Preschool Age Predictors of Adolescent Borderline Personality Symptoms

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Objective: Developmental models of borderline personality disorder (BPD) have highlighted the interplay of psychological variables (ie, impulsivity and emotional reactivity) with social risk factors, including invalidating parenting and childhood trauma. Prospective longitudinal studies have demonstrated the association of BPD with social, familial, and psychological antecedents. However, to date, few of these studies have studied the interaction of multiple risk domains and their potential manifestations in the preschool period.

Method: Participants were 170 children enrolled in a prospective longitudinal study of early childhood depression. Participants completed a baseline assessment between ages 3 and 6 years. Psychopathology, suicidality, and self-harm were assessed using a semistructured age-appropriate psychiatric interview before age 8 and self-report after age 8. BPD symptoms were assessed between ages 14 and 19 by self-report. Adverse childhood experiences (ACEs) and peer relationships were reported by parents. Maternal support was assessed using an observational measure between ages 3 and 6.

Results: Preschool ACEs accounted for 14.9% of adolescent BPD symptom variance in a regression analysis. Controlling for gender and preschool ACEs, preschool and school-age externalizing symptoms, preschool internalizing symptoms, and low maternal support were significant predictors of BPD symptoms in multivariate analyses. Preschool and school-age suicidality composite scores significantly predicted BPD symptoms.

Conclusion: These findings suggest that preschool factors may be early predictors of BPD symptoms. Findings demonstrate that preschoolers with internalizing and externalizing psychopathology, high ACEs, and early suicidality are at greater risk of developing BPD symptoms. However, further research is needed to guide key factors for targeted early intervention.

Key words: adverse childhood experiences, early childhood, maternal support, preschool onset disorders, suicidality

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Borderline personality disorder (BPD) is a common and highly impairing disorder characterized by instability in affect, impulse control, interpersonal relationships, and self-image. BPD is associated with severe psychosocial impairment and high mortality: up to 10% of patients commit suicide, a rate almost 50 times higher than the general population. The severe impairment BPD causes is reflected in its prevalence in clinical settings. While the community prevalence of BPD is 1%–2%, it is present in up to 20% of people in outpatient psychiatric care and up to 50% of people receiving inpatient psychiatric treatment. The personal and social costs of BPD are heavy with high rates of unemployment and increased disability and health services utilization. Although effective psychotherapy-based treatments for BPD have been developed, their delivery is resource intensive with limited availability. The diagnosis of BPD before adulthood remains controversial owing to the hesitancy of some practitioners to assign a personality disorder diagnosis to patients still in a period of identity formation. However, there is growing evidence of general continuity of BPD symptoms from adolescence to adulthood as well as impairment related to BPD symptoms in adolescence strikingly similar to that experienced by adults.

The morbidity, mortality, and social burden of BPD make prevention, early detection, and intervention urgent public health priorities. However, research on early predictors of BPD has been relatively limited. While there have been some prospective longitudinal studies examining BPD as an outcome, few have looked at preschool risk factors. In a systematic review, Stepp et al. found that, on average, risk factors were assessed at 13 years of age. Thus, there is a need for research on BPD risk factors assessed during early childhood, as treatments focused on parenting and emotion regulation can be highly impactful at this age.

Prominent developmental models of BPD highlight the interaction of psychological variables such as impulsivity,
emotional sensitivity, and overreactivity with social risk factors including invalidating parenting, disorganized attachment, and childhood trauma. These biopsychosocial developmental models have been supported by prospective longitudinal studies demonstrating that BPD is associated with social antecedents such as child abuse and neglect, maternal hostility, early life stress, and peer victimization. Psychological antecedents including attention-deficit/hyperactivity disorder (ADHD) symptoms and infant emotionality and biological antecedents including genetic liability and prenatal exposures have been reported. Family psychiatric history, which includes biological, psychological, and social components, is a strong indicator of risk. However, few studies have accounted for multiple risk domains simultaneously, making it difficult to determine if there is a separable role for each of these risk factors in predicting BPD. Moreover, many of these studies are limited to the adolescent time period, leaving the effects of earlier childhood psychopathology, occurring during a key period of emotional regulatory development, unexplored. Finally, while a number of studies use high-risk community (eg, enriched for poverty) and clinical (eg, enriched for externalizing psychopathology) samples, to our knowledge no study to date has prospectively examined BPD risk factors in a sample selected for internalizing psychopathology ascertained in early childhood, which is also associated with impaired development of emotion regulation.

The goal of the present study was to use longitudinal data from a sample enriched for preschool depression to examine the relationships between family, social, and psychological factors prospectively assessed during early childhood with adolescent BPD symptoms. Following key findings on predictors of BPD, we specifically examined the following potential early developmental antecedents of borderline symptoms: demographic predictors, adverse childhood experiences (ACEs), maternal support, maternal psychopathology, childhood internalizing and externalizing psychopathology, peer problems, and preschool suicidality (which, while unusual, has been observed in this age group). We also attempted to determine the separable effects of ACEs, maternal support, and preschool internalizing and externalizing psychopathology through a multiple regression model.

**METHOD**

**Participants**

Participants were enrolled in the Preschool Depression Study (PDS), a prospective, longitudinal investigation of preschoolers and their families conducted at the Washington University School of Medicine Early Emotional Development Program that has been extensively described elsewhere. Initially, 306 children 3–6 years old and their caregivers were recruited from primary care clinics and day care centers in the St. Louis Metropolitan area oversampling for depression using the Preschool Feelings Checklist, a validated measure assessing depressive symptoms in preschool-age children, enrolling children with symptoms of depression, children with symptoms of disruptive behavior, or children with at least 1 symptom of either type. All children and parents were invited to participate in up to an additional 6 yearly assessments including clinical interviews, observational assessments, and behavioral questionnaires, and a subset of these children and their parents were invited to participate in an additional 3 assessments including similar interviews and 4 neuroimaging scans, in total spanning more than 10 years. The present study included 170 children who completed the Borderline Personality Features Scale for Children (BPFS-C) during assessment 9 (ages 14–19 years, mean [SD] age 16.0 [0.96]). The participants who completed the BPFS-C at assessment 9 were not statistically different in demographics or any of the domains of psychopathology and functioning assessed from participants who did not complete the ninth assessment (Table S1, available online).

**Assessments**

For a composite summary of all assessments and timing of administration, see Table S2, available online.

**Demographic Assessments**

*Income-to-Needs Ratio.* Income-to-needs ratio was assessed at baseline by dividing parent-reported income by the federal poverty guideline for parent-reported number of people living in the home.

**IQ.** IQ was assessed using either the Weschler Abbreviated Scale of Intelligence Second Edition at assessment 5 (ages 8–12) or the Kauffman Brief Intelligence Test Second Edition at assessment 7 (ages 9.17–14.89). Owing to the presumed general stability across development, IQ was considered as a lifetime risk factor.

**Adolescent Psychopathology and Outcome Assessments**

**Borderline Symptoms.** Borderline symptoms in children were assessed using the BPFS-C, a widely used self-report measure of borderline pathology with excellent criterion validity. The borderline symptoms were assessed at assessment 9 (ages 14–19). Our analyses used a continuous Q.
BPFS-C score as well as the standard BPFS-C cutoff score of 65 for presumptive clinical diagnosis.34

Psychopathology. Symptoms of major depressive disorder (MDD), generalized anxiety disorder (GAD), separation anxiety disorder (SAD), social phobia (SOC), posttraumatic stress disorder (PTSD), ADHD, oppositional defiant disorder (ODD), and conduct disorder (CD) were assessed at assessment 9 using the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Present and Lifetime version) (K-SADS-PL),35 a reliable, semistructured clinical interview for parents and children ages 6–18. All diagnoses were based on DSM-5 symptoms endorsed by either the caregiver or the youth.

Impairment. At assessment 9 (ages 14–19), impairment was assessed by the clinician-rated Children’s Global Assessment Scale (CGAS), a widely used, reliable measure of general functioning in children and adolescents.36 The Health and Behavior Questionnaire (HBQ), a reliable and valid parent-report measure of overall impairment, academic functioning, peer relationships, and both overt and relational aggression in children and adolescents,37 was assessed at multiple time points.

Assessments of Early Life Experiences and Risk Factors

Maternal Support. Maternal support was measured at assessment 2 (ages 4.0–7.11 years) through observational coding of the waiting task, a laboratory task designed to produce mild stress for both members of the parent–child dyad. The task requires the child to wait 8 minutes before opening a wrapped gift while the parent completes questionnaires. The parent’s supportive caregiving strategies (providing guidance, verbal assists, warmth and affection, emotion coaching, changing proximity, incorporating the child in their work, and clear directions and explanations) were coded by staff trained to reliability. Support score was calculated as the number of supportive events per minute (unpublished data, January, 2006).

Adverse Childhood Experiences. We created an ACEs variable based on items identified by Felitti et al.,38 summing items assessing parent-reported poverty, parental suicide attempts, substance abuse, and psychopathology and parent- or child-reported traumatic events, then converting to a z score, as detailed elsewhere.39 ACEs variables included a variable comprising experiences only during the preschool period (3–6 years), one comprising experiences only in the school-age period (7–9 years), and one comprising lifetime experiences from all assessments in which these were captured (assessments 1–8). See Table S3, available online, for the rates of ACEs during each developmental period. To ensure the robustness of results, 2 alternative z-transformed ACEs variables were created, one excluding parental psychopathology and one excluding a history of poverty, owing to the inclusion of these factors as independent predictors in our analyses.

Assessments of Maternal Psychopathology. Maternal BPD symptoms were measured using the borderline subscale of the Personality Assessment Inventory (PAI-BOR), a widely used self-report measure of borderline pathology with high criterion validity,40 during assessment 9. General maternal psychopathology was assessed using the Family Interview for Genetic Studies41 and questions about current treatment; mothers comprised 92% of informants. Maternal history of psychopathology (any affective, psychotic, or anxiety disorder or ADHD) and maternal history of depression were calculated as dichotomous variables. They were assessed at assessment 7, when participants were 9.17–14.89 years of age and were assumed to be lifetime risk factors for the child.

Early Life Psychopathology. Symptoms of MDD, GAD, SAD, SOC, ADHD, ODD, and CD were assessed from the first to the eighth assessments using the semistructured parent-report interview Preschool Age Psychiatric Assessment (PAPA)42 from ages 3 to 7 years and the parent- and child-report Child and Adolescent Psychiatric Assessment (CAPA)43 from age 8 and older. MDD, anxiety disorders (GAD/SAD/SOC), ADHD, and ODD/CD symptom scores were sums of the total number of DSM-IV diagnostic symptoms endorsed by either the caregiver or the child during each assessment. These psychopathology variables were collapsed into internalizing (sum of MDD and anxiety disorder (GAD/SAD/SOC) symptoms) and externalizing (sum of ADHD, ODD, and CD symptoms) composites. Preschool composites were averages of all assessments completed between ages 3 and 5.11 years; school-age composites were averages of all assessments completed between ages 6 and 9.11 years.

Assessments of Peer Relationships. Peer relationships were assessed with the parent-reported HBQ,37 collected at each assessment. The global peer relationships scale assessed the participants’ relationships with peers during the preschool (ages 3–5.11 years) and school-age (ages 6–9.11 years) periods. The composite assesses the quality of a child’s friendships, the extent to which the child is liked by peers, and how frequently the child is teased. The HBQ bullied by peers and relational victimization subscales were used in planned post hoc analyses.
Suicidality and Self-Injury Composite Variables. Suicidality composite variables were derived from the PAPA/CAPA during the preschool and school-age periods and from the K-SADS-PL at assessment 9. Each composite summed items measuring recurrent thoughts of death, suicidal ideation, and nonsuicidal self-injury. The preschool/school-age composites from the PAPA/CAPA also included items measuring death and suicide themes in play; the K-SADS-PL composite included items measuring suicidal intention and attempts, similar to Whalen et al. Of the 306 children in the PDS sample, 56 (18%) had suicidality/self-injury during the preschool period.

Plan of Analyses

Regressions. Regression analyses were run to evaluate the association between adolescent BPFS-C score and concurrent functional impairment, with follow-up multiple regression analyses controlling for significant predictors (gender, preschool ACEs, and concurrent MDD) and a separate regression controlling for other psychiatric diagnoses (MDD, anxiety disorders, ADHD, ODD, and CD) (Table 2).

Bivariate multiple regression analyses controlling for gender and preschool ACEs were run to evaluate the association between early life risk factors with adolescent BPFS-C score. To assess if predictors of dimensional borderline symptoms also predicted likely BPD diagnosis (BPFS-C score $>65$), we ran logistic regressions, again controlling for gender and preschool ACEs.

As above, we calculated ACEs variables excluding parental psychopathology and excluding poverty and performed regressions predicting BPD symptoms from these variables. A separate linear regression predicted BPFS-C score excluding the small subset of participants that experienced sexual abuse. We tested whether preschool ACEs differentially predicted the 4 BPFS-C subscales with planned regressions of each subscale. Post hoc analysis of the association between preschool ACEs and depression symptoms from age to 14 to 19 evaluated the specificity of the relationship between ACEs and BPFS-C score. We tested whether there was a significant difference in the variance attributable to ACEs in BPD as opposed to MDD symptoms using the Steiger's $z$-test.

Correction for Multiple Comparisons

All $p$ values were corrected for multiple comparisons, using the Benjamini-Hochberg procedure. All $p$ values reported in Results are the false discovery rate (FDR)–corrected $p$ values and are denoted as FDR-corrected $p$. 
.001), increased relational aggression (β = .22, FDR-corrected p = .048), and increased overt aggression (β = .29, FDR-corrected p = .010). In the multivariate models controlling for gender and comorbid diagnoses, including ever having a diagnosis of MDD, anxiety disorder (GAD, SOC, PTSD), ADHD, ODD, and CD, BPFS-C scores were significantly associated with decreased global functioning as assessed by CGAS parent (β = -.23, FDR-corrected p = .014) and child (β = -.41, FDR-corrected p = .008) interview, poorer academic functioning (β = -.16, FDR-corrected p = .037), increased relational aggression (β = .25, FDR-corrected p = .014), overt aggression (β = .26, FDR-corrected p = .012), and suicidality and self-injury (β = .19, FDR-corrected p = .045).

Preschool and School-Age Adversity, Maternal Psychopathology, and Problematic Peer Relationships Predict BPD Symptoms and Diagnosis

Outside the domain of peer relationships, each childhood variable examined was significantly associated with BPD symptoms (Table 3) (correlations between variables are shown in Table S4, available online). Bivariate analyses demonstrated that BPFS-C scores were associated with female gender (t = 2.44, FDR-corrected p = .024), lower IQ (β = -.15, FDR-corrected p = .027), and lower family income-to-need ratio during preschool (β = -.21, FDR-corrected p = .024). Preschool (β = .38, FDR-corrected p < .001) and school-age (β = -.29, FDR-corrected p < .001) ACEs predicted increased BPFS-C score, as did lower preschool maternal support (β = -.20, FDR-corrected p = .027), maternal borderline features (β = .26, FDR-corrected p = .001), maternal depression history (β = .19, FDR-corrected p = .021), and maternal history of any psychopathology (β = .25, FDR-corrected p = .002). However, preschool and school-age peer relationships were not significantly associated with BPFS-C score when accounting for multiple comparisons. Planned post hoc analyses found that childhood bullying victimization and relational victimization were also not significantly associated with BPFS-C score.

When controlling for gender and preschool ACEs, low preschool maternal support continued to predict increased BPFS-C scores (β = -.20, FDR-corrected p = .024). However, IQ, preschool income-to-needs ratio, school-age ACEs, maternal BPD symptoms, maternal depression history, and maternal psychopathology history no longer significantly predicted BPFS-C scores.

In assessing whether predictors of BPD symptoms also predicted presumptive BPD diagnoses (BPFS-C score >65), we found that female gender, lower IQ, low baseline income-to-needs ratio, and higher preschool and school-age ACEs all were predictive of presumptive BPD. However, in a stepwise multiple logistic regression, preschool ACEs were the only significant predictor of presumptive BPD diagnosis when all were included in the model.

Preschool ACEs Are a Robust Predictor of BPD Symptoms

When calculated without parental psychopathology (β = .31, p < .001) and poverty (β = .37, p < .001) as potential ACEs, preschool ACEs continued to predict BPFS-C scores. When recalculated excluding children who experienced sexual abuse, which was done given prior reports of specific associations between sexual abuse and BPD, preschool ACEs also continued to predict BPFS-C scores (preschool ACEs β = .36, p < .001), though adolescents with a lifetime history of sexual abuse had significantly higher BPD symptoms (mean [SD] 69.43 [11.574]) than adolescents who had not experienced sexual abuse (mean [SD] 55.01 [13.055]; t = 3.987, p < .001).

Post hoc analysis to determine whether preschool ACEs predicted significantly more variance in any specific BPFS-C subscale than others demonstrated that the variance

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Participants with BPD (n = 41)</th>
<th>Participants without BPD (n = 129)</th>
<th>Participants with vs. without BPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDD</td>
<td>39.0 (16)</td>
<td>3.9 (5)</td>
<td>24.61 <strong>&lt; .0001</strong> FDR p &lt; .001 OR 15.87 (5.32, 47.31)</td>
</tr>
<tr>
<td>GAD/SAD/SOC</td>
<td>48.8 (20)</td>
<td>20.9 (27)</td>
<td>11.35 .008 FDR p .0011 OR 3.60 (1.71, 7.58)</td>
</tr>
<tr>
<td>ADHD</td>
<td>19.5 (8)</td>
<td>6.3 (8)</td>
<td>5.78 .0162 FDR p .0162 OR 3.64 (1.27, 10.43)</td>
</tr>
<tr>
<td>ODD/CD</td>
<td>19.5 (8)</td>
<td>1.6 (2)</td>
<td>11.27 .008 FDR p .0011 OR 15.39 (3.12, 75.95)</td>
</tr>
</tbody>
</table>

**Note:** ADHD = attention-deficit/hyperactivity disorder; BPD = borderline personality disorder; CD = conduct disorder; FDR p = false discovery rate-corrected p; GAD = generalized anxiety disorder; MDD = major depressive disorder; ODD = oppositional defiant disorder; OR = odds ratio; SOC = separation anxiety disorder; SOC = social phobia.
### TABLE 2 Borderline Personality Disorder Symptoms Predict Impairment Across a Range of Domains

<table>
<thead>
<tr>
<th>Measure of adolescent functioning</th>
<th>Bivariate model</th>
<th>Model with covariates: gender, preschool ACEs-z, concurrent MDD diagnosis</th>
<th>Model with covariates: gender, diagnosis MDD, anxiety disorder, ADHD, CD, ODD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Stand. Est.</td>
<td>t</td>
</tr>
<tr>
<td>Parent CGAS</td>
<td>169</td>
<td>-0.37</td>
<td>-5.16</td>
</tr>
<tr>
<td>Child CGAS</td>
<td>170</td>
<td>-0.52</td>
<td>-7.85</td>
</tr>
<tr>
<td>HBQ functional impairment</td>
<td>169</td>
<td>0.30</td>
<td>4.12</td>
</tr>
<tr>
<td>HBQ global peer relationships</td>
<td>169</td>
<td>0.36</td>
<td>4.02</td>
</tr>
<tr>
<td>HBQ academic functioning</td>
<td>169</td>
<td>0.27</td>
<td>-3.68</td>
</tr>
<tr>
<td>HBQ relational aggression</td>
<td>168</td>
<td>0.31</td>
<td>4.20</td>
</tr>
<tr>
<td>HBQ overt aggression</td>
<td>168</td>
<td>0.30</td>
<td>4.10</td>
</tr>
<tr>
<td>Child-reported suicidality and self-injury</td>
<td>170</td>
<td>0.34</td>
<td>4.70</td>
</tr>
</tbody>
</table>

Note: When controlling for correlated factors (gender, preschool adverse childhood experiences, and concurrent diagnosis of MDD), borderline personality disorder symptoms continue to predict global functioning and aggression. When controlling for comorbid psychopathology, borderline personality disorder symptoms continue to predict global and academic functioning as well as aggression. Boldface indicates significant results. ACEs-z = z-transformed adverse childhood experiences; ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder; CGAS = Child Global Assessment Scale; FDR = false discovery rate-corrected; HBQ = Health and Behavior Questionnaire; MDD = major depressive disorder; ODD = oppositional defiant disorder; Stand. Est. = standardized estimate.
TABLE 3  Early Life Factors Predict Borderline Personality Disorder Symptoms in Adolescence in Bivariate and Multivariate Regression Models

<table>
<thead>
<tr>
<th>Demographic</th>
<th>Bivariate model</th>
<th>Model with covariates: gender, preschool ACEs-z</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Stand. Est.</td>
</tr>
<tr>
<td>Female gender</td>
<td>170</td>
<td>0.18</td>
</tr>
<tr>
<td>IQ score</td>
<td>163</td>
<td>−0.17</td>
</tr>
<tr>
<td>Baseline income-to-needs ratio</td>
<td>149</td>
<td>−0.21</td>
</tr>
<tr>
<td>Early life experiences</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preschool ACEs-z</td>
<td>137</td>
<td>0.39</td>
</tr>
<tr>
<td>Age 6–8 ACEs-z</td>
<td>141</td>
<td>0.29</td>
</tr>
<tr>
<td>Preschool maternal support</td>
<td>130</td>
<td>−0.19</td>
</tr>
<tr>
<td>Maternal psychopathology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal borderline symptoms</td>
<td>169</td>
<td>0.26</td>
</tr>
<tr>
<td>Maternal depression history</td>
<td>169</td>
<td>0.18</td>
</tr>
<tr>
<td>Maternal psychopathology history</td>
<td>169</td>
<td>0.25</td>
</tr>
<tr>
<td>Early psychopathology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preschool internalizing symptoms</td>
<td>137</td>
<td>0.28</td>
</tr>
<tr>
<td>School-age internalizing symptoms</td>
<td>154</td>
<td>0.17</td>
</tr>
<tr>
<td>Preschool externalizing symptoms</td>
<td>137</td>
<td>0.34</td>
</tr>
<tr>
<td>School-age externalizing symptoms</td>
<td>152</td>
<td>0.29</td>
</tr>
<tr>
<td>Problems with peers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preschool HBQ peer relationships</td>
<td>135</td>
<td>−0.18</td>
</tr>
<tr>
<td>School-age HBQ peer relationships</td>
<td>154</td>
<td>−0.14</td>
</tr>
<tr>
<td>Suicidality</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preschool suicidality and self-injury composite</td>
<td>137</td>
<td>0.25</td>
</tr>
<tr>
<td>School-age suicidality and self-injury composite</td>
<td>154</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Note: Boldface indicates significant results. ACEs-z = z-transformed adverse childhood experiences; FDR p = false discovery rate–corrected p; HBQ = Health and Behavior Questionnaire; Stand. Est. = standardized estimate.

Corrected \( p = .033 \) suicidality/self-injury composites significantly predicted increased BPFS-C score, though neither variable significantly predicted BPFS-C score when controlling for gender and preschool ACEs.

Preschool Risk Factors Explain Variance in Adolescent BPD Symptoms

A multiple regression model including all significant preschool predictors (preschool ACEs, gender, preschool externalizing symptoms, preschool internalizing symptoms, and preschool maternal support) was conducted (Table 4). In this model, preschool ACEs (\( \beta = 0.24, p = 0.011 \)), female gender (\( \beta = 0.019, p = 0.022 \)), and low maternal support (\( \beta = -0.17, p = 0.038 \)) predicted adolescent borderline symptoms. The overall model accounted for more than 20% of the variance in adolescent borderline symptoms (adjusted \( R^2 = 0.216 \)).

DISCUSSION

This study examined relationships between early childhood family, social, and psychological factors and adolescent BPD symptoms using longitudinal data. Interestingly, we found
more adolescents ages 14–19 with presumed BPD than with MDD. As expected, BPD symptoms were associated with increased risk of concurrent psychopathology as well as overall impairment. Bivariate analyses emphasized the role of stressful early life experiences in BPD symptoms, as preschool ACEs strongly predicted borderline symptoms, accounting for 14.9% of variance. Multivariate analyses controlling for gender and preschool ACEs revealed additional roles for maternal support and early psychopathology in development of BPD symptoms as low preschool maternal support, preschool internalizing psychopathology, and preschool and school-age externalizing psychopathology were also robust predictors of BPD symptoms. Analyses showed that preschool and school-age suicidal thoughts and behaviors also predicted adolescent borderline symptoms more than 10 years later. Interestingly, while bivariate analyses replicated past research demonstrating that female gender, low IQ, childhood poverty, and maternal psychopathology predicted increased borderline symptoms, these associations were not significant after controlling for gender and ACEs, underscoring the importance of early childhood trauma in BPD symptoms.

Unexpectedly, the rates of presumed BPD were quite high in this sample of children initially enriched for preschool-onset depression. While children with presumed BPD were more likely to have a co-occurring depression or anxiety diagnosis, less than half of the adolescents with presumed BPD concurrently met criteria for MDD or an anxiety disorder, and thus high levels of BPD symptoms manifested even in the absence of comorbid illness. Moreover, our analyses showed that adolescent borderline symptoms as assessed by the BPFS-C were associated with substantial impairment over and above the contribution of the clinical diagnosis of MDD, anxiety disorder, ADHD, ODD, or CD.

Additionally, multiple regression analyses demonstrated that internalizing and externalizing psychopathology in preschool predicted adolescent borderline symptoms, even after controlling for gender and ACEs. These results are consistent with the biopsychosocial model of BPD and add to the small number of studies specifically using psychopathology variables as borderline predictors. However, it is not clear if the risk of later BPD symptoms is conferred by having a given diagnosis or by dimensional components of these diagnoses, such as affective lability, impulsivity, or difficulty with emotion regulation. Future research should examine whether such dimensional constructs underlie the relationship between early psychopathology and adolescent BPD.
In keeping with the biopsychosocial model of BPD,\textsuperscript{22} bivariate and multiple regression analyses found that preschool ACEs were the strongest predictor of adolescent borderline symptoms in our sample, accounting for almost 15\% of the variance in bivariate analyses. This result is consistent with substantial literature showing that early life stress and adversity, including stressful life events,\textsuperscript{16} lower socioeconomic status,\textsuperscript{27} and family adversity,\textsuperscript{46} prospectively predict borderline symptoms. However, the significant portion of variance in BPD symptoms predicted by preschool ACEs over a decade later suggests that early life stress may be a particularly strong predictor of borderline symptoms. Further, the finding that preschool, but not school-age, ACEs were significant in multiple regression models combining both variables suggests that early childhood may be a particularly sensitive period for the impact of ACEs on the development of borderline personality features. This finding warrants further study, given that there have been no other studies to our knowledge prospectively investigating such early associations, though it provides an important opportunity for preventive interventions.

While bivariate analyses replicated previous results showing that lower IQ,\textsuperscript{30} low childhood socioeconomic status,\textsuperscript{25} and parental psychopathology\textsuperscript{47} are prospectively associated with BPD symptoms, none of these factors remained a significant predictor of BPD symptoms after controlling for gender and preschool ACEs. The lack of association of baseline income-to-needs ratio, maternal BPD symptoms, and psychopathology history with adolescent BPD symptoms after controlling for gender and preschool ACEs might be expected given that our ACEs construct includes items measuring parental psychopathology and concurrent poverty. However, we demonstrate that ACEs continue to predict later BPD symptoms even when ACE scores were calculated excluding either parental psychopathology or poverty.

Our bivariate and multiple regression analyses demonstrated that low preschool maternal support was a robust predictor of adolescent BPD symptoms. While not a novel result, this adds to the few studies\textsuperscript{16,48} that have shown that maladaptive parenting observationally assessed during early childhood is prospectively associated with BPD symptoms. Our parenting data did not allow us to test specific caregiving behaviors, such as invalidation,\textsuperscript{23} that contribute to BPD risk in developmental models, though this is an important future direction given the potential role for parenting-based interventions in early BPD treatment and prevention.

Preschool and school-age peer relationships were not significant predictors of BPD symptoms. In particular, we failed to replicate the finding of Wolke et al.\textsuperscript{18} that school-age bullying victimization and relationship victimization prospectively predict borderline symptoms. This discrepancy may be due to school-age bullying being measured less proximally to BPD symptoms in this study than in Wolke et al.\textsuperscript{18} (at least 5 years versus 2–4 years).

In a novel analysis, we found that preschool and school-age suicidality/self-harm were significant predictors of BPD symptoms. This result builds on the earlier research of Whalen et al.\textsuperscript{31} showing that early childhood suicidal cognitions and behaviors were a robust predictor of school-age suicidal cognitions and behaviors. The current results suggest that for some patients with BPD, self-harm and suicidal thoughts and behaviors may precede or be some of the earliest indicators of their BPD symptoms and may begin as early as the preschool period. Further research should investigate whether early suicidality/self-harm specifically predicts BPD symptoms in the domain of self-harm or also predicts other BPD symptom domains (eg, identity problems). Further research should also assess the specificity of early suicidality as a predictor of BPD versus other forms of psychopathology.

This study was limited by its sample size, which did not provide sufficient power to investigate the interaction of risk factors in mediating and moderating analyses. Additionally, the enrichment for preschool depression, while enhancing the clinical relevance of this study, may make the results less applicable to the general population. BPD symptoms were measured in adolescence, possibly limiting inferences to adult BPD. However, there is a growing consensus\textsuperscript{8,12} that a diagnosis of BPD is as reliable in adolescence as in adulthood. Nonetheless, future research should assess the effect of these early childhood predictors on adult BPD as well. Owing to low base rates of trauma outside of poverty and maternal psychopathology, we were unable to isolate specific
components of the ACEs variable to predict BPD. Mothers were used as primary informants during the preschool years, and therefore some of the information collected may be prone to social desirability and related biases. Future work should capitalize on the strengths of a multi-informant approach.

Overall, the findings presented here have considerable implications for BPD prevention and early intervention. The overall prevalence of adolescent BPD (24%) in this preschool depression enriched cohort suggests that children with early affective psychopathology may be at high risk of developing BPD symptoms. Preschool variables explained a large amount of the variance in adolescent borderline symptoms: the model including gender, preschool ACEs, internalizing psychopathology, and externalizing psychopathology accounted for more than 20% of variance. These results suggest that preschoolers with psychopathology, ACEs, and suicidal thoughts/behavior are at high risk of borderline symptoms and should be targeted for further evaluation and intervention. Children with early psychopathology and stressful experiences, particularly children with early suicidality and low maternal support could receive targeted interventions aimed at developing emotion regulation skills, such as dialectical behavioral therapy for children or Parent Child Interaction Therapy Emotional Development, hopefully limiting the extremity morbidity and mortality associated with BPD. Further research investigating the role of various aspects of emotional competence in the pathways from early BPD risk factors to BPD development could result in the discovery of more specific clinical markers and new targets for early intervention.

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