Developmental Predictors of Adult Borderline Personality Disorder: A Prospective, Longitudinal Study of Females with and without Childhood ADHD

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Abstract

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Research on the precursors of borderline personality disorder (BPD) suggests numerous child and adolescent risk factors, with impulsivity and trauma among the most salient. Yet few prospective longitudinal studies have examined pathways to BPD; even fewer examine multiple risk domains or childhood risk factors in prospective designs. We prospectively examined theory-informed predictors of young-adult BPD in both childhood and late adolescence via a diverse (47% non-white) sample of females with (n = 140) and without (n = 88) carefully diagnosed childhood attention-deficit hyperactivity disorder (ADHD). After stringent adjustment for key covariates, we found that childhood hyperactivity/impulsivity did not predict later BPD; instead, low levels of objectively measured executive functioning in childhood served as a predictor. Consistent with previous research and the Biosocial Theory of BPD, a history of childhood trauma was also a salient predictor. In terms of late-adolescent predictor variables, after stringently adjusting for all predictors and relevant covariates, only inattentive symptoms maintained marginal significance. Our findings underscore the need for preventive interventions before adolescence in populations with enhanced risk, particularly interventions focused on improving executive functioning skills and reducing risk for trauma as well as trauma related symptomology. Understanding both inattention and hyperactivity/impulsivity in youth with ADHD is also indicated. Further research is needed to identify salient pathways to BPD in prospective, longitudinal studies, with extensions to male samples essential.

Borderline personality disorder (BPD) is a persistent and highly impairing disorder characterized by intense and pervasive dysregulation of emotion, behavior, cognition, and relationships (Crowell et al., 2009). Individuals with BPD are at extremely high risk for suicide; up to 10% of individuals with BPD die by suicide each year, 50 times higher than that of the general population (Olfson et al., 2016; Pompili et al., 2005). In addition, BPD is associated with significant personal (i.e., severe psychosocial impairment) and economic/public health (i.e., high rates of underemployment and increased disability) consequences. In fact, although individuals with BPD comprise 1-2% of the general population (Torgersen et al., 2001), they have extremely high rates of health service use, representing up to 50% of individuals receiving inpatient psychiatric treatment and 20% receiving outpatient psychiatric care (Grilo et al., 1998; Korzekwa et al., 2008). Evidence-based psychological treatments (e.g., Dialectical Behavior Therapy [DBT]) exist for BPD, with strong evidence for efficacy (Linehan et al., 2015), but are resource intensive, with limited availability of expert providers.

Given the high morbidity, mortality, and public health consequences of BPD, there is an urgent need to identify individuals at risk for its development. Relevant research is accumulating. A leading theory is Linehan's Biosocial Theory, which proposes that BPD emerges from transactions between biological vulnerabilities to emotional sensitivity, as well as impulsivity, plus specific environmental influences such as social invalidation, adversity, or trauma (Crowell et al., 2009; Linehan, 1993). Across development, such variables appear to transact and give rise to increasingly more extreme emotional, behavioral, cognitive, and interpersonal dysregulation for the most vulnerable individuals. Although empirical research has generated numerous risk factors for BPD (Beeney et al., 2021; Stepp, Lazarus, et al., 2016), relatively few studies have examined longitudinal pathways to BPD. In particular, little research has examined childhood risk factors for BPD in prospective designs (Geselowitz et al., 2021). Indeed, a 2016 systematic review of risk factors for BPD revealed that most risk factors were assessed during early adolescence (M_{age} = 13 years), highlighting the need for further investigation of childhood variables and processes (Stepp, Lazarus, et al., 2016). In the present investigation, we examine both child and late-adolescent risk factors.

Developmental Risk Factors for BPD

ADHD symptoms. Impulsivity is a key clinical feature in both the development and presentation of BPD (Crowell et al., 2009). It is also a core feature of attention-deficit hyperactivity disorder (ADHD). In fact, several studies have reported high comorbidity between BPD and ADHD (Black et al., 2007; Coolidge et al., 2000; Jacob et al., 2007; S. D. Matthies & Philipsen, 2014; T. W. Miller et al., 2007; Rösler et al., 2004, 2009; Storebø & Simonsen, 2016). In a large national study of 34,000 adults in the United States, among adults with ADHD the lifetime comorbidity with BPD was 33.7%, compared with 5.2% in the general population (OR: 2.84) (Bernardi et al., 2012). Comorbid ADHD and BPD is a particularly impairing combination (Ferrer et al., 2010; Speranza et al., 2011). As for childhood ADHD, most prior research has involved retrospective reports of symptoms. Key studies note that 50-60% of adults with BPD endorsed high levels of ADHD symptoms in childhood (Andrulonis, 1991; Fossati et al., 2002). Philipsen et al. (2008) reported estimates of ADHD among adult women with BPD to be especially high in childhood (41.5%) compared to adulthood (16.1%). Severity of childhood ADHD symptoms has also been associated with higher frequency of personality disorder diagnoses, including BPD, by adulthood (Matthies et al., 2010).

Hypothesized mechanisms linking ADHD and BPD focus on the interaction across development between early vulnerability to impulsivity (a highly heritable trait) and emotion

dysregulation, which are in turn shaped over time by adverse socialization processes (e.g., maltreatment, family reinforcement of emotion lability; Beauchaine et al., 2019). Proposed neuropsychological mechanisms in this link focus on dysfunction in the prefrontal cortex, which is also implicated in emotion regulatory capacities (Stepp et al., 2012; Beauchaine et al., 2019). Essentially, early ADHD is hypothesized to confer risk for later BPD due to its associated behavioral dysregulation which may lead to environmental reinforcement of maladaptive behaviors, leading to a pervasive and difficult to treat cycle of dysregulation. Yet few prospective studies have examined the link between childhood ADHD and later BPD. Notable exceptions (see Miller et al., 2008; Rasmussen & Gillberg, 2000; Rey et al., 1995) reveal that childhood ADHD predicts personality disorders, including BPD, later in life. In a follow-up investigation of "hyperactive" children in young adulthood, Fischer et al. (2002) found that 14% of hyperactive participants met criteria for BPD compared to 3% of their comparison group. Additionally, Miller et al. (2008) found that among a group with childhood ADHD, 13.5% were diagnosed with BPD in adolescence compared to 1.2% in their comparison group. Furthermore, in the Pittsburgh Girls Study, Stepp et al. (2012) found that higher levels of ADHD symptoms during childhood predicted BPD in adolescence (see also Burke & Stepp, 2012, for parallel findings in males). As well, using latent-class analysis, Thatcher et al. (2005) found that the presence of ADHD symptoms in adolescents along with substance use disorders predicted more severe BPD symptoms at young adult follow-up.

The majority of relevant research has focused on the categorical diagnosis of ADHD rather than the core ADHD dimensions of (a) hyperactivity/impulsivity and (b) inattention. Exceptions include Carlson et al. (2009) who reported data from a prospective, longitudinal study that teacher-rated severity of both attentional disturbance and behavioral instability (including impulsivity) at age 12 was predictive of adult BPD. This finding was recently replicated by Beeney et al. (2021) in a prospective study of females. In this study, parent- and child-reported severity of hyperactivity, impulsivity, and inattention at ages 14-15 predicted BPD at ages 16-18. In a prospective study of twins, maternal- and teacher-rated symptoms of impulsivity at age 5 were related to borderline symptoms at age 12 (Belsky et al., 2012).

Some adult research has examined presentations of ADHD as related to BPD. Among adults with ADHD, one study reported a higher prevalence rate of comorbid BPD and the Combined presentation of ADHD (ADHD-C; 24%), of which hyperactivity/impulsivity and inattention are key components, compared to comorbid BPD and the Inattentive presentation (ADHD-I; 10%) (Cumyn et al., 2009). Using latent class analysis with adult females, van Dijk et al. (2011) found one pathway to adult BPD emanated from a childhood profile with at least low levels of hyperactive/impulsive symptoms but no inattentive symptoms.

Finally, considered either categorically or dimensionally, ADHD is clearly linked with increased risk for self-harm (including suicidal behavior and nonsuicidal self-injury (NSSI); see Hinshaw et al., 2022 for a recent review). Population studies have demonstrated increased risk for self-harm among individuals with ADHD (Chen et al., 2014; Hurtig et al., 2012; see review in Garas & Balazs, 2020), replicating findings from longitudinal studies (Chronis-Tuscano et al., 2010; Hinshaw et al., 2012). Notably, females with ADHD, especially those who demonstrate high levels of impulsivity as characterized by the ADHD-C presentation, are markedly at risk for attempted suicide and moderate-to-severe NSSI (Hinshaw et al., 2012). Mechanisms linking ADHD with later self-harm include internalizing and externalizing symptoms, as well as peer victimization and preference (Meza et al., 2016; Swanson et al., 2014). Risk for suicidality is

greatly increased when females with ADHD have histories of childhood maltreatment (Guendelman et al., 2016).

Executive functioning (EF). EF includes goal-oriented cognitive processes such as planning, inhibition, organization, set shifting, working memory, and problem solving. EF deficits have frequently been linked to both ADHD and BPD (Barkley, 1997; Pennington & Ozonoff, 1996). For a review of ADHD and EF see Brown (2013) and for a review of ADHD and BPD, see Garcia-Villamisar et al. (2017). In general, individuals with BPD show greater EF deficits compared to typically developing controls (Haaland et al., 2009). However, there is a lack of consensus regarding which specific domains of EF are salient in predicting BPD. Several studies indicate that deficits in planning are characteristic of people with BPD (Gvirts et al., 2012; Ruocco, 2005, but see Bustamante et al., 2009 for conflicting results). Deficits in working memory have also been demonstrated (Hagenhoff et al., 2013). Additionally, among individuals with BPD who engaged in self-injury, severity of self-injury was related to increased deficits in the domains of response inhibition and problem solving (Williams et al., 2015). In a sample of individuals with ADHD, BPD, and the combination, individuals with ADHD alone performed worse on tasks measuring motor and cognitive inhibition than did those with BPD alone; individuals with comorbid ADHD/BPD showed impairments only in motor inhibition compared to controls (Lampe et al., 2007). Additionally, a meta-analysis revealed that BPD samples with higher rates of comorbid psychopathology performed worse on EF tasks compared to samples with lower rates of comorbidity (Unoka & Richman, 2016). Few prospective longitudinal studies examined childhood EF as predictive of later BPD, with the notable exception of Belsky et al. (2012), who found that a composite measure of EF at age 5 was negatively related to BPD symptoms at age 12.

Early internalizing & externalizing symptoms. BPD is commonly comorbid with a variety of other psychological disorders, both internalizing and externalizing disorders (Eaton et al., 2011). In a systematic review of risk factors for BPD from longitudinal research, 16 of 19 studies examining internalizing and externalizing psychopathology found predictions to later BPD (Stepp, Lazarus, et al., 2016). For example, Stepp et al. (2013) found that dimensions of internalizing (depression) and externalizing (substance use disorder) behaviors in adolescence were associated with subsequent adult BPD symptoms. Parent and teacher ratings of internalizing and externalizing symptoms at age 5 were related to BPD symptoms at age 12 (see, again, Belsky et al., 2012). Geselowitz et al. (2021) provided parallel findings (but see Burke & Stepp, 2012, for negative results). Hypothesized mechanisms in this link include emotion dysregulation which characterizes both internalizing and externalizing psychopathology (Beauchaine et al., 2019). In short, understanding the contribution and developmental timing of internalizing and externalizing symptoms has the potential to inform early interventions to prevent the development of clinically significant BPD symptomology.

Adverse childhood experiences/trauma. Consistent with Linehan's Biosocial Model, a large body of research has linked a history of environmental invalidation and adversity—and at its extreme, trauma—to the development of BPD (Ball & Links, 2009; Crowell et al., 2009; Herman et al., 1989; Zanarini & Frankenburg, 1997). A history of physical abuse, sexual abuse, and neglect in childhood has long been linked with BPD (Bornovalova et al., 2013; Ogata et al., 1990; Stepp, Scott, et al., 2016; Westen et al., 1990; Zanarini, 2000). For key prospective longitudinal investigations, see Johnson et al. (1999) and Widom et al. (2009). Additional family-related risk factors for BPD include (a) having a parent with psychopathology (including depression and substance use problems) and (b) lower parental educational attainment and lack

of full-time employment (see Barnow et al., 2013; Stepp et al., 2013). Similarly, high parenting stress has been linked to BPD, although results have been mixed (Infurna et al., 2016; Schuppert et al., 2015). Children who were physically maltreated or exposed to high maternal negative expressed emotion developed high levels of BPD characteristics in a prospective, longitudinal study of twins followed from age 5 to 12 (Belsky et al., 2012), replicating other prospective studies (Carlson et al., 2009; Crawford et al., 2009; Johnson et al., 2006).

Present Study and Hypotheses

In sum, numerous risk factors for BPD have been posited, including ADHD symptoms, low executive functioning, early internalizing and externalizing psychopathology, and childhood adversity and trauma. Yet with the notable exception of Beeney et al. (2021), few studies have examined these risk factors simultaneously, limiting our current understanding of the independent or combined contributions of each. In addition, many studies examine limited developmental periods (e.g., childhood to adolescence, adolescence to young adulthood). Finally, there is a dearth of research examining the core ADHD dimensions of hyperactivity/impulsivity and inattention as related to risk for later BPD (for a review of developmental issues, see Beauchaine et al. (2019).

We therefore leverage a sample of females with childhood-diagnosed ADHD and a matched comparison sample followed prospectively from childhood through adulthood. Consistent with Linehan's Biosocial Model and much extant literature, we hypothesize that both childhood and late adolescent (a) impulsivity and (b) adversity/trauma will emerge as significant risk factors for BPD after adjusting for covariates as well as additional evidence-based risk factors. We also predict that, by late adolescence, internalizing and externalizing symptoms will be significant predictors of adult BPD. Our aim is to add to the literature on developmental risk factors for BPD to inform existing models of BPD development and preventive approaches.

Method

Procedure & Participants

The current data were drawn from the Berkeley Girls with ADHD Longitudinal Study (BGALS), an ongoing prospective, longitudinal study of females with and without carefully diagnosed childhood ADHD (see Hinshaw et al., 2002 for more complete details). Participants were initially recruited across the San Francisco Bay Area from schools, mental health centers, pediatric practices, and through advertisements to participate in research-based, 5-week summer day camps between 1997-1999. These programs were designed to be enrichment programs featuring classroom and outdoor environments for ecologically valid assessment, rather than intensive therapeutic interventions. All participants and their families underwent a rigorous, multi-step psychodiagnostic assessment process (see below) after which 140 girls with ADHD and 88 age- and ethnicity-matched comparison girls were selected to participate in the first program (Wave 1; M_{age} = 9.6 years, range = 6-12 years).

To establish a baseline diagnosis of ADHD, we used the parent-administered Diagnostic Interview Schedule for Children, 4th ed. (DISC-IV) (Shaffer et al., 2000) and SNAP rating scale (Swanson, 1992; see Hinshaw, 2002, for the diagnositc algorithm). Comparison girls could not meet diagnostic criteria for ADHD on either measure. Some comparison girls met criteria for internalizing disorders (3.4%) or disruptive behavior disorders (6.8%) at baseline, yet our goal was not to match ADHD participants on comorbid conditions, rather our aim was to obtain a representative comparison group. Study exclusion criteria included intellectual disability, pervasive developmental disorders, psychosis, overt neurological disorder, lack of English spoken at home, and medical problems preventing summer camp participation. A sample of 228

girls with ADHD-Combined presentation (n = 93) and ADHD-Inattentive presentation (n = 47), plus an age- and ethnicity-matched comparison sample (n = 88) was selected. Participants were ethnically diverse (53% White, 27% African-American, 11% Latina, 9% Asian American), reflecting the composition of the San Francisco Bay Area in the 1990's. Family income was slightly higher than the median California household income in the mid-1990s, yet income and educational attainment of families was highly variable. On average, parents reported being married and living together (65.8%) at the baseline assessment.

Participants were then assessed 5 (Wave 2; M_{age} = 14.2 years, range = 11-18; 92% retention [data not included from this wave in the present study]), 10 (Wave 3; M_{age} = 19.6 years, range = 17-24 years; 95% retention), and 16 (Wave 4; M_{age} = 25.6 years, range = 21-29 years; 93% retention) years later. Data collection included multi-domain, multi-informant assessments, performed in our clinic for most individuals but when necessary, we performed telephone interviews or home visits (for additional information on the follow-up assessments, see Hinshaw et al., 2006, 2012; Owens et al., 2017).

Measures

Predictor variables. Predictor variables were measured during the baseline assessment at Wave 1 (childhood), with repeated assessment of several key measures at Wave 3 (late adolescence).

ADHD Symptom Severity: Swanson, Nolan, and Pelham rating scale, 4th Ed. (SNAP-IV; Swanson, 1992*).* We measured severity of both hyperactivity/impulsivity and inattentive symptoms using an average of parent- and teacher-report (childhood) or parent- and self-report (late adolescence) on a dimensionalized checklist of these two respective symptom domains (9 items for each) to obtain multi-informant composite scores (Inattentive Symptoms; α = 0.968; Hyperactive/Impulsive Symptoms; α = 0.950). The severity of each symptom was scored 0 (not at all) to 3 (very much). Thus, scores of both hyperactivity/impulsivity and inattention symptoms ranged from 0-27, with higher scores indicating more severe symptomology. The SNAP-IV is a widely used scale of ADHD symptom severity in both research and clinical settings (e.g., MTA Cooperative Group, 1999). It has good internal consistency and test-retest reliability (Bussing et al., 2008).

Internalizing and Externalizing Symptoms: Child Behavior Checklist, Adult Self Report, and Adult Behavior Checklist (CBCL; ASR; ABCL; Achenbach, 1991; Achenbach & Rescorla, 2003). In childhood, we measured severity of internalizing (α = 0.892) and externalizing (α = 0.925) symptoms via parent-report on the Internalizing and Externalizing scales of the CBCL. In late adolescence, we averaged participant self-report on the Adult Self-Report (ASR) and parent-report on the Adult Behavior Checklist (ABCL) to obtain a multi-informant composite score of these domains. The ASR and ABCL constitute parallel versions of the CBCL for older individuals. We used T-scores (M = 50, SD = 10) to obtain dimension symptom measures, with scores above 60 indicating elevated/at-risk and scores above 70 indicating clinically significant symptomology. The CBCL, ASR, and ABCL have good-excellent validity, test-retest reliability, and internal consistency (Achenbach & Rescorla, 2003; Nakamura et al., 2009).

Executive Functioning: Rey Osterrieth Complex Figure (ROCF; Osterrieth, 1944). We measured executive functioning using the ROCF, a laboratory-based cognitive task that requires an individual to copy and later recall a complex image composed of 64 segments. The ROCF measures multiple domains of executive functioning such as planning, inhibitory control, attention to detail, working memory, and organization. It is often considered a "global" measure of executive functioning. We analyzed the Copy condition of this task, during which participants

are timed as they view the stimulus figure and draw the figure on a piece of paper. We used the Error Proportion Score (EPS; the ratio of number of errors divided by the total number of segments drawn), a well-validated method of scoring the ROCF, indexing efficiency (Sami et al., 2003). In previous research with this sample, only the Copy condition (versus Delayed Recall condition) differentiated girls with ADHD from our comparison sample at baseline. The ROCF EPS showed the largest effect size (d = 0.90) out of all other EF measures in our battery (Hinshaw et al., 2002; Sami et al., 2003). As well, childhood EPS predicts later academic and occupational functioning compared to other EF measures (Miller et al., 2012).

Adverse childhood experiences/trauma.

Physical abuse, sexual abuse, and neglect. We ascertained experiences of physical abuse, sexual abuse, and neglect via thorough, multi-step chart review between Waves 1-3, as described by Guendelman et al. (2016). In brief, blinded coders rated participants' charts, which included multi-informant measures and reports (i.e., project-derived Background Information Questionnaire and Family Information Profile; Report from Summer Program, Child Protective Services reports, and medical reports) for documented experiences of abuse or neglect. Only substantiated (versus suspected) incidents (e.g., confirmation of abuse/neglect from reports from CPS, school districts, or treating clinicians) were included (Briscoe-Smith & Hinshaw, 2006). History of physical abuse was coded if there was "intentional use of a physical force against a child that results in, or has the potential to result in, physical injury" (Leeb et al., 2008) and the perpetrator was an adult over the age of 18, caregiver, or romantic partner (e.g., a participant's father hit the participant). Sexual abuse was coded if the participant had any history of a completed or attempted sexual act or sexual contact by a caregiver, peer, stranger, or acquaintance (e.g., a participant was raped by a stranger). Neglect was coded if there was clear failure by a caregiver to meet the participant's basic physical, medical, and/or educational needs (e.g., parents failing to provide the participant with adequate nutrition). Inter-rater reliability was good-excellent (kappa = 0.78, range = 0.64-0.89; (Bakeman & Gottman, 1997; Fleiss, 1981).

Parent psychopathology: Beck Depression Inventory (BDI-I; BDI-II; Beck et al., 1961, 1996). We measured depressive symptoms of the primary caregiver (typically the mother) using self-report on the BDI-I at Wave 1 and the BDI-II at Wave 3. Mothers rated each of the 21 items on a 4-point severity scale. Total possible scores could range from 0-63, with higher scores indicating greater severity of depression. The BDI is a widely used and extensively validated self-report measure of depression in adults (Bumberry et al., 1978; Sprinkle et al., 2002).

Parenting stress: Parenting Stress Index-Short Form (PSI-SF; Abidin, 1995). We measured maternal parenting stress using maternal self-report on the PSI-SF, a widely used 36-item self-report measure assessing stress experienced by parents related to their role as a parent. Participants' mothers rated each item on a scale from 1 (strongly agree) to 5 (strongly disagree). We used the Parenting Distress subscale, measuring distress a parent feels in their role as a parent. Higher scores indicated higher levels of maternal parenting stress. The PSI-SF has demonstrated good test-test reliability and internal consistency (Abidin & Brunner, 1995), as well as validity (Haskett et al., 2006).

Cumulative childhood adversity: Adverse Childhood Experiences questionnaire (ACE; Felitti et al., 1998). We measured cumulative experiences of childhood adversity via retrospective report by each participant on the ACE questionnaire at Wave 4, which assesses experiences of childhood abuse, neglect, and household dysfunction during the first 18 years of life. The total possible ACE score ranged from 0-10, with higher scores indicating experiences of multiple types of childhood adversity. The ACE questionnaire is a commonly used measure to

assess for the cumulative effect of multiple forms of childhood adversity (Petruccelli et al., 2019), and has good reliability and validity (Hardt & Rutter, 2004)—including at least moderate test-retest reliability of retrospective reports (Dube et al., 2004).

Criterion variable. Our criterion variable was measured at Wave 4 (Adulthood).

Borderline Personality Disorder. A licensed clinical psychologist or a graduate student in clinical psychology conducted a clinical interview with participants using the Structured Clinical Interview for DSM-IV-TR (SCID; First et al., 2002) and the Borderline Personality Disorder (BPD) module of the SCID-II (SCID-II; First et al., 1997). The SCID-II is a semi-structured interview widely used in both research and clinical practice, with research indicating good to excellent inter-rater reliability (Huprich et al., 2015). A participant met criteria for a diagnosis of BPD if the clinician rated the participant at or above threshold on five of the nine symptom traits. A single dichotomous variable (0 or 1) reflected a BPD diagnosis.

Covariates. To ascertain whether domains of impairment were related specifically to BPD status, we added stringent covariates empirically associated with BPD and associated predictors: (1) socioeconomic status (SES)—parent report of family income and maternal education in childhood; (2) parent report of child's race/ethnicity in childhood; and (3) participant age in young adulthood.

Data Analytic Plan

Statistical analyses were performed with RStudio, version 1.2.1335. First, we computed zero-order correlations for all study variables, before conducting a series of independent-samples *t*-tests to assess relations between variables of interest and an adult diagnosis of BPD (vs. non-diagnosed). We calculated effect sizes of Cohen's *d* for continuous variables, and odds ratios (OR) for non-continuous variables. Second, to examine predictors of adult BPD, we conducted a series of binary logistic regressions to test whether each theory-informed predictor independently predicted a diagnosis of BPD by young adulthood. Given the many potential predictors, we deployed the stringent criterion of including predictors only they displayed on an effect size of at least 0.5 in differentiating adults with vs. without BPD.

Third, using binary logistic regressions, we tested whether the relevant predictor variable predicted BPD when adjusting for sociodemographic covariates (parent report of family income, maternal education at baseline, participant race/ethnicity, and participant age). Finally, we added all predictor variables that maintained significance in these analyses into two separate models according to developmental period (Model 1 = childhood predictors, Model 2 = late-adolescent predictors). We used Firth's penalized likelihood method to address our relatively small sample size and to minimize bias introduced by several independent variables (Firth, 1993).

Results

Intercorrelations and descriptive analyses

Initial analyses revealed that a total of 19 women met criteria for a diagnosis of BPD in young adulthood. Of these, 14 (74%) had received a childhood diagnosis of ADHD ($\chi^2(3, N = 199) = 1.1$, $p = 0.3$, *OR*: 1.31, CI: 0.79, 2.17), with the majority having received a childhood diagnosis of ADHD-C (58%).

Next, Table 1 presents intercorrelations among key variables. Wave 1 SNAP-HI severity ($r = .17$, $p < .05$), Wave 1 RCFT ($r = .22$, $p < .01$), ACE ($r = .32$, $p < .01$), Wave 3 SNAP-HI severity ($r = .40$, $p < .01$), Wave 3 SNAP-IA severity ($r = .38$, $p < .01$), Wave 3 Maternal BDI-II ($r = .16$, $p < .05$), Wave 3 ASR/ABCL Internalizing ($r = .34$, $p < .01$), and Wave 3 ASR/ABCL Externalizing ($r = .39$, $p < .01$) were all significantly correlated with Wave 4 BPD, with small to medium effect sizes (for Pearson's *r*, 0.1 = small, 0.3 = medium, and 0.5 = large).

Predictors: Binary logistic regressions and Cohen's *d*

As noted above, for predictor analyses we used binary logistic regressions with Firth's penalized likelihood method to assess independent predictors of the dichotomous outcome of meeting (vs. not meeting) criteria for BPD at Wave 4. We tested whether each predictor of interest was significantly associated with BPD alone, followed by inclusion of (a) covariates and (b) other predictor variables according to developmental period (see Table 2).

In childhood, HI severity ($p = .022$; $d = 0.58$) and low EF ($p < .01$; $d = 0.76$) each predicted BPD status in adulthood. Yet only low EF maintained significance after adjusting for covariates ($p < .05$). Regarding adversity/trauma during in childhood, only the ACE score had an effect size over 0.5. A cumulative history of childhood adversity, via ACE, significantly predicted young adult BPD ($p < .001$; $d = 1.14$), after adjusting for covariates ($p < .01$).

In late adolescence, symptoms of hyperactivity/impulsivity ($p < .001$; $d = 1.45$) and inattention ($p < .001$; $d = 1.36$) each predicted young adult BPD, after adjusting for covariates. Maternal depression in late adolescence initially predicted young adult BPD ($p < .05$; $d = 0.54$) but not after adjusting for covariates ($p = 0.093$). Late-adolescent internalizing ($p < .001$; $d = 1.23$) and externalizing ($p < .001$; $d = 1.41$) symptoms each predicted young adult BPD, even with inclusion of covariates. No other variables emerged as statistically significant.

Finally, we entered all predictors that maintained significance after inclusion of covariates into two different models, divided by developmental period. First, in childhood, both low EF ($p = 0.012$) and the ACE score maintained significance ($p = 0.003$). Second, in the late-adolescent predictor model, only inattentive symptoms maintained marginal significance ($p = 0.059$).

Discussion

Leveraging a well-characterized longitudinal sample of females with and without carefully diagnosed childhood ADHD, we examined theory-informed predictors of adult BPD from both childhood and late adolescence. Using binary logistic regressions with correction for small sample size, we found that, contrary to our predictions, childhood hyperactivity/impulsivity did not predict BPD after adjusting for covariates. Yet consistent with the hypotheses, a cumulative history of childhood adversity, as measured by the ACE score, predicted BPD. Additionally, low EF in childhood was also a predictor of later BPD, even adjusting for ACE and covariates. Regarding late-adolescent predictors, hyperactivity/impulsivity, inattention, internalizing, and externalizing symptoms each independently predicted adult BPD after adjusting for covariates, but maternal depression did not. In stringent analyses accounting for all independently significant predictors in late adolescence, only symptoms of inattention were independently (albeit marginally), related to adult BPD.

Overall, the child and adolescent predictors of later BPD are largely consistent with those from previous investigations (Beeney et al., 2021; Belsky et al., 2012; Carlson et al., 2009; Geselowitz et al., 2021; Stepp et al., 2012, 2013)—and emanate from a carefully controlled prospective investigation. Regarding ADHD symptoms, almost 75% of women who met criteria for BPD in adulthood had diagnoses of childhood ADHD, most often characterized by high levels of impulsivity (ADHD-C). This finding is consistent with both cross-sectional and longitudinal research, as well as theoretical models of the developmental course of individuals with high levels of early impulsivity, related to BPD as an end-point (Beauchaine et al., 2019).

For ADHD dimensions, our findings add to the limited number of studies examining hyperactive, impulsive, and inattentive symptoms and their severity across development,

especially beginning in childhood (Beeney et al., 2021; Belsky et al., 2012; Carlson et al., 2009). That hyperactivity/impulsivity in childhood and adolescence did not significantly predict later BPD, when adjusting for covariates and other predictors, was unexpected. Yet the symptom dimensions of (a) hyperactivity/impulsivity and (b) inattention, as well as (a) hyperactivity/impulsivity and (b) executive functioning are substantially intercorrelated, and our sample size is small. To our knowledge, only one study has found prospective associations between childhood impulsivity (assessed at age 5) and later BPD (assessed at age 12; see Belsky et al., 2012). Two studies have found prospective links between both adolescent hyperactivity/impulsivity and inattention and later BPD (Beeney et al., 2021; Carlson et al., 2009). The (marginal) finding that adolescent inattention plays a predictive role replicates both Carlson et al. (2009) and a recent machine learning study revealing that among 128 variables related to risk for BPD, inattention in adolescence emerged as an important predictor (Beeney et al., 2021). Our prior research indicates that severity of inattention, as well as hyperactivity/impulsivity, is a potent predictor of young-adult self-harm (Meza et al., 2020), a common symptom of BPD.

The finding linking low EF in childhood to adult BPD is also consistent with previous (yet limited) research. In the only known prospective longitudinal study to date examining childhood EF as predictive of later BPD, a composite measure of EF at age 5 predicted BPD symptoms at age 12 (Belsky et al., 2012). Additional research is needed to probe the interconnections of impulsivity and EF in relation to pathways to BPD.

Regarding internalizing and externalizing symptoms, we found that high levels of such symptoms in adolescence were related to later BPD, yet after the adjustment for other important predictors this effect was no longer significant (for additional research, see Belsky et al., 2012; and Geselowitz et al., 2021). Our findings replicate findings from Stepp et al. (2013), who reported that adolescent internalizing and externalizing symptoms predicted adult BPD. Still, adolescence appears to be a particularly sensitive period during which vulnerability for the development of severe and pervasive dysregulation across the lifespan may be realized (Sharp et al., 2018).

Consistent with a large body of research linking a history of childhood adversity/trauma with later BPD (Ball & Links, 2009; Bornovalova et al., 2013; Herman et al., 1989; Stepp, Scott, et al., 2016; Widom et al., 2009; Zanarini & Frankenburg, 1997), we found that a cumulative history of childhood adversity, as measured by the ACE scale, was an important predictor of adult BPD. This finding supports theories that transactions between dispositional and environmental factors over time lead to a cycle of dysregulation of emotion, behavior, cognition, and relationships (Beauchaine et al., 2019; Crowell et al., 2009). We note that ACE scale incorporates indictors of physical abuse, sexual abuse, and neglect (Bornovalova et al., 2013; Ogata et al., 1990; Stepp, Scott, et al., 2016; Westen et al., 1990; Widom et al., 2009; Zanarini, 2000) as well as additional family-risk variables.

Clinical Implications

These findings have several clinical and public health implications. First, they highlight the longstanding effects of early experiences of adversity and trauma. Prevention of these childhood experiences, especially through public health initiatives, cannot be overemphasized. Second, we call upon other investigators to challenge the primacy of impulsivity per se by including global EF deficits in childhood as indicators of risk for BPD. This finding has implications for guiding early clinical assessment and intervention (e.g., through early EF skills

coaching) to prevent later BPD. In short, we highlight the need for interventions *before* risk increases, especially before the adolescent period (Beauchaine et al., 2019).

Children with histories of adversity/trauma and deficits in EF could receive targeted interventions such as Dialectical Behavior Therapy for children (DBT-C; Perepletchikova et al., 2017), Parent-Child Interaction Therapy (PCIT; Luby et al., 2018), or potentially an integration of these therapies in the future (Zalewski et al., 2020). Clearly, widespread assessment of these early risk factors to identify individuals at risk remains a challenge. Our findings also underscore the need for leading evidence-based treatments for severe dysregulation, including BPD, (i.e., DBT) for adolescents and adults to consider the role of EF deficits and inattentive symptomology in clinical treatments in addition to the focus on emotion dysregulation (e.g., increased emphasis of multimodal treatments).

Limitations and Future Directions

Our study has several important limitations. First, our sample size is small, with only 19 females meeting criteria for BPD in adulthood, clearly limiting statistical power. We emphasize the need for replication of findings. Note that we used Firth's penalized likelihood method to statistically account for our small sample (Adhikary & Rahman, 2021). Second, and relatedly, we were not able to assess mediating and moderating analyses and cannot add to the literature on mechanisms (e.g., emotion dysregulation)—an important area of future research. Third, we did not have symptom-level BPD data available, so that we were not able to analyze dimensions of BPD symptomology as related to predictors of interest. Future research would benefit from examining dimensional severity of BPD symptoms, as well as specific traits, some of which have recently been linked to increased risk for a suicide attempt (Yen et al., 2020). Fourth, our measure of externalizing symptoms (CBCL, ASR, and ABCL) included measures of aggression, but we did not examine aggression alone as related to risk for later BPD. Future research would benefit from examining aggressive symptomology across development, given empirical research and theory linking high levels aggression and peer problems with later BPD (Beauchaine et al., 2019; Stepp et al., 2012; Wolke et al., 2012). Fifth, we did not examine perinatal risk for later BPD, yet we believe future research efforts in this area are needed (see Winsper et al., 2015). Finally, a key limitation is the timing of our BPD measure. We measured BPD only during early adulthood, acknowledging that participants may already have met criteria for BPD in adolescence.

Even so, key strengths include a carefully diagnosed, ethnically and socioeconomically diverse, sample of females; emphasis on multiple-domain and multi-informant measures; high sample retention; and a prospective (and ongoing) longitudinal design. Moreover, we employed stringent use of covariates and statistical penalization. Finally, we examined multiple domains of risk for BPD simultaneously.

In summary, these findings add to existing research on developmental pathways to BPD, in particular among females with ADHD. Future directions must include further examination of dimensions of hyperactivity/impulsivity and inattention, as well as EF, across development as related to later BPD, as well as more sensitive measures of early emotional invalidation and replication in male samples.

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Table 1. Intercorrelations among study variables.

Variables of interest	1.	2.	3.	4.	5.	6.	7.	8.
1. W4 BPD	--							
2. W1 SNAP-HI Severity	0.17[b]	--						
3. W1 RCFT	0.22[a]	0.42[a]	--					
4. W1-W3 ACE	0.32[a]	0.24[a]	0.11	--				
5. W3 SNAP-HI Severity	0.4[a]	0.56[a]	0.28[a]	0.29[a]	--			
6. W3 SNAP-IA Severity	0.38[a]	0.54[a]	0.25[a]	0.18[a]	0.69[a]	--		
7. W3 Maternal BDI-II	0.16[b]	0.15[b]	0.04	0.22[a]	0.24[a]	0.28[a]	--	
8. W3 ASR/ABCL Int.	0.34[a]	0.33[a]	0.06	0.42[a]	0.56[a]	0.57[a]	0.34[a]	--
9. W3 ASR/ABCL Ext.	0.39[a]	0.51[a]	0.22[a]	0.33[a]	0.74[a]	0.69[a]	0.26[a]	0.73[a]

Note: W4: Wave 4; BPD: Borderline Personality Disorder; W1: Wave 1; SNAP: Swanson, Nolan and Pelham Questionnaire; HI: Hyperactivity/Impulsivity; RCFT: Rey Osterrieth Complex Figure Task; W3: Wave 3; ACE: Adverse Childhood Experiences Scale; IA: Inattention; BDI-II: Beck Depression Inventory-II; ASR: Adult Self-Report; ABCL: Adult Behavior Checklist; Int: Internalizing; Ext: Externalizing.

[a]Correlation significant at 0.01 level.

[b]Correlation significant at 0.05 level.

Table 2. Predictors of Risk for BPD

	No BPD (*n*=182) M(*SD*)	**BPD (*n*=19) M(*SD*)**	***p***	**Cohen's *d***	***p* with covariates[1]**
Childhood Predictors					
Impulsivity (W1 SNAP-HI Severity)	8.51(7.56)	12.89(7.96)	0.022*	0.58	0.094
Low Executive Functioning (W1 RCFT)	0.28(0.18)	0.42(0.22)	0.004**	0.76	0.031*
Childhood-Adolescent Predictors (Retrospectively Reported)					
Cumulative History of Childhood Adversity (ACE)	1.74(1.76)	3.84(2.63)	0.000***	1.14	0.005**
Late Adolescence Predictors					
Impulsivity (W3 SNAP-HI Severity)	4.98(4.66)	12.29(7.86)	0.000***	1.45	0.000***
Inattention (W3 SNAP-IA Severity)	8.65(6.52)	17.45(6.3)	0.000***	1.36	0.000***
Maternal Psychopathology (BDI-II)	5.92(7.88)	10.41(12.12)	0.046*	0.54	0.093
Internalizing Symptoms (W3 ASR/ABCL Int.)	52.46(10.67)	65.53(11.02)	0.000***	1.23	0.000***
Externalizing Symptoms (W3 ASR/ABCL Ext.)	53.74(10.19)	68.11(10.89)	0.000***	1.41	0.000***

Note: BPD: Borderline Personality Disorder; W1: Wave 1; SNAP: Swanson, Nolan and Pelham Questionnaire; HI: Hyperactivity/Impulsivity; RCFT: Rey Osterrieth Complex Figure Task; ACE: Adverse Childhood Experiences Scale; W3: Wave 3; IA: Inattention; BDI-II: Beck Depression Inventory-II; ASR: Adult Self-Report; ABCL: Adult Behavior Checklist; Int: Internalizing; Ext: Externalizing.
[1]Covariates: Race, Family SES at Wave 1; Participant age at Wave 4
*Correlation significant at 0.05 level.
**Correlation significant at 0.01 level.
***Correlation significant at 0.001 level.