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RECIPROCAL INFLUENCES OF PARENT AND ADOLESCENT BORDERLINE PERSONALITY SYMPTOMS OVER 3 YEARS

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Leading etiological theories implicate the family environment in shaping borderline personality disorder (BPD). Although a substantive literature explores familial aggregation of this condition, most studies focus on parent influence(s) on offspring symptoms without examining youth symptom influence on the parent. The current study investigated reciprocal relations between parent and adolescent BPD symptoms over time. Participants were 498 dyads composed of urban-living girls and their parents enrolled in a longitudinal cohort study (Pittsburgh Girls Study). The authors examined BPD severity scores assessed yearly when youth were ages 15–17 years in a series of cross-lagged panel models. After controlling for autoregressive effects, a measure of parent–child conflict, and an indicator of socioeconomic status, evidence of parental influence on adolescent symptoms did not emerge. However, adolescent BPD symptoms at age 16 predicted greater parent BPD symptoms at age 17 above the influence of depression. Results highlight the importance of considering the influence of youth BPD on parental symptoms.

Keywords: borderline personality disorder, adolescents, parent–child relations, developmental psychopathology

Borderline personality disorder (BPD) is a serious mental disorder characterized by interpersonal turmoil, severe emotion dysregulation, and impulsivity (American Psychiatric Association [APA], 2013). Most etiological models claim that BPD is shaped through social processes, and many models place particular emphasis on familial mechanisms (see Winsper, 2018, for a review). This follows from evidence that vulnerability to BPD often aggregates among families (Distel et al., 2010; Jang, Livesley, Vernon, & Jackson, 1996; Livesley, Jang,

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& Vernon, 1998), and maladaptive parent–youth transactions often increase BPD risk (i.e., by reinforcing emotional lability and impulsive behavior, and by disrupting social and self-regulatory skill development; Crowell, Beauchaine, & Linehan, 2009; Frankel-Waldheter, Macfie, Strimpfel, & Watkins, 2015; Gratz et al., 2014; Jensen, Dumontheil, & Barker, 2014; Musser, Zalewski, Stepp, & Lewis, 2018).

Given the importance of parental scaffolding and effective coregulation for youth development (see, e.g., Hughes, Crowell, Uyeji, & Coan, 2012), much of the available literature focuses on parent-to-child influence. Offspring of mothers with BPD have compromised socioemotional development, higher rates of internalizing and externalizing psychopathology, poorer psychosocial functioning, and increased risk for developing BPD themselves compared to children of mothers without BPD (Barnow, Spitzer, Grabe, Kessler, & Freyberger, 2006; Eyden, Winsper, Wolke, Broome, & MacCallum, 2016; Herr, Hammen, & Brennan, 2008; Macfie, 2009; Stepp, Whalen, Pilkonis, Hipwell, & Levine, 2012; Weiss et al., 1996). Data from the Pittsburgh Girls Study (PGS), a longitudinal cohort study of urban girls and their caregivers, have linked caregiver BPD to parenting difficulties and poor parent–child relationship quality (Hallquist, Hipwell, & Stepp, 2015; Stepp et al., 2014), and demonstrated that maternal affective and behavioral dysregulation have the strongest effects on parenting behaviors (Zalewski et al., 2014). Evidence from the PGS also suggests a reciprocal association between punitive parenting practices and BPD symptoms during adolescence (Stepp et al., 2014). Specifically, youth impulsivity, youth negative affectivity, and caregiver psychopathology were each related to parenting trajectories (e.g., low parental warmth, emotional invalidation, rejection); *and* the developmental trajectories of BPD symptoms and parenting were moderately associated, suggesting a reciprocal relationship. Taken together, available evidence indicates that offspring of affected parents are particularly vulnerable to a range of problematic outcomes, and parents of affected youth may struggle with effective caregiving. However, no studies have examined the influence of youth BPD symptoms on parental BPD over time.

PGS data have also demonstrated that positive familial interactions can buffer against youth BPD, further supporting developmental theories that emphasize how the parent–child relationship can influence the severity and manifestation of the disorder over time (e.g., Dixon-Gordon, Whalen, Scott, Cummins, & Stepp, 2016; Whalen et al., 2014). For example, Whalen and colleagues (2014) found that positive dyadic behaviors during a mother–daughter conflict discussion task (i.e., satisfaction and positive escalation) were associated with decreases in adolescent girls' BPD severity scores over time. In contrast, dyadic negative escalation (i.e., conflict and/or negative affect from one partner followed by negative behavior from the other partner) was associated with higher BPD severity scores among girls. These findings suggest that the familial context can function as an important protective factor in shaping the course of BPD during adolescence and may be a valuable target in assessment, intervention, and prevention efforts.

Previous research has bolstered efforts to better understand and prevent intergenerational transmission of BPD (ClinicalTrials.gov, 2017). However,

most studies have been limited by focusing exclusively on unidirectional parent influence(s) (i.e., parental BPD symptoms or compromised parenting) on youth symptoms. Risky relational processes are bidirectional in nature—meaning that youth behavior also has an evocative effect on their social environment (Beauchaine, Gatzke-Kopp, & Mead, 2007; Snyder & Patterson, 1995; Winsper, 2018). Although many assume that a parent has a greater influence on his or her offspring than vice versa, both individuals are being shaped through their interactions (Holmbeck, Paikoff, & Brooks-Gunn, 1995; Noller, 1995; Sroufe & Rutter, 1984).

To our knowledge, no prior work has examined whether youth BPD symptoms influence parental BPD expression or severity. Parents of youth who manifest BPD features are likely to share some degree of underlying vulnerability (Distel et al., 2010; Jang et al., 1996; Livesley et al., 1998)—even if they have not developed the clinical disorder themselves. Parental BPD risk may increase, and any existent symptoms may worsen as a function of increases in youth symptoms. Research indicates that offspring psychopathology escalates parental stress (Douma, Dekker, & Koot, 2006), and youth with temperamental vulnerability to negative affectivity are more challenging to parent effectively (e.g., Papoušek & von Hofacker, 1998). Thus, vulnerable parents and youth alike are expected to be affected by BPD in their interaction partner.

The present study addresses important gaps in the literature by examining reciprocal effects of child–parent BPD symptoms during adolescence. This age period is when a constellation of maladaptive BPD features often coalesce and first reach diagnostic threshold (APA, 2013). Furthermore, adolescence is also often marked by role transitions and negotiations, increasing youth independence, and decreasing parental involvement, monitoring, and control—making it a particularly dynamic and often stressful time (Masche, 2010; Phinney, Kim-Jo, Osorio, & Vilhjalmsdottir, 2005; Saphir & Chaffee, 2002; Williams, 2003). By examining parent–adolescent dyads during this critical developmental period, we can determine whether and how adolescent and parent BPD features predict each other over time, controlling for prior levels of each individual's symptoms. An increase in parental symptoms due to adolescent BPD expression would highlight a potentially unmet need for parental/caregiver support and would provide further empirical evidence for the importance of family-level BPD intervention, regardless of which family member exhibits the most pronounced BPD symptomatology. We hypothesize that reciprocal effects will emerge whereby parental BPD will predict adolescent BPD symptoms longitudinally and vice versa.

METHOD

SAMPLE DESCRIPTION

Participants were drawn from an urban community sample of girls and their primary caregivers who were enrolled in a longitudinal cohort study (PGS). Girls were initially enrolled into four age cohorts at 5, 6, 7, and 8 years and were followed annually according to an accelerated longitudinal design. Low-income neighborhoods were oversampled, such that neighborhoods in which

at least 25% of families were living at or below poverty level were fully enumerated, and a random selection of 50% of households in all other neighborhoods were enumerated (see Hipwell et al., 2002, and Keenan et al., 2010, for details on study design and recruitment). This sampling strategy is ideal for examination of BPD and its effects, as longitudinal research shows that low socioeconomic status has a significant influence on level of BPD symptoms, even above the effects of other risk factors such as trauma history, stressful recent life events, and poor parenting (Cohen et al., 2008). Thus, individuals from low-income backgrounds are at particularly high risk.

Adolescent and parent BPD symptoms were assessed annually beginning in Wave 11 of the PGS study. In this assessment wave, Cohort 5 girls were age 15, Cohort 6 were age 16, Cohort 7 were age 17 and Cohort 8 were age 18. Parent interviews ended when the girls reached 18 years of age. Thus, in order to examine reciprocal associations between adolescent and parental BPD symptoms across multiple adolescent ages (ages 15–18 years), analyses were limited to dyads recruited as part of Cohort 5 ($N = 588$).

Participant retention in Cohort 5 was high; 87.10% in Wave 11, and 84.69% in Waves 12 and 13. Because of the use of maximum likelihood methods to estimate missing data (see Data Analytic Plan, below), dyads could be included in the foregoing analyses as long as data were provided for at least one assessment wave ($n = 523$ dyads). Dyads were then removed from analysis if there was evidence that the participating caregiver changed across the assessments at ages 15, 16, and 17, either due to changes in relationship to adolescent, or implausible increases (or decreases) in caregiver age. This applied to 25 dyads, leaving a total sample size of 498.

More than half of the adolescents were identified by their primary caregivers as Black (52.21%), while 41.97% were identified as White, 4.42% as more than one race, and 1.41% as Asian American (see Table 1). The vast majority of caregivers were biological mothers (84.94%) or fathers (6.22%), followed by adoptive mothers or foster mothers (2.81%) and grandmothers (2.61%). Less frequent relationships among caregivers and girls included stepfathers (0.20%) and other female relatives, such as aunts (0.60%). Given that the overwhelming majority of the caregivers were parents, we refer to caregivers as "parents." In Wave 11, 51.61% of parents were cohabiting with a spouse or domestic partner. Parents' ages at the Wave 11 assessment ranged from 28 to 73 years ($M = 43.07$, $SD = 8.11$).

DATA COLLECTION

Separate in-home interviews for both the girl and the parent were conducted annually by trained interviewers using a laptop computer. All study procedures were approved by the University of Pittsburgh Institutional Review Board. Families were compensated for their participation.

MEASURES

BPD Symptoms. Girls and parents self-reported on their BPD symptoms annually using questions from the screening questionnaire of the Fourth Edition

TABLE 1. Descriptive Characteristics for Included PGS Dyads (Cohort 5 Only, *n* = 498)

Retention*	***n* (%)**
Wave 11 (Age 15)	512 (87.10)
Wave 12 (Age 16)	498 (84.69)
Wave 13 (Age 17)	498 (84.69)
Child Racial Identity	***n* (%)**
African American/Black	260 (52.21)
White/Non-Hispanic White	209 (41.97)
Asian American/Asian	7 (1.41)
Biracial or multiracial	22 (4.42)
Caregiver Relationship	***n* (%)**
Biological mother	423 (87.22)
Biological father	31 (6.39)
Adoptive or foster mother	14 (2.89)
Grandmother	13 (2.68)
Stepfather	1 (0.21)
Other female relative	3 (0.62)
Caregiver Characteristics	***n* (%) or *M* (*SD*)**
Age	43.07 (8.11)
Less than HS/GED	55 (11.36)
HS diploma/GED	136 (28.10)
More than HS diploma/GED	293 (60.54)
Household Characteristics	***n* (%)**
Age 15 receipt of public assistance	196 (40.41)
Age 16 receipt of public assistance	198 (41.86)
Age 17 receipt of public assistance	192 (41.03)
Age 15 single parent household	241 (49.90)
Age 16 single parent household	228 (48.10)
Age 17 single parent household	236 (50.43)

Note. *Retention refers to the percentage of dyads where one or both girl and/or parent report were available.

version of the International Personality Disorders Examination (IPDE-BOR; Loranger et al., 1994). The IPDE-BOR consists of 10 true or false items (e.g., "I get into very intense relationships that don't last"). Although originally developed for adults, adequate concurrent validity and sensitivity and specificity of BPD symptom scores to clinicians' diagnosis have been demonstrated for the IPDE-BOR in a sample of youth (Smith, Muir, & Blackwood, 2005). We excluded one item, phrased "I show all my feelings for the world to see," which did not clearly map onto the nine BPD criteria. A score equal to or greater than 4.0 is considered clinically significant, experiencing distress and/or impairment (see Table 2 for participant scores by wave). Internal consistency for BPD symptoms was good for both parent (α = .71, α = .72, and α = .70) and adolescent (α = .70, α = .71, α = .72) in Waves 11 to 13, respectively. We previously demonstrated convergent validity of the IPDE-BOR with

BPD symptom severity scores from a semistructured clinical interview (the Structured Interview for *DSM-IV* Personality Disorders; Pfohl, Blum, & Zimmerman, 1997; $r = .71$, $p < .001$) in a subsample of the PGS ($n = 65$). Parent and adolescent BPD symptoms are denoted by the abbreviations P-BPD and A-BPD, respectively.

Depressive Symptoms. In order to determine whether any transgenerational effects of BPD were unique to this disorder or apply more generally to other types of psychopathology, we examined the association of parent and adolescent depressive symptoms over the same time period. Depression commonly emerges among those with BPD (see Zanarini et al., 2019) and shares some associated features (e.g., emotion dysregulation, self-harm; Abravanel & Sinha, 2015; Ballou et al., 2019; Eldesouky, Thompson, Oltmanns, & English, 2018;

TABLE 2. Characteristics of BPD Symptoms for Across Waves (Cohort 5 Only, *n* = 498)

Adolescent BPD Symptoms Total	**Age 15**	**Age 16**	**Age 17**
M (*SD*)	2.31 (1.81)	2.18 (1.80)	2.01 (1.77)
Range	0–9	0–9	0–9
Clinically significant, *n* (%)	111 (23.13)	106 (22.60)	86 (18.38)
Child BPD–Specific Symptoms, *n* (%)	**Age 15**	**Age 16**	**Age 17**
Impulsivity	194 (40.42)	131 (27.93)	139 (29.70)
Unstable relationships	67 (13.96)	66 (14.07)	58 (12.39)
Self-harm/suicide	46 (9.58)	42 (8.96)	41 (8.76)
Emptiness	65 (13.57)	71 (15.14)	68 (14.53)
Anger	166 (34.58)	155 (33.05)	138 (29.49)
Affective instability	212 (44.17)	211 (44.99)	195 (41.67)
Paranoia/dissociation	95 (19.79)	95 (20.26)	67 (14.32)
Abandonment fears	32 (6.67)	32 (6.84)	27 (5.78)
Identity disturbance	140 (29.17)	125 (26.65)	130 (27.84)
Parent BPD Symptoms Total	**Age 15**	**Age 16**	**Age 17**
M (*SD*)	1.89 (1.79)	1.96 (1.89)	1.81 (1.82)
Range	0–8	0–9	0–8
Clinically significant, *n* (%)	75 (15.69)	85 (18.16)	68 (14.85)
Parent BPD–Specific Symptoms, *n* (%)	**Age 15**	**Age 16**	**Age 17**
Impulsivity	123 (25.95)	126 (27.27)	107 (23.46)
Unstable relationships	51 (10.81)	46 (10.00)	41 (8.95)
Self-harm/suicide	160 (33.47)	162 (34.76)	162 (35.22)
Emptiness	74 (15.35)	75 (16.09)	60 (13.10)
Anger	77 (16.08)	73 (15.67)	69 (14.97)
Affective instability	121 (25.37)	138 (29.55)	123 (26.68)
Paranoia/dissociation	47 (9.81)	58 (12.37)	34 (7.41)
Abandonment fears	28 (5.82)	35 (7.49)	27 (5.90)
Identity disturbance	96 (20.13)	99 (21.29)	96 (20.96)

Note. Clinically significant scores are values of 4 or greater.

Zubrick et al., 2017). Adolescents' depressive symptoms (hereafter referred to as A-DEP) were assessed using the Adolescent Symptom Inventory–Fourth Edition (Gadow & Sprafkin, 1998). This interviewer-administered set of items examines the nine symptoms of major depressive disorder, each rated on a 4-point scale from 0 (*never*) to 3 (*very often*). The sum score of these items was used as a dimensional measure of depression severity among girls. Internal consistency of the summed score was high for all ages (age 15 α = .81, age 16 α = .81, age 17 α = .88).

Parent depressive symptoms (hereafter referred to as P-DEP) were assessed by self-report using the Beck Depression Inventory, 2nd Edition (BDI-II; Beck, Steer, Ball, & Ranieri, 1996). The BDI-II is a commonly used measure of depressive symptoms among adults. Internal consistency reliability in this sample was high (age 15 α = .93, age 16 α = .94, age 17 α = .93).

Covariates. We covaried household receipt of public assistance (e.g., Medicaid, food stamps) at each age as an indicator of socioeconomic status (0 = no public assistance, 1 = receipt of public assistance). We also examined the role of parent–child conflict as a potential contributor to transmission of BPD symptoms across time. Parent–child conflict was assessed by child- and parent-report on the Conflict Tactics Scale–Parent Child Version (CTSPC; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). The CTSPC measures parental discipline, as well as parents' psychological and physical maltreatment and neglect toward their children. Thirty-five questions were phrased as how often did the primary caregiver use harsh punishment on a 3-point scale (1, *almost never*, to 3, *often*). A composite score was calculated for each dyad at each wave based on both parent and child report. The maximum score across reporters was taken for each item, and these scores were then summed to create a composite index of parent–child conflict, which showed adequate internal consistency (age 15 α = .54, age 16 α = .59, age 17 α = .60).

DATA ANALYTIC PLAN

A cross-lagged panel model was tested to model the changes in BPD symptoms and depressive symptoms from ages 15 to 17 years. In this model, adolescent and parent BPD symptoms were allowed to covary at each assessment time point, as were adolescent and parent depressive symptoms. Auto-regressive effects for both BPD and depression within each individual were also modeled over time. In addition, cross-lagged effects were tested, such that earlier parent BPD or depressive symptoms predicted subsequent adolescent BPD or depressive symptoms, and vice versa. By including auto-regressive effects, the cross-lagged effects can be interpreted as the effect of each dyad member on the other dyad, *above and beyond* their own symptoms assessed at the prior time point. We also allowed for cross-lagged cross-disorder effects, such that earlier adolescent depressive symptoms could influence subsequent parent BPD symptoms, and vice versa. Household receipt of public assistance and parent–child conflict were modeled as predictors of parent and adolescent BPD symptoms and depressive symptoms at the same age (assessment wave) and were allowed to covary over time.

Analyses were conducted in Mplus version 8.2 (Muthén & Muthén, 2017) using Bayesian Markov chain Monte Carlo (MCMC) estimation, which provides more stable parameter estimates than other methods (Ozechowski, 2014). Variable prior parameters were estimated based on median values from observed data. The model converged with fewer than 5,000 iterations, meaning the potential scale reduction factor (PSR) value approached 1 (PSRs < 1.003). The model was subsequently rerun with 50,000 iterations to ensure that PSR values did not increase. The final model had a final PSR value below 1.001, indicating satisfactory convergence. In Bayesian estimation, exact *p* values are not available; statistical significance is evaluated on the basis of whether the 95% credible interval (CI) for each parameter includes 0.

BPD symptom severity scores, depression severity scores, and parent–child conflict scores were grand-mean centered for each participant at each age. Models included estimates of variances for all observed variables, which allowed missing data on these variables to be estimated using maximum likelihood procedures. All results are reported as standardized effects (betas) unless otherwise specified.

RESULTS

CONCURRENT ASSOCIATIONS OF BPD AND DEPRESSIVE SYMPTOMS

There was no concordance between P-BPD and A-BPD severity scores when assessed concurrently at any age (see Table 3 for concurrent effects), nor between P-DEP and A-DEP scores at any age. This may be a function of the amount of variance explained by within-person stability. Receipt of public assistance at age 15 was associated with greater P-BPD scores, A-BPD scores, and P-DEP scores, but not A-DEP scores at the same age. Parent–child conflict at age 15 was also associated with higher P-BPD, A-BPD, P-DEP, and A-DEP scores. At age 16, receipt of public assistance was associated with higher A-BPD scores, but no other psychopathology measures, whereas parent–child conflict at age 16 was associated with greater A-BPD, P-BPD, and A-DEP scores (but not P-DEP scores). At age 17, receipt of public assistance was associated with greater P-BPD scores, and parent–child conflict was associated with greater A-BPD scores, but neither covariate had a significant effect on depression scores.

WITHIN-INDIVIDUAL EFFECTS OVER TIME

As expected, there was significant year-to-year stability in self-reported symptom severity for A-BPD scores (age 15 to 16 $B = 0.49$, 95% CI [0.41, 0.57]; age 16 to 17 $B = 0.51$, 95% CI [0.44, 0.58]) and P-BPD scores (age 15 to 16 $B = 0.63$, 95% CI [0.56, 0.71]; age 16 to 17 $B = 0.59$, 95% CI [0.52, 0.66]). This was also true for A-DEP scores (age 15 to 16 $B = 0.55$, 95% CI [0.48, 0.62]; age 16 to 17 $B = 0.50$, 95% CI [0.42, 0.58]) and P-DEP scores (age 15 to 16 $B = 0.71$, 95% CI [0.63, 0.79]; age 16 to 17 $B = 0.59$, 95% CI [0.53, 0.66]).

TABLE 3. Model Results for Concurrent Effects (Within Assessment Wave)

Variable by Age	*B* [95% CI]	*B* [95% CI]	*B* [95% CI]	*B* [95% CI]
Age 15	**P-BPD**	**A-BPD**	**P-DEP**	**A-DEP**
Public assistance	0.16 [0.08, 0.25]	0.11 [0.02, 0.20]	0.19 [0.10, 0.28]	0.06 [-0.03, 0.15]
P-C conflict	0.26 [0.17, 0.34]	0.23 [0.14, 0.32]	0.15 [0.06, 0.24]	0.19 [0.10, 0.28]
A-BPD	0.02 [–0.07, 0.11]			
A-DEP			0.01 [–0.08, 0.10]	
Age 16	**P-BPD**	**A-BPD**	**P-DEP**	**A-DEP**
Public assistance	0.004 [–0.07, 0.08]	0.09 [0.01, 0.17]	0.06 [–0.02, 0.13]	0.06 [–0.02, 0.14]
P-C conflict	0.11 [0.03, 0.18]	0.18 [0.11, 0.26]	0.07 [0.00, 0.14]	0.13 [0.05, 0.21]
A-BPD	–0.01 [–0.11, 0.09]			
A-DEP			0.09 [–0.01, 0.18]	
Age 17	**P-BPD**	**A-BPD**	**P-DEP**	**A-DEP**
Public assistance	0.10 [0.03, 0.17]	0.03 [–0.05, 0.11]	0.04 [–0.04, 0.11]	–0.06 [–0.14, 0.03]
P-C conflict	0.03 [–0.05, 0.10]	0.08 [0.002, 0.16]	0.09 [0.02, 0.16]	0.07 [–0.01, 0.15]
A-BPD	0.01 [–0.09, 0.11]			
A-DEP			0.06 [–0.04, 0.15]	

Note. Presented values are standardized betas and 95% credibility intervals (CIs) for these results. P-BPD: parent's borderline personality disorders symptoms; A-BPD: adolescents' borderline personality disorders symptoms; P-DEP: parents' depressive symptoms; A-DEP: adolescents' depressive symptoms; P-C conflict: parent–child conflict.

CROSS-LAGGED EFFECTS OVER TIME

The primary aims of this model are to test the extent to which parental BPD and depressive symptoms influence subsequent adolescent BPD and depressive symptoms, and vice versa, controlling for auto-regressive effects within each individual and the same-time covariation of symptoms and covariates assessed concurrently.

A-BPD scores at ages 16 and 17 were not significantly predicted by P-BPD or P-DEP scores at ages 15 and 16, respectively. This was also true for A-DEP scores, which were not influenced by prior-year P-BPD or P-DEP scores over either interval. Thus, no indicator of parental psychopathology predicted subsequent adolescent psychopathology, controlling for covariate and auto-regressive effects. Furthermore, P-BPD and P-DEP scores at age 16 were not predicted by A-BPD or A-DEP scores at age 15. However, A-BPD score at age 16 explained significant variance in P-BPD scores at age 17 (B = 0.09, 95% CI [0.002, 0.17]). P-BPD score at age 17 was not associated with A-DEP score at age 16. For a visual depiction of these effects, see Figure 1.

DISCUSSION

To our knowledge, this is the first study to explore reciprocal influences of parent and adolescent BPD symptoms longitudinally. We included parent and youth depressive symptoms in our models to assess if any parent/adolescent influence was attributable to BPD specifically, as compared with

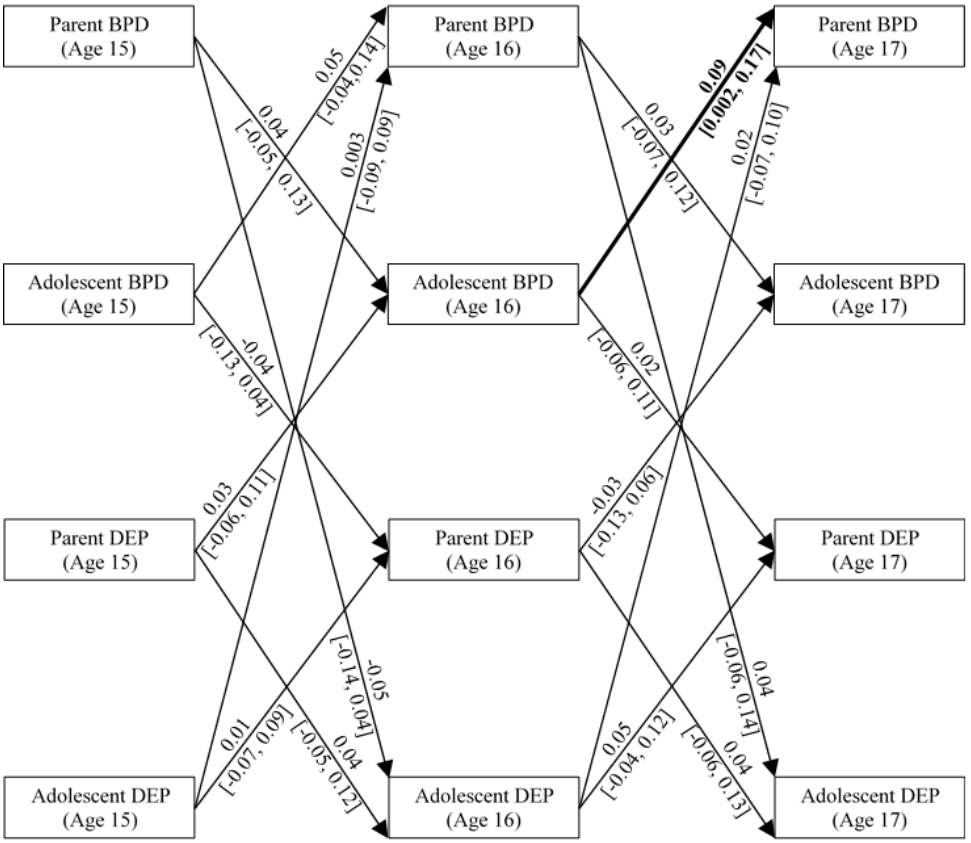

FIGURE 1. Model results for cross-lagged effects. Bolded arrows and values were statistically significant. All values are standardized. Effects of covariates (receipt of public assistance and parent–child conflict) and concurrent covariances within a particular age are not represented here for ease of interpretation (see Table 3 for details on these results).

psychopathology more broadly. Consistent with the developmental psychopathology perspective (Sroufe & Rutter, 1984), we investigated whether parent and adolescent symptoms prospectively predict and influence one another. Although others have attended to the effects of parental BPD (typically maternal) on parenting, attachment quality, and adolescent outcomes (e.g., Hallquist et al., 2015; Lyons-Ruth, Riley, Patrick, & Hobson, 2019; Stepp et al., 2014), work investigating youth-to-parent pathways is largely absent. Our findings revealed that adolescent BPD symptoms influenced parental BPD symptoms 1 year later above and beyond the effects of depression, socioeconomic status, and parent–child conflict.

Although etiological theorists describe interactions between adolescent temperament and parental behavior as contributing to BPD (e.g., Linehan, 1993), we did not find evidence of parental influence on adolescent BPD or depressive symptoms when accounting for measures of parent–child conflict and socioeconomic status. This result was surprising and contrary to our

hypotheses. When considered in the context of research showing that parental BPD influences youth earlier in the developmental trajectory, our results may be capturing the latter portion of a developmental turn-taking effect (whereby the parent influences youth symptoms, which in turn leads adolescent to influence parent). The lack of observed parental symptom influence during adolescence may be a by-product of normative developmental shifts. Although we examined a narrow developmental window, potential influence of parental BPD symptom severity on adolescents may diminish as a function of adolescents' increasing autonomy. For example, adolescents become eligible for a driver's licenses at age 16, and minors in Pennsylvania can begin working longer hours at a wider variety of jobs (e.g., cook, lifeguard, server in a restaurant without alcohol; Pennsylvania Department of Labor & Industry, 2012). Peer and romantic relationships become increasingly important as youth progress through adolescence, whereas interactions with parents generally decrease (Carver, Joyner, & Udry, 2003; Csikszentmihalyi & Larson, 1984; Furman & Buhrmester, 1992; Meier & Allen, 2009). Studies that assesses time spent within the home and conflict over adolescent autonomy are needed, in addition to research that examines reciprocal effects over a longer developmental time period.

Importantly, *adolescent* BPD symptom severity influenced parent scores from ages 16 to 17, yet not from ages 15 to 16. This pattern is particularly interesting in light of the fact that the overall percentage of youth reporting symptoms in the clinically significant range *decreased* over time. Thus, although fewer participants were reporting clinically significant symptoms by age 17, adolescent symptom influence on parents was the strongest at this time point. Further research is needed to examine why parents may be affected by their adolescent's symptoms at this age.

In line with a developmental conceptualization, it may be that parents with BPD features are more likely to experience stress associated with renegotiating familial roles and expectations, which typically increase during later adolescent years (Bulcroft, Carmody, & Bulcroft, 1996). However, we did not measure processes related to role transitions, and it is unclear whether associated changes from age 16 to 17 are pivotal enough to explain our results. A related, yet distinct explanation for the increased influence of adolescent symptoms on parents in later adolescence is that symptoms of BPD such as impulsive and reckless behavior may become more intense and/or problematic as adolescents become more independent and parental monitoring decreases (e.g., drug and alcohol use, early sexual behavior; Hayatbakhsh et al., 2008; Jackson & Schulenberg, 2013; Latendresse et al., 2008; Lenciauskiene & Zaborskis, 2008). In this sample, rates of endorsement of the BPD impulsive behavior criterion generally *decreased* among adolescents over time (see Table 2); however, it is possible that the *severity* of impulsive behaviors among those youth who continued to endorse this criterion changed in ways that we were unable to capture using a single yes/no indicator. It is also likely that adolescents' self-reported symptoms do not map neatly onto parental perceptions of their child's symptoms, such that parents may view behaviors as more problematic or intense over time, even as adolescents themselves view the same behaviors as less impulsive or reckless. Further research is needed to

investigate these questions, for instance, by examining whether some specific symptoms of BPD exhibit stronger cross-dyad effects than others.

This study has several limitations. Although we examined a relatively large and diverse sample, adolescents were exclusively female and BPD symptoms were assessed only when adolescents were between ages 15 and 17. While BPD typically reaches diagnostic threshold during adolescence, examining features of the condition and their influence on parent pathology earlier in development would significantly enhance our understanding of the emergence and maintenance of BPD. There are also many potential influences on BPD symptoms and relevant confounds that we were not able to examine in the current model, such as indices of parent stress, child exposure to bullying, and parental exposure to domestic violence. Although we have hypotheses regarding potential mediators that may aid in explaining the developmental effects we observed (e.g., degree of adolescent autonomy, relationship dynamics between the parent and adolescent), we did not have independent or dyadic measurements of these variables to include in our current work. Now that we have observed adolescent-to-parent influence on BPD severity, each of these topics is a ripe area for further inquiry. Finally, we wish to highlight that the largest effect sizes in our model are the auto-regressive effects of parental BPD symptoms and parental depressive symptoms, followed by adolescent BPD symptoms and adolescent depressive symptoms. The magnitude of our significant cross-lagged effect of adolescent BPD symptoms at age 16 influencing parental BPD symptoms at age 17 should be considered small, which reinforces the need to replicate youth-to-parent BPD influence in future samples.

Our results have implications for intervention. Effective treatment programs exist for both adults and adolescents with BPD (e.g., dialectical behavior therapy [DBT]; Fleischhaker et al., 2011; Linehan, 1993). Although helpful, standard approaches and family-facilitated education-based interventions typically do not treat families as a whole. Within DBT programs for adolescents, a parent is often invited to accompany the child to a skills group (Rathus & Miller, 2015). However, the focus is typically on supporting the teen in his or her skill usage and may not be enough involvement for many families. In standard DBT for adults, the identified patient is given skills and is often tasked with using these skills to interrupt family-level patterns.

Models such as Family Connections (Fruzzetti & Hoffman, 2004), Family Skills (Miller & Skerven, 2017) and Staying Connected (Grenyer et al., 2019) are each designed with the understanding that BPD is an interpersonal disorder that has a profound influence on those closest to the affected individual (e.g., extreme expressions of anger, self-injury, impulsive behavior; APA, 2013). Each of these approaches teaches skills to the family members of persons diagnosed with BPD, so as to provide support to families of affected individuals, and to help family members engage effectively with their loved one. However, the diagnosed individual does not attend, and thus reciprocal interpersonal influence may be more challenging to explore in depth. Given our results, families may benefit most from interventions where parent(s) and adolescent(s) are both present and the family is conceptualized as the client, rather than any single person. Integrative borderline adolescent family therapy (Santisteban, Muir, Mena, & Mitrani, 2003) and multifamily DBT (Uliaszek,

Wilson, Mayberry, Cox, & Maslar, 2014) each hold promise for reducing family and individual-level risk, yet are woefully understudied in comparison to standard models. Results suggest that treatment approaches that explicitly involve the whole family hold great promise—particularly during adolescence. Although treating a parent or an adolescent independently is likely to have positive effects on other family members (e.g., Kazdin & Wassell, 2000), deliberately intervening with the family system could meaningfully reshape the home environment such that maladaptive interpersonal patterns are reduced by both parent and adolescent.

Much of the literature examining familial effects focuses on parent-to-youth transmission (e.g., Hallquist et al., 2015; Herr et al., 2008; Lyons-Ruth et al., 2019; Stepp et al., 2014). Many authors have rightly suggested that we treat parents as a means of reducing their offspring's risk (e.g., Kehoe, Havighurst, & Harley, 2014). While this is extremely important, attention should also be paid to the effects of adolescent psychopathology on parent outcomes, as well as to the reciprocal escalation of psychopathology within families.

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