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Adolescent Precursors of Adult Borderline Personality Pathology in a High Risk Community Sample

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Abstract

Longitudinal studies of the exact environmental conditions and personal attributes contributing to the development of borderline personality disorder (BPD) are rare. Further, existing research typically examines risk factors in isolation, limiting our knowledge of the relative effect sizes of different risk factors and how they act in concert to bring about borderline personality pathology. The present study investigated the prospective effects of diverse acute and chronic stressors, proband psychopathology, and maternal psychopathology on BPD features in a high risk community sample ($N = 700$) of youth followed from mid-adolescence to young adulthood. Multivariate analyses revealed significant effects of maternal externalizing disorder history, offspring internalizing disorder history, family stressors, and school-related stressors on BPD risk. Contrary to expectations, no interactions between chronically stressful environmental conditions and personal characteristics in predicting borderline personality features were detected. Implications of these findings for etiological theories of BPD and early screening efforts are discussed.

Keywords

adolescence; Borderline Personality Disorder; developmental psychopathology; diathesis-stress; young adulthood

Borderline Personality Disorder (BPD) is a severe mental disorder characterized by impulsive behavior, identity disturbance, emotional lability, and tumultuous social relationships. It is among the most prevalent of the personality disorders, reaching rates of 1–3% in the general population and nearly 10% in outpatient mental health settings (Lenzenweger, Lane, Loranger, & Kessler, 2007; Trull, Jahng, Tomko, Wood, & Sher, 2010; Zimmerman, Rothschild, & Chelminski, 2005). BPD is typically accompanied by high rates

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of functional impairment, health service use, and suicide (Skodol et al., 2002). As a result, research into the etiology and early detection of BPD represents a key public health priority.

Major theories of BPD posit that the disorder is a product of adverse early experiences and inherited vulnerability to emotion dysregulation (Crowell, Beauchaine, & Linehan, 2009; Linehan, 1993; Zanarini & Frankenburg, 1997). Yet, longitudinal investigations of the specific environments or personal characteristics hypothesized to confer risk for BPD are rare. A more comprehensive account of the adolescent antecedents of BPD would not only advance scientific knowledge of the origins of BPD but would also inform early screening and intervention programs designed to reduce the personal and societal burden of the disorder.

Environmental and Familial Antecedents

A preponderance of evidence indicates that adverse environmental conditions early in development are linked with subsequent risk for BPD. Retrospective studies in clinical samples suggest that the majority of patients with BPD are exposed to some form of early abuse or neglect (Horesh, Ratner, Laor, & Toren, 2008; Zanarini et al., 1997). Prospective data from the Children in the Community study demonstrate that early stressful experiences in the home and at school are risk factors for BPD in adolescence and adulthood (Cohen, 2008; Cohen, Crawford, Johnson, & Kasen, 2005). Research in adult clinical and community populations also finds that proximal acute life events trigger BPD symptomatology and that stressful life events may be more prevalent among those with BPD, relative to clinical disorder comparison groups (Distel et al., 2011; Jovev & Jackson, 2006). As others have noted (e.g., Bradley, Jenei, & Westen, 2005), the precise role of each of these putative etiological factors in determining risk for BPD is unclear, given that exposures to different classes of environmental stress are highly intercorrelated.

Research on the origins of BPD has also focused on psychopathology among family members, given moderate to high heritability estimates for BPD (Gunderson et al., 2011). Family studies consistently show that a broad array of psychiatric disorders—including major depression, substance use disorder, and antisocial personality disorder—is more common in first-degree relatives of those with BPD (e.g., Riso, Klein, Anderson, & Ouimette, 2000). In a recent longitudinal study of adolescent risk factors for adult BPD symptoms, Stepp and colleagues (2013) reported that parental substance use disorders, along with parental BPD, showed the strongest prospective association with offspring borderline personality features. As parents are responsible for transmission of environmental conditions as well as genetic information, it is likely that genetic risk for BPD (due to parental disorder) overlaps with maladaptive parenting behaviors that contribute to deleterious family environments shown to put offspring at risk for BPD (Johnson, Cohen, Chen, Kasen, & Brook, 2006).

Intrapsychic Antecedents

Another important sector of developmental research in BPD is the investigation of personality trait profiles and clinical disorders in adolescence that portend BPD. Several

theorists have posited that trait negative affectivity and disinhibition constitute the core of borderline pathology (Depue & Lenzenweger, 2001; Paris, 2005; Siever & Davis, 1991). This hypothesis has been corroborated by empirical investigations of personality traits underlying BPD risk, several of which have found that negative affectivity exhibits a stronger association with borderline symptomatology than does disinhibition (Gratz, Latzman, Tull, Reynolds, & Lejuez, 2011; Trull, 2001; see also James & Taylor, 2008). In contrast, other research groups have documented that, as compared to internalizing syndromes, externalizing disorders—presumed to represent manifestations of a latent disinhibition trait (see Krueger & Markon, 2006)—exhibit more robust prospective associations with borderline pathology (Bernstein, Cohen, Skodol, Bezirgianian, & Brook, 1996; Helgeland, Kjelsberg, & Torgersen, 2005; Stepp, Burke, Hipwell, & Loeber, 2012).

Present Study

The present study aimed to combine risk factors explored in previous research into a more comprehensive developmental model of borderline pathology in a community sample at high risk for psychopathology due to maternal depression. We built on recent research by Stepp and colleagues (2013) from the Oregon Adolescent Depression Project (OADP). These authors examined adolescent and parental psychopathology, along with several facets of family functioning, at proband age 16 as predictors of BPD symptoms at age 30. In a multivariate model, offspring and father substance use disorder, offspring depression, maternal BPD, and elevated maternal-child discord during adolescence emerged as unique predictors of borderline pathology.

We expanded on this previous study by assessing a more extensive set of potentially adverse environmental conditions in adolescence, including occurrence of acute stressors and chronic stressors across individual, family, peer, and academic contexts. Additionally, we examined the joint effects between environmental stressors and personal characteristics to test the general hypothesis that BPD results from the interaction between pathogenic environments and individual vulnerabilities (see Crowell et al., 2009; Linehan, 1993). Specifically, we predicted that stressful conditions would lead to heightened borderline pathology especially among adolescents with a history of internalizing and/or externalizing disorders.

Aside from contextually-based assessment of environmental risk factors, our model included adolescent clinical disorders from both internalizing and externalizing spectrums. These sets of disorders were presumed to be manifestations of underlying propensities to internalizing distress and disinhibited behavior, two traits that, as previously mentioned, are theorized to be central to borderline pathology (Siever & Davis, 1991). Maternal internalizing disorders, externalizing disorders, and self-reported BPD symptoms were also examined in light of consistent evidence that they are all over-represented in first-degree relatives of BPD probands (Riso et al., 2000). Gender was not considered as a risk factor in the present study given inconsistent evidence for gender differences in BPD prevalence (Lenzenweger et al., 2007; Torgersen et al., 2001; Widiger & Trull, 1993), but it was included in all models as a covariate.

Our primary aims were to clarify the unique roles of each of these vulnerabilities and to examine how they act in concert to increase liability to BPD at the transition to adulthood. We hypothesized that all risk factors would evidence statistically significant zero-order correlations with young adult borderline pathology, and we expected that many of the risk factors would no longer exert unique effects on offspring BPD symptoms after accounting for overlap with other vulnerabilities. As stated above, we hypothesized that acute and chronic stressors would be more predictive of BPD symptoms among adolescents with a prior history of internalizing or externalizing difficulties.

Methods

Participants

A sample of 815 15-year-old youth was selected from the Mater-University Study of Pregnancy (MUSP) in Brisbane, Australia (Keeping et al., 1989), which followed a birth cohort of over 5,000 mothers and their offspring born between 1981 and 1984 at the Mater Misericordiae Mother's Hospital to study children's health and development. Responses to peripartum mood questionnaires were used to select a sample of adolescents whose mothers had a wide range of depressive experiences. Out of the 815 mothers in the original sample, 354 (43.4%) had a lifetime history of major depression or dysthymia according to diagnostic assessments at offspring age 15. Full details of the sampling procedure are provided in Hammen, Shih, Altman, & Brennan (2003).

When offspring reached age 20, they were recontacted and invited to participate in a follow up assessment. Seven hundred offspring (362 females; 85.9% of the sample for the age 15 assessment) were available and completed BPD assessments at age 20. The final sample was 92% Caucasian and 8% minority (Asian, Pacific Islander, and Aboriginal). The median family income fell in the lower middle class and mothers' median education level was grade 10. Youth participating at age 20 did not differ from those participating at age 15 but not 20 in terms of family income at age 15 ($t(782) = -1.49, p = .14$), maternal depression history by age 15 ($\chi^2(1, 815) = 0.18, p = .67$), or history of any depressive, anxiety, or externalizing disorder by age 15 ($\chi^2 s < 1, ps > .10$). Youth not participating at age 20 were more likely to be male ($\chi^2(1, 815) = 11.08, p < .01$).

Procedures

Interviews to assess stress exposure and diagnostic status were administered separately to youth and mothers in their home at youth age 15. At age 20, youth completed questionnaire and interview measures to assess borderline pathology. Interviewers were advanced graduate students in psychology and were blind to maternal and offspring psychiatric history. All participants gave their written informed consent (or assent), and offspring were compensated AU\$15 at the age 15 timepoint and AU\$50 at the age 20 timepoint. All procedures were approved by the UCLA Institutional Review Board, Emory University Investigations Committee, and the University of Queensland Ethics Review Committee.

Measures

Offspring Clinical Disorder Diagnoses—The Schedule for Affective Disorders and Schizophrenia in School-Aged Children (K-SADS-E; Orvaschel, 1995) was administered during age 15 data collection to determine offspring current and lifetime diagnoses of psychiatric disorders. The K-SADS-E is a widely used and validated semi-structured interview for assigning clinical disorder diagnoses in children and adolescents with well-established reliability and concurrent validity estimates in past research (Kaufman et al., 1997). Diagnoses were assigned if either the adolescent or maternal interview indicated that the adolescent qualified for a given syndrome. Interrater reliability was assessed using a random sample of 75 K-SADS-E interview recordings evaluated by clinicians blind to the original diagnostic ratings. Weighted kappas were in the acceptable range (i.e., greater than 0.75) for all internalizing and externalizing disorders. If offspring endorsed a depressive or anxiety disorder, they were judged to have a lifetime history of internalizing psychopathology; likewise, substance use and disruptive behavior disorders were considered when determining whether offspring had a history of externalizing psychopathology.

Offspring Borderline Personality Disorder Symptoms—Offspring BPD symptomatology at age 20 was assessed using the Structured Clinical Interview for *DSM-IV* Axis II Personality Disorders, Version 2.0 (SCID-II, First, Spitzer, Gibbon, Williams, & Benjamin, 1994) administered to the youth. The SCID-II instrument has been studied extensively in prior PD research, and it has shown excellent psychometric properties, including convergent validity with other semistructured interviews (Crawford et al., 2005; Schotte et al., 2004; Skodol, Oldham, Rosnick, Kellman, & Hyler, 1991). Following administration guidelines (First et al., 1994), the SCID-II self-report questionnaire was administered first to screen for the presence of all PD symptoms, and the SCID-II interview probed only those symptoms that were endorsed in the self-report screening phase. The SCID-II interview yields both a dimensional and a categorical assessment of BPD (i.e., diagnosis is present if at least 5 of 9 criteria are met). The number of BPD symptoms present (i.e., dimensional score) is used as the dependent variable in the present analyses (see Conway, Hammen, & Brennan, 2012a, for evidence of a unidimensional trait underlying BPD symptoms in the present sample). Cronbach's alpha for the 15 self-report items (*DSM-IV* criteria 3, 5, and 8 were assessed with multiple items) was 0.78, and kappa coefficients indexing the inter-rater reliability for each symptom across a randomly-selected sample of 34 respondents ranged from 0.76 to 1.0 (median = 0.96).

Maternal Clinical Disorder Diagnoses—Maternal psychopathology was assessed using the Structured Clinical Interview for *DSM-IV* (SCID; First, Spitzer, Gibbon, & Williams, 1995). The SCID is supported by extensive reliability and validity data (First et al., 1995; Kessler et al., 2005), and is one of the most widely used measures of clinical disorders. As for offspring, the SCID was administered at the age 15 assessment and documented current and lifetime history of clinical disorder. Inter-rater reliability based on ratings of independent judges was very good for all diagnoses (i.e., kappa values greater than 0.80). SCID diagnoses were used to determine maternal histories of internalizing (any depressive or anxiety disorder) and externalizing (any substance use or antisocial disorder) psychopathology.

Maternal Borderline Personality Disorder Symptoms—Mothers answered questions about their own BPD symptoms using the self-report PD section of the Structured Clinical Interview for *DSM-III-R* (SCID-Q; Spitzer, Williams, Gibbon, & First, 1990) at offspring age 15. The BPD subscale of this instrument includes 13 true or false questions related to the 8 BPD criteria in *DSM-III-R*. Since the stress-linked paranoia criterion was only added in *DSM-IV*, this symptom was not assessed on the SCID-Q. Prior research has shown this subscale to have good internal consistency (Cronbach's alpha range: .75–.80), stability estimates that are comparable to normal-range personality dimensions, and strong associations with clinical disorder features (see Ball, Rounsaville, Tennen, & Kranzler, 2001). The count of symptoms endorsed served as a dimensional index of maternal borderline pathology ($M = 2.67$, $SD = 2.72$). One prior study in this dataset demonstrated that maternal BPD symptoms are cross-sectionally associated with offspring social dysfunction even after controlling for offspring depression (Herr, Hammen, & Brennan, 2008), supporting the concurrent validity of this dimensional measure of maternal BPD symptomatology. Cronbach's alpha internal consistency reliability estimate for the 13 BPD items was 0.79.

Acute Stress—Acute life events occurring over the past year and chronic stress occurring over the past six months was assessed at age 15 using the semi-structured UCLA Life Stress Interview (LSI; Hammen, Henry, & Daley, 2000) administered by trained interviewers. Subjective ratings of the impact of events and ongoing conditions may confound perceived severity with symptomatology, and actual symptomatology may bias individual perceptions of the significance and impact of events and circumstance. Therefore, the goal was to obtain information that could be rated as objectively as possible by independent raters without taking the interviewee's actual reactions into consideration.

Interviewers obtained information about each acute life event in the past six months, gathering specific information about circumstances surrounding the event, so that contextual information could be presented to an independent rating team, blind to the interviewee's actual reaction to the event. The team judged how a "typical" person in similar situations would experience the impact of the event. Raters judged the severity of each event on a 5-point scale with higher scores representing more severe impact. The rating team also rated independence, the extent to which each event's occurrence was independent of the actions of the individual, on a 5-point scale ranging from completely fateful (independent) to completely caused by the person (dependent). In the present study, interrater reliabilities based on independent ratings by separate teams for 89 cases yielded intraclass correlations (ICCs) of .92 for severity rating and .89 for independence.

Chronic Stress—The UCLA LSI also assessed severity of stressfulness of ongoing circumstances across 6 domains in the past 6 months: best friendship, romantic relationship, relationships with family members, peer relationships, academic performance, and school behavior. Interviewers used behaviorally-specific scales ranging from 1 (no stress; superior circumstances) to 5 (severe stress; major difficulties) to score each domain as objectively as possible. For example, on the peer relationships scale, a "2" indicated good social life including some close friends; engages in average number of social activities; and good

quality of social contacts with no significant problems with peers. A “4” indicated serious social problems; somewhat isolated from peers and spends much time alone, or some acquaintances but lacks stable friendships; or has one or two friends but frequent conflicts. The LSI chronic stress ratings have demonstrated good reliability, predictive validity, and concurrent validity in previous investigations (e.g., Daley, Hammen, & Rao, 2000; Hammen, Kim, Eberhart, & Brennan, 2009; see Hammen, 2005, for a review). For example, convergent validity of the chronic stress ratings was provided by comparisons with independent sources of information on similar functions where available. Academic performance chronic stress ratings correlated significantly with teacher-reported grades (Teacher Report Form; Achenbach, 1991) for up to four courses (r s ranged from $-.51$ to $-.59$) and $-.56$ for performance in the teacher’s own class, all p s $< .001$). Teachers’ ratings of youth peer popularity and social functioning at school were correlated with interview-based ratings of youth social life, r s = $-.23$ and $.24$, p s $< .001$, respectively (Hammen, Brennan, & Keenan-Miller, 2008). An average ICC of $.77$ in the present study suggested good reliability between independent ratings for each chronic stress domain. In a separate sample of high school women the average ICC estimate across the chronic stress domains was $.87$ (range $.82-.91$; Rao, Hammen, & Daley, 2000).

Data Analytic Plan

As a first step, zero-order correlations between putative risk factors and borderline pathology were examined to compare the degree of association across risk factors. Second, all 12 risk factors—including 6 chronic stress domains; total acute stress exposure¹; maternal internalizing, externalizing, and borderline personality pathology; and offspring internalizing and externalizing pathology—and gender were simultaneously entered into a negative binomial regression analysis to evaluate their unique prospective influences on BPD symptoms. Negative binomial regression analyses were appropriate given that BPD symptoms were treated as a count variable and the variance of symptoms was not approximately equal to the mean (Gardner, Mulvey, & Shaw, 1995). Third, interactions between stress domains and adolescents’ internalizing and externalizing characteristics were tested in the prediction of borderline pathology.

All continuous predictor variables were standardized in the regression analyses reported below to enhance interpretability of effect sizes and minimize multicollinearity (Aiken & West, 1991). Incidence rate ratios (IRR), or the factor by which the incidence of BPD symptoms is expected to change per unit increment in the predictor, are presented here as effect size measures (e.g., an IRR of 1.21 indicates that the expected incidence of BPD symptoms increases by a factor of 1.21, or 21%, for a one unit increment in the predictor). All analyses were conducted using STATA 12.1 (StataCorp, 2011).

¹Preliminary analyses examined whether independent and dependent acute stressors should be examined separately. *Dependent* stressors refer to those that are caused at least in part by the individual’s own actions. *Independent* stressors, on the other hand, are fateful and are not influenced by the individual. These separate types of stressful events have occasionally demonstrated different patterns of association with internalizing disorders in prior research (see Hammen, 2005). However, in the present data, the effect of stress exposure on BPD features did not differ for dependent versus independent events ($z = 1.74$, $p = .08$) and they were therefore collapsed into one acute stress exposure variable in all analyses.

Results

An average of 0.65 interviewer-rated BPD symptoms ($SD = 1.54$) was present at age 20, and 16 individuals (2.3% of the sample) qualified for a *DSM-IV* diagnosis of BPD. Depressive and anxiety disorders, as compared to substance use and antisocial disorders, were more prevalent among both mothers and offspring (see Table 1). Depressive disorders were especially common among mothers, consistent with the sampling procedures described above. Table 2 shows that internalizing and externalizing disorders were moderately correlated among both mothers and offspring.

Bivariate analyses were conducted on a preliminary basis to examine the prospective effects of each risk factor on borderline pathology in isolation. As seen in Table 3, 10 of the 12 putative risk factors exhibited a statistically significant and positive zero-order association with BPD symptoms at age 20. Additionally, females tended to endorse higher rates of symptoms than did males, although this effect was only marginally statistically significant. There was some variation in the magnitude of bivariate associations among risk factors and BPD symptoms, with offspring diagnostic characteristics exerting stronger effects than maternal diagnoses and family- and school-related stressors having more robust influences than other domains of chronic stress.

To identify the unique effect of each risk factor, all predictors were simultaneously entered into a negative binomial regression (see Table 3). After accounting for the covariation among risk factors, only maternal externalizing disorder ($IRR = 2.74$), offspring internalizing disorder ($IRR = 1.60$), school stressors ($IRR = 1.52$), and family stressors ($IRR = 1.34$) were significantly associated with BPD symptoms. Results were equivalent when stress domains that were not related to BPD in the full model were omitted, with one exception: in the reduced model, the effect of gender on borderline pathology reached statistical significance ($b = 0.36$, $SE = 0.18$, $p < .05$, $IRR = 0.70$), with women reporting 30% more BPD symptoms than men.

To investigate the general hypothesis that adverse environmental conditions and adolescent personal characteristics have a synergistic effect on risk for BPD, the interactions of acute stress exposure and the various chronic stress domains with offspring clinical disorders in predicting borderline pathology were examined. Contrary to predictions, none of the 6 types of ongoing stress moderated the associations between adolescent internalizing or externalizing problems and future BPD symptoms ($ps > .10$). Acute stress exposure interacted with internalizing ($b = -0.51$, $SE = 0.19$, $p < .01$), but not externalizing disorder history ($b = 0.06$, $SE = 0.30$, $p = .83$), in predicting BPD features, but in an unexpected fashion. That is, acute stress had a significant effect for those without an internalizing diagnosis ($b = 0.24$, $SE = 0.09$, $p < .01$), whereas stress had no pathogenic effect for those with an internalizing diagnosis ($b = -0.10$, $SE = 0.15$, $p = .57$). Secondary analyses that examined the interaction between adolescent disorders and gender also revealed that the strength of effects of adolescent psychopathology on BPD symptoms did not vary for men versus women ($ps > .10$).

Discussion

The present study investigated the prospective effects of multiple adolescent vulnerability factors on adult BPD symptomatology in a high risk sample. Our analyses revealed that 10 out of the 12 putative risk factors demonstrated significant zero-order correlations with BPD features, but some of these associations were attenuated in a multivariate context. When all risk factors were accounted for, vulnerabilities from individual, family, and school domains during mid-adolescence were unique antecedents of borderline pathology at the transition to adulthood.

Regarding the family domain, both chronically stressful family conditions and maternal externalizing problems at proband age 15 were independently linked to offspring symptomatology five years later. The former finding converges with a line of research documenting that adult BPD patients report higher rates of adverse family environments in childhood and adolescence (e.g., Zanarini et al., 1997). The present study expands on prior research by demonstrating a unique effect of family stress—including deficiencies in communication, trust, availability, and conflict resolution—even after accounting for several other family characteristics (i.e., maternal mental illnesses) and ongoing stressors across other domains. Maternal externalizing disorders exhibited the strongest effect out of all risk factors on offspring BPD symptoms in the multivariate model, highlighting the unique importance of mothers' disinhibited behavior in the development of offspring BPD symptoms. Maternal internalizing disorders, in contrast, were not related to BPD outcome on either a univariate or multivariate basis. Maternal BPD symptoms showed a significant zero-order correlation with offspring BPD symptoms, but did not influence offspring symptoms over and above the effects of other risk factors. This result suggests that there is no one-to-one relationship between maternal BPD symptoms and offspring BPD symptoms; instead, other maternal characteristics may contribute equally (perhaps even more strongly) to the development of offspring borderline pathology. This interpretation is consistent with prior family studies that document higher rates of diverse mood, anxiety, and substance abuse disorders in first-degree relatives of BPD patients (Riso et al., 2000). Indeed, research has yet to convincingly disentangle the effects of multiple parental disorders on offspring risk for BPD.

Although both adolescent internalizing and externalizing histories were prospectively associated with BPD symptoms in bivariate analyses, only adolescent internalizing psychopathology continued to predict borderline pathology after controlling for the presence of other risk factors. The influence of internalizing disorder history on risk for BPD accords with the notion that trait negative affectivity is a core component of BPD (Siever & Davis, 1991), and several investigations that have found trait negative affectivity to be more closely related to borderline pathology than trait disinhibition (Gratz et al., 2011; Trull, 2001). On the other hand, the null result for externalizing disorders in the final model stands in contrast to several prior studies that have shown externalizing problems (e.g., attention deficit hyperactivity disorder, disruptive behavior disorders) to be especially potent predictors, relative to emotional disorders, of BPD trajectories through adolescence and into adulthood (Burke & Stepp, 2012; Helgeland et al., 2005). It could be speculated that in these prior studies the effects of externalizing disorders and (unmeasured) ongoing stressors were

conflated to some extent, leading to an overestimate of the unique externalizing-BPD association. For instance, in the present data, history of externalizing problems was moderately correlated with chronic family stress and school-related stress ($r_s = .32$ and $.44$, respectively). Alternatively, the lower prevalence of externalizing disorders in the present study could account for the discrepant findings. Additional research assessing both externalizing dysfunction and chronically stressful conditions, especially in relation to adjustment in educational settings, may be needed to resolve these inconsistencies.

Stressful conditions at school represented another unique prospective risk factor for borderline pathology in the present study. That is, adolescents' difficulties with getting along with other students and teachers, attendance problems, and infractions leading to suspension or expulsion signaled risk for elevated BPD symptomatology. Clearly, the majority of these stressful experiences are at least partially dependent on adolescents' own behavior, and trait disinhibition in particular is known to evoke at least a subset of these stressors (Conway, Hammen, & Brennan, 2012b). However, school-related chronic stress continued to predict borderline features even after statistically controlling for adolescent externalizing disorder history, suggesting that success in negotiating the social environments and regulations associated with high school education is an important indicator of BPD-proneness in its own right. This finding is consistent with results from a 28-year longitudinal study that showed school-specific behavioral difficulties in early adolescence, as rated by teachers, predict the development of borderline personality symptoms in emerging adulthood (Carlson, Egeland, & Sroufe, 2009). In the same vein, Trull and colleagues (1997) reported that borderline features were strong correlates of academic probation and suspension in a college student sample.

It is informative to compare the results from the present investigation with those from the recent study of Stepp and colleagues (2013) in light of similarities in samples and designs. In general, findings from both projects suggest that adolescent clinical disorders, maternal clinical disorders, and adverse family conditions combine to shape offspring borderline pathology. Additionally, both internalizing and externalizing mental illnesses appear relevant, consistent with several psychobiological perspectives on BPD (Depue & Lenzenweger, 2001; Siever & Davis, 1991). Offspring internalizing history and family discord were independent predictors of BPD risk in both studies, and the relative magnitude of effect sizes between these two risk factors was equivalent across investigations. At the same time, there were some noteworthy inconsistencies in results between the studies. For instance, the strongest predictor of adult BPD in the OADP was proband substance use disorder, whereas in the present study externalizing problems carried almost no predictive power in the final model, although they were the most robust diagnosis-based predictor of BPD symptoms at the bivariate level. As mentioned above, this discrepancy may be attributed to the moderate overlap in the present sample between externalizing disorders and elevated exposure to various types of chronically stressful conditions, which also had substantial effects on BPD risk.

A distinctive feature of the present study was the examination of joint effects between adolescent characteristics and adverse environmental conditions. This interactive effect was hypothesized as an extension—or indirect evaluation, given that propensity to internalizing

or externalizing dysfunction served as a proxy for emotion dysregulation—of Linehan's (1993) developmental model of BPD that posits a positive feedback loop between emotion dysregulation and pathogenic environmental responses. Contrary to expectations, we did not observe a consistent pattern of interactions between stressful experiences and adolescent history of psychopathology on BPD symptoms. No joint effects were detected for the chronic stress domains, and acute stressors were more pathogenic for those without a history of internalizing disorder. Replication studies will be needed to evaluate the reliability of this unanticipated finding.

The present data are also relevant to the puzzling question of gender differences in BPD. The large majority of patients diagnosed with BPD in clinical settings are women (Widiger & Trull, 1993), yet studies in community populations, including several large epidemiological surveys, have not consistently detected gender differences in BPD (Lenzenweger et al., 2007; Torgersen et al., 2001; Trull et al., 2010). Our results do not convincingly support or refute the possibility of gender differences in borderline pathology. Women endorsed approximately 30% more BPD symptoms than men, but this effect was not statistically significant in the full multivariate analysis. Additional research is clearly needed to identify the nature and causes of gender differences in BPD symptoms in community populations.

Limitations

Several limitations of the present study should be considered when evaluating the results. First, there are several potential caveats to our tests of person-environment interaction. It is possible that our stress measures did not capture the specific environmental experiences that are most etiologically relevant to BPD (e.g., invalidation, abuse, neglect; see Crowell et al., 2009). Also, it may be that trait negative affectivity and disinhibition—theorized to represent the diatheses of offspring internalizing and externalizing pathology, respectively, assessed in the present study—potentiate the BPD-inducing effects of adverse environments only in earlier developmental stages. For instance, Linehan (1993) posited that reciprocal influences between emotion dysregulation and invalidating responses from significant others commences in early childhood. Thus, our analysis, while consistent with the diathesis-stress framework, did not provide a direct test of the interactions hypothesized by Linehan and colleagues (Linehan, 1993; Crowell et al., 2009). More targeted assessment of theory-based environmental constructs, across different developmental epochs, is needed in future longitudinal research on BPD.

Second, rates of maternal internalizing disorder were relatively high due to a sampling procedure that overselected mothers with a history of unipolar depression. On one hand, the high risk design and concomitant higher base rates of offspring borderline pathology could be considered methodological advantages. On the other hand, comparisons to unselected community samples (e.g., Stepp et al., 2013) may be more complicated. It is also interesting to note that we did not find the expected relation between maternal internalizing disorder and offspring BPD features, although it is unclear to what extent this result can be explained by relatively high prevalence of maternal internalizing disorders.

Third, while we assumed that the presence of internalizing and externalizing disorders reflected trait elevations on negative affectivity and disinhibition, respectively, we were not

able to directly assess these latter constructs. Additional research that incorporates measures of five-factor model personality traits into developmental studies of BPD will be better suited to addressing diathesis-stress hypotheses (e.g., Trull, 2001). Fourth, maternal borderline pathology was assessed via self-report—without a subsequent interview to confirm the severity and persistence of symptoms as was the case with offspring SCID-II assessment—in the present study, and those symptom counts may not have been as faithful an index of borderline pathology as a BPD diagnosis assigned via gold-standard interview techniques. Transient elevations on the BPD questionnaire due to mood-state effects may have attenuated the association between maternal and offspring BPD symptoms in the multivariate model. Finally, offspring BPD symptoms were not assessed in mid-adolescence, and we could therefore not determine the portion of the observed effects that was independent of continuity in borderline pathology. Future developmental research with BPD assessments at multiple timepoints is needed to examine risk factors that change the trajectory of BPD symptoms over time. Along these same lines, longitudinal studies are needed to account for the possible influence of BPD on stress exposure. Although in the present study stressors were assessed five years prior to BPD symptoms, it is possible that incipient borderline pathology increased the likelihood of certain stressful conditions. Future investigations with multiple BPD assessments can clarify the dynamic relations between BPD symptoms and stress over time.

Clinical Implications

Despite these limitations, the present results may be useful for informing certain areas of clinical practice. For instance, effect size estimates from the multivariate model could contribute to the development of an early detection algorithm to more precisely identify young people at risk for BPD. Several intervention programs for adolescents are effective in shifting the trajectory of borderline pathology (Chanen & McCutcheon, 2013), and more comprehensive developmental models of BPD can improve accessibility of these programs for at-risk youth. Additionally, our results point to a number of possible targets for intervention or prevention programs. For instance, a family-based intervention focused on the influence of maternal externalizing problems on adolescent emotional development could buffer adolescents from the pathogenic effects of maternal externalizing disorders observed here. Substance misuse and antisocial behavior likely interfere with mothers' capacity to deliver validation, model adaptive coping strategies, and maintain secure attachment styles, all of which are processes emphasized in prominent developmental models of BPD (e.g., Linehan, 1993; Zanarini & Frankenburg, 1997). Further, the sizable effect of chronically poor school functioning on young adult BPD symptoms indicates that school-based intervention may be a useful supplement to family and/or individual psychotherapies.

Conclusions

Perhaps the main contribution of this study is the demonstration that any theory of BPD focused exclusively on one etiological system is almost certainly incomplete. For instance, our data indicate that BPD is not entirely attributable to inherited BPD-proneness, problematic parenting, or a complication of a depressive disorder. Indeed, it appears that theoretical integration, consistent with the explanatory pluralism perspective (see Kendler,

2005), is needed for an accurate model of BPD risk, and a pressing task for future research is to test multifactorial developmental models of BPD. In the same vein, current opinions and guidelines regarding the relative importance of various liability markers for BPD may require revision, given that much of the relevant research has focused on one particular risk factor or another in isolation. That is, the present data show that the effect size assigned to any one risk factor may be substantially inflated if commonly co-occurring vulnerabilities are not taken into account. This limitation in the research literature introduces uncertainty for clinicians interested in discerning whether a young patient is at risk for borderline pathology. Much more investigation is needed on complex predictive algorithms for BPD in order to guide clinical risk assessments and identify the BPD precursors that warrant the most attention in prevention or intervention settings. We hope that this study and studies like it can advance both theoretical and applied efforts to determine who is vulnerable to BPD and, ultimately, how to prevent it.

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Table 1

Descriptive Statistics for Risk Factor Variables

Risk Factor	Offspring		Mother	
	<i>N</i>	%	<i>N</i>	%
<i>Categorical Predictors</i>				
Depressive Disorder	97	13.9	310	44.3
Anxiety Disorder	69	9.9	160	22.9
Disruptive Behavior Disorder	34	4.9	2	0.3
Substance Use Disorder	15	2.1	36	5.2
<i>Continuous Predictors</i>				
	<i>M</i>	<i>SD</i>		
Maternal BPD Symptoms	2.67	2.72	—	—
Close Friendship Stress	2.21	0.50	—	—
Romantic Relationship Stress	2.15	0.37	—	—
Peer Relationship Stress	2.28	0.47	—	—
Family Relationship Stress	2.34	0.58	—	—
Academic Performance Stress	2.68	0.94	—	—
School Behavior Stress	2.19	0.78	—	—
Acute Stressful Life Events	6.03	3.98	—	—

All analyses based on the sample participating in the follow up assessment at offspring age 20. *N*= number of participants qualifying for a diagnosis. Maternal BPD symptoms were assessed via mothers' self-report; offspring clinical disorders were assessed via interviews to both mothers and offspring; all other variables were assessed via offspring interview.

Table 2

Correlation Matrix for Main Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Offspring Internalizing Disorder	—													
2. Offspring Externalizing Disorder	.36	—												
3. Maternal Internalizing Disorder	.28	.31	—											
4. Maternal Externalizing Disorder	.12	.16	.44	—										
5. Maternal BPD Symptoms	.09	.14	.32	.13	—									
6. Close Friendship Stress	.13	.05	.09	.09	.07	—								
7. Romantic Relationship Stress	.18	.07	.03	.02	.02	.07	—							
8. Peer Relationship Stress	.18	.13	.09	.07	.07	.51	.16	—						
9. Family Relationship Stress	.21	.32	.20	.10	.14	.16	.18	.20	—					
10. Academic Performance Stress	.14	.27	.04	-.03	.07	.09	.06	.15	.21	—				
11. School Behavior Stress	.15	.44	.08	.06	.10	.05	.09	.13	.31	.57	—			
12. Gender	-.15	.08	.02	.02	.01	.10	-.12	.02	-.05	.16	.14	—		
13. Offspring BPD Symptoms	.22	.28	.07	.16	.10	.03	.09	.13	.25	.17	.26	-.07	—	
14. Acute Stressful Life Events	.18	.22	.10	-.05	.02	-.11	.13	-.01	.19	.09	.25	-.21	.17	—

Correlations involving categorical or ordinal variables are polychoric or polyserial, given evidence that a continuous latent dimension underlies the diagnostic constructs assessed in this study. Correlations greater than |.07| are significant at the .05 level (uncorrected). For gender, 0 = female, 1 = male.

Table 3
 Negative Binomial Regression of Borderline Personality Symptoms on Psychiatric Diagnoses and Chronic Stress Domains

Predictor	Univariate			Multivariate		
	<i>b</i>	<i>SE</i>	IRR	<i>b</i>	<i>SE</i>	IRR
Offspring Internalizing Disorder	0.90	0.21	2.47	0.47	0.23	1.60
Offspring Externalizing Disorder	1.33	0.35	3.79	-0.17	0.39	0.85
Maternal Internalizing Disorder	0.25	0.19	1.28	-0.17	0.20	0.85
Maternal Externalizing Disorder	0.80	0.39	2.22	1.01	0.38	2.74
Maternal BPD Symptoms	0.20	0.09	1.22	0.07	0.09	1.07
Close Friendship Stress	0.09	0.09	1.09	-0.03	0.11	0.97
Romantic Relationship Stress	0.19	0.09	1.20	0.06	0.09	1.06
Peer Relationship Stress	0.28	0.10	1.32	0.17	0.11	1.19
Family Relationship Stress	0.55	0.10	1.73	0.30	0.11	1.34
Academic Performance Stress	0.40	0.10	1.45	0.05	0.11	1.05
School Behavior Stress	0.53	0.10	1.70	0.42	0.12	1.52
Acute Stressful Life Events	0.35	0.09	1.41	0.18	0.10	1.20
Gender	-0.34	0.19	0.71	-0.32	0.19	0.80

For gender, female = 0, male = 1. IRR = incidence rate ratio. Continuous variables were standardized in the regression. All effects with $p < .05$ are displayed in bold font.