 2008). Neglecting to assess the potential influence of paternal BPD/ASPD on child outcomes may contribute to disproportionate blame and stigma falling on mothers with mental health problems. Despite these drawbacks to focusing solely on maternal-child dynamics, mothers are often the primary caregiver and are more likely to be the only caregiver in single-parent households (Seifer & Dickstein, 2000). Thus, mothers generally have more opportunities to interact and model behaviors for their children.

A notable strength of the current study was its broad inclusion criteria. Our findings are likely to generalize more broadly than if we had excluded mothers who failed to meet full BPD or ASPD criteria. The data presented here indicate that maternal BPD/ASPD symptoms, even those that are sub-threshold, are associated with child emotion regulation difficulties. These difficulties contribute to child behavior problems even within a community sample and are not better explained by maternal mood disturbance or other relevant contextual factors such as marital status, income, or maternal education level. Future studies should examine whether relations between child psychopathology, child emotion regulation difficulties, and maternal BPD/ASPD symptoms are more robust among families with mothers who meet full diagnostic criteria. Our results point to the importance of understanding how risk is promoted and maintained within families. Emotion dysregulation appears to be a promising target for preventing the intergenerational transmission of psychopathology.

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