Published in final edited form as:
Personal Disord. 2013 July ; 4(3): . doi:10.1037/per0000015.

Unique Influences of Adolescent Antecedents on Adult Borderline Personality Disorder Features

Stephanie D. Stepp, **Thomas M. Olino**, **Daniel N. Klein**, **John R. Seeley**, and **Peter M. Lewinsohn**
From the University of Pittsburgh School of Medicine, Pittsburgh, PA (Drs. Stepp and Olino), Stony Brook University (Dr. Klein) and the Oregon Research Institute, Eugene, OR (Drs. Seeley and Lewinsohn)

Abstract

There is a dearth of prospective information regarding adolescent precursors of borderline personality disorder (BPD). This study aims to determine the unique associations between early maladaptive family functioning, parental psychiatric diagnoses, proband early-onset psychiatric diagnosis and BPD symptoms in adulthood using an existing longitudinal study. Participants were randomly selected from nine high schools in western Oregon. A total of 1,709 students (ages 14-18 years) completed two assessments during adolescence. All adolescents with a history of a depressive disorder ($n = 360$) or a history of non-mood disorders ($n = 284$), and a random sample of adolescents with no history of psychopathology ($n = 457$) were invited to participate in a third and fourth evaluation when participants were on average 24 years and 30 years, respectively. Biological parents were interviewed at the third assessment. The multivariate model with all early risk factors found that maternal-child discord ($p < .05$), maternal BPD ($p < .05$), paternal Substance Use Disorder (SUD) ($p < .05$), and proband depression ($p < .05$), SUD ($p < .001$), and suicidality ($p < .05$) were associated with later BPD symptoms. Maternal SUD and proband anxiety, Conduct Disorder/Oppositional Defiant Disorder, and Attention Deficit Hyperactivity Disorder were also associated with proband BPD symptoms in univariate analyses, but were no longer significant when the other risk factors were included in the model. Multivariate assessment models are needed to identify unique risk factors for Borderline Personality Disorder. This will enhance the efficiency of screening efforts for early detection of risk.

Prominent theories of Borderline Personality Disorder (BPD) propose that the disorder results from a combination of individual characteristics and early experiences, particularly transactions between the child and caregivers (e.g., Bateman & Fonagy, 2003; Kernberg, 1984; Linehan, 1993). Consistent with these theories, antecedents of BPD can be traced to experiences and events during early development, including parental history of psychiatric disorder (White, Gunderson, Zanarini, & Hudson, 2003), early maladaptive family experiences (Fruzzetti, Shenk, & Hoffman, 2005; Zanarini et al., 1997), and early-onset psychiatric disorder (Zanarini et al., 2006). Much of our knowledge regarding these associations is limited to adults with BPD retrospectively reporting about childhood events (Zanarini et al., 2006). Retrospective assessment of perceptions of childhood experiences among adults with BPD is problematic since the illness may impact the validity of the reports. Few longitudinal studies are able to examine the prospective links between early experiences and BPD in adulthood. However, more empirical work has examined the prospective association between childhood psychiatric diagnoses and subsequent BPD in

Corresponding author: Stephanie D. Stepp, Ph.D., Western Psychiatric Institute and Clinic, 3811 O'Hara St., Pittsburgh, PA 15213, steppsd@upmc.edu.

Disclosures: All authors have no financial interests or affiliations relevant to the subject of this manuscript.

adulthood (e.g., Lewinsohn, Rohde, Seeley, & Klein, 1997; Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001). Longitudinal studies that assess personality pathology (the Children in the Community study has been at the forefront of this line of scientific inquiry (Cohen, Crawford, Johnson, & Kasen, 2005)) typically collapse symptoms across PD clusters or create a general PD severity score without examining developmental pathways specific to BPD.

Previous studies have largely focused on one set of early risk factors to predict BPD, or personality pathology more generally, in adulthood. Dysfunctional family environments, parental psychiatric disorders, and early-onset psychiatric diagnoses have been identified as important precursors to BPD in adulthood. Many of these risk factors are interdependent and testing univariate associations inflates the association between any one childhood risk factor and BPD in adulthood. For example, family functioning is inherently connected to both parental and proband psychiatric diagnoses (Johnson, Cohen, Kasen, Smailes, & Brook, 2001). Without testing a multivariate model of risk, it is unclear which set of risk factors are uniquely associated with BPD in adulthood. Additionally, only a small percentage of youth with any one of these risk factors will go on to develop BPD in adulthood. According to developmental theory, the presence of risk factors across child and family levels is necessary for the emergence of BPD (Bateman & Fonagy, 2003; Kernberg, 1984; Linehan, 1993). Maladaptive family functioning, parental psychiatric diagnoses, or early-onset psychiatric diagnoses are neither necessary nor sufficient and a multivariate approach is needed to create a more robust model of prospective risk.

Parental Psychiatric Disorders

Parental psychiatric disorder confers genetic, environmental, and genetic X environmental risk for psychiatric disorder to offspring. Available information on parental psychiatric histories of individuals with BPD is limited as most studies have examined psychiatric histories of all first-degree relatives. As parents play a greater role in shaping environmental influences than other first-degree relatives, parental psychiatric disorder is more likely to play a role in the transmission of disorder. Family members of individuals with BPD have higher rates of psychiatric disorders compared to the general population (White et al., 2003). Family studies assessing the rates of BPD diagnoses and related traits in first-degree relatives have found a 4- to 20-fold increase in prevalence or morbidity risk for BPD compared to the general population (Barnow, Grabe, Kessler, & Freyberger, 2006). Research also supports strong familial aggregation of core features of BPD, namely affective instability, interpersonal problems, cognitive impairments, and impulsivity (Gunderson, Zanarini, Choi-Kain, Mitchell, Jang, & Hudson, 2011; Silverman et al., 1991). Given the familial aggregation of these traits in BPD, it is not surprising that relatives of probands with BPD are also at increased risk for related psychiatric disorders, including Major Depressive Disorder (MDD), Substance Use Disorders (SUDs), and Antisocial Personality Disorder (ASPD) (1); (Riso, Klein, Anderson, & Ouimette, 2000).

Family Functioning

Maladaptive parenting has been found to mediate the association between parental psychiatric disorder and offspring psychiatric disorder (Johnson et al., 2001). Developmental models of BPD posit that invalidating parenting experiences transact with a child's genetic vulnerabilities to put them at risk (Bateman & Fonagy, 2003; Linehan, 1993).

Individuals with BPD have retrospectively described their parents as invalidating, emotionally over- as well as under-involved, and indifferent (Gunderson & Lyoo, 1997; Weaver & Clum, 1993). These individuals also describe relationships with caregivers and the ambience in their households as conflictual and inconsistent (Zanarini et al., 2000). In

the only prospective investigation of this topic, low parental warmth and harsh punishment during childhood was predictive of elevated risk for BPD in adulthood (Johnson, Cohen, Chen, Kasen, & Brook, 2006).

Adolescent Axis I Disorders

Early-onset Axis I psychiatric disorders increase risk for BPD in adulthood. In a longitudinal community sample that overlaps with the current study, a small subsample (n = 299) was assessed for personality disorder diagnoses at age 24. Those who onset to MDD, Anxiety Disorders, disruptive behavior disorders (Attention Deficit Hyperactivity Disorder [ADHD], Conduct Disorder [CD], Oppositional Defiant Disorder [ODD]), or SUDs by age 19 had increased risk for elevated BPD scores as adults (Lewinsohn et al., 1997; Rohde et al., 2001). In another prospective community sample, Kasen, Cohen, Skodol, Johnson, and Brook (1999) found that disruptive behavior disorders and MDD diagnosed by age 18 increased the odds four-and six-fold, respectively, for a cluster B personality disorder diagnosis by age 25. Fischer, Barkley, Smallish, and Fletcher (2002) found that children with ADHD (age at first assessment 4-12 years) had higher rates of BPD as young adults at follow-up compared to community controls. Similarly, Burke and Stepp (2012) found that childhood ADHD and ODD predicted BPD symptoms in a clinic-referred sample of young adult men. In a sample of adolescents in substance use treatment, adolescent-onset alcohol use disorders, ADHD, CD, Post-Traumatic Stress Disorder (PTSD), and MDD predicted BPD symptom severity after age 18 (Thatcher, Cornelius, & Clark, 2005). When studies have adjusted for effects of multivariate risk factors, several of these univariate associations between early-onset psychiatric disorders and BPD in adulthood are diminished. Specifically, after controlling for family functioning and sociodemographic characteristics, the presence of MDD by age 18 did not increase the risk for BPD by age 25 (Kasen et al., 2001). After adjusting for sociodemographic variables and other childhood Axis I disorders, only early-onset Anxiety Disorders, but not MDD or disruptive behavior disorders, elevated the risk for BPD by age 24 (Lewinsohn et al., 1997).

Based on previous findings and developmental theory, we expected univariate associations between maladaptive family functioning variables, parental psychiatric diagnoses, and proband adolescent onset psychiatric diagnoses and BPD symptoms in adulthood. Specifically, we expected to find lower family cohesion, less maternal and paternal support, and higher maternal-child discord predictive of symptoms. In terms of parental psychopathology, we expected to find maternal and paternal depression, anxiety, SUDs, BPD, and ASPD predictive of proband BPD symptoms in adulthood. Finally, we expected proband depression, anxiety, SUDs, CD/ODD, ADHD, and suicidality to predict BPD symptoms in adulthood. However, we expected these associations to be mitigated when examining a multivariate model of risk. The present study goes beyond our earlier study (Lewinsohn et al., 1997) by using a much larger sample, an additional assessment at age 30, and including parental psychopathology and contemporary adolescent reports of experience of parenting as prospective predictors of BPD symptoms.

Methods

Participants

Data come from the Oregon Adolescent Depression Project (OADP) (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993), a longitudinal study of a large cohort of high school students who were assessed twice during adolescence, a third time when the average age was 24, and a fourth time when the average age was 30. Participants were randomly selected from nine high schools in western Oregon. All study procedures were approved by the institutional review board at the Oregon Research Institute. All participants provided

informed consent. A total of 1,709 adolescents (ages 14-18; mean age 16.6, *SD* = 1.2) completed the initial (T_1) assessments between 1987 and 1989. The participation rate at T_1 was 61%. Approximately one year later, 1,507 of the adolescents (88%) returned for a second evaluation (T_2). Differences between the sample and the larger population from which it was selected, and between participants and those who declined to participate or dropped out of the study before T_2, were small (Lewinsohn et al., 1993). However, individuals with a history of disruptive behavior disorder at T_1 were more likely to drop-out of the study (16.8% vs. 6.0%, $\chi^2[1, N=1,709] = 31.22$, $p < .001$).

For the third assessment, all adolescents with a history of a psychiatric illness ($n = 644$) and a random sample of adolescents with no history of psychopathology by T_2 ($n = 457$) were invited to participate in a third (T_3) evaluation. All non-white T_2 participants were retained in the T_3 sample to maximize ethnic diversity. Of the 1,101 T_2 participants selected for T_3 interviews, 941 (85%) completed the evaluation. The T_2 diagnostic groups did not differ on the rate of participation at T_3. At age 30, all T_3 participants were asked to complete another interview assessment. Of the 941 who participated in the T_3 assessment, 816 (87%) completed the T_4 assessment. Differences between those who participated in T_3, but not T_4, and those who participated in T_3 and T_4 were small (Olino, Klein, Lewinsohn, Rohde, & Seeley, 2008). For the overall sample at T1, 52.1% (891 of 1709) were female. For the subsample included in the present report, 58.8% (480 of 816) were female. At T2, the average age was 17.71 (SD = 1.24) for the full sample and 17.23 (SD = 1.27) for the subsample reported on here. At T3, the average age was 24.50 (SD = .73) for the full sample and 24.46 (SD = .70) for the subsample reported on here.

We assessed lifetime psychopathology in the biological parents of the OADP participants near the time of the T_3 evaluation. Family history data were also obtained from the OADP participants, and when a parent could not be directly interviewed, informant data were obtained from a second informant. Of the 816 probands who completed the T4 assessment, diagnostic information was available for 701 mothers and 691 fathers.

Measures

Diagnostic measures—At T_1 and T_2, participants were interviewed with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children [K-SADS; (Orvaschel, Puig-Antich, Chamebrs, Tabrizi, & Johnson, 1982)] that combined features of the Epidemiologic and Present Episode versions, and included additional items to derive *Diagnostic and Statistical Manual of Mental Disorders,* 3rd edition revised [DSM-III-R; (American Psychiatric Association, 1987)] diagnoses. Assessments at T_2 and T_3 also included the Longitudinal Interval Follow-Up Evaluation [LIFE; (Keller et al., 1987)]. The K-SADS/LIFE procedure provided information regarding the onset and course of disorders since the previous interview. The T_4 interview consisted of a joint administration of the LIFE and the Structured Clinical Interview for DSM–IV [SCID; (First et al., 1996)] to probe for new or continuing episodes since T_3. Diagnoses were based on DSM-III-R criteria for T_1 and T_2 and *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) (American Psychiatric Association, 1994) criteria for T_3 and T_4. Based on audiotaped interviews (*n*s ranged from 124-233 across T_1-T_4), the interrater reliability (indexed by kappa) for depressive disorders (MDD or dysthymia) was .82 at T_1 and 1.00 at T_2. The interrater reliability for anxiety disorders was .76 at T_1 and .80 at T_2. The interrater reliability for SUD was .82 at T_1 and .96 at T_2. Due to low base rates of CD, ODD, and ADHD, interrater reliability could only be formally assessed at T_1. The interrater reliability for CD was .71, for ODD was .77, and for ADHD was .89.

At T_4, BPD symptoms were assessed using the International Personality Disorder Examination (IPDE) (Loranger et al., 1994). The IPDE has documented interrater reliability,

temporal stability, and reflects minimal state effects (Loranger et al., 1991). The IPDE has an extensive scoring manual, which defines the scope and meaning of each item. Items were classified as to how they reflected specific DSM BPD criteria and the total number of criteria met through the T_4 assessment was used as the dependent measure. Based on audiotaped interviews, the interrater reliability (indexed by intra-class correlations) for BPD count scores was .86 at T_4.

Biological parents of the original OADP participants were interviewed using the SCID, non-patient version [SCID-NP; (First, Spitzer, Gibbons, & Williams, 1996)]. All interviews were conducted without knowledge of the offspring's diagnoses. Independent raters reviewed audiotapes of 184 randomly selected interviews. The interrater reliability of lifetime diagnoses were excellent for depressive disorders (MDD or dysthymia; K = .94), any anxiety disorder (K = .90), alcohol abuse/dependence (K = .86), and drug abuse/dependence (K = .89). Parents were also interviewed using the Structured Clinical Interview for Axis II Personality Disorders [(SCID-II; (First et al., 1996)] to assess BPD and ASPD. Due to low base-rates of BPD and ASPD, inter-rater reliability was assessed on symptom counts and was high for both disorders, based on a sample of 100 parental interviews (both ICCs = .94).

Family history data were collected from the original OADP participants and at least one other family member using a modified version of the Family Informant Schedule and Criteria [FISC; (Mannuzza & Fyer, 1990)], supplemented with all items necessary to derive DSM-IV diagnoses. Independent raters reviewed audiotapes of 242 randomly selected informant interviews. The interrater reliability of lifetime diagnoses were good-excellent for depressive disorders (MDD or dysthymia; K = .90), any anxiety disorder (K = .77), alcohol abuse/dependence (K = .90), and drug abuse/dependence (K = .82).

Lifetime best-estimate DSM-IV diagnoses were derived from all available information (Leckman, Sholomskas, Thompson, Belanger, & Weissman, 1982). Two diagnosticians, from a team of four senior clinicians, independently derived best-estimate diagnoses without knowledge of offspring diagnoses. Disagreements were resolved by consensus. Interrater reliability of the independently derived best-estimate diagnoses prior to the resolution of discrepancies was excellent for depressive disorders (MDD or dysthymia; K = .91), any anxiety disorder (K = .94), alcohol abuse/dependence (K = .97), and drug abuse/dependence (K = .96).

Participant interviews at T_3 and T_4 and interviews with their parents were conducted by telephone, which generally yields comparable results to face-to-face interviews (Rhode, Lewinsohn, & Seeley, 1997). Most interviewers had advanced degrees in a mental health field and several years of clinical experience.

Self-report measures—During the T_2 assessment, probands completed a subset of items assessing enjoyable and aversive interactions with family members using items from the Appraisal of Parents subscale of the Conflict Behavior Questionnaire [11 items; (Prinz, Foster, Kent, & O'Leary, 1979)], and the Parent Attitude Research Instrument [6 items; (Schaefer, 1965)]. These items indexed adolescent experience of maternal support (α = .76), paternal support (α = .77), and maternal-child discord (three items that reflect angry affect between mother and child and two items reflect maternal behavior inducing guilt in the child) (α = .68). Youth also completed the Cohesion subscale of the Family Environment Scale (5 items; (Moos, 1974); α = .81). Maternal support, paternal support, and maternal-child discord are scored such that higher scores reflect more dysfunctional levels. Family cohesion is scored such that lower levels reflect more dysfunctional levels.

Data analysis—All analyses were conducted using Mplus 6.1 (Muthen & Muthen, 1998-2010). As proband BPD symptoms were computed as a count variable, Poisson regression models were estimated. All analyses were weighted by the probability of being included in the T_3 assessment. We examined unadjusted associations of proband sex, proband reported measures of family functioning, proband psychopathology through T_2, and maternal and paternal history of psychopathology with BPD symptoms at age 30. As parental education, as a proxy for SES, was associated with BPD symptoms, we adjusted these basic models for parental education. Next, we examined multivariate models to investigate the influence of each domain of predictors. This produced three multivariate models: family functioning reported at T_2; parental psychopathology; and proband lifetime psychopathology through T_2. We then examined a final model that included all variables from each of the three domains.

Results

The observed mean for the number of BPD symptoms endorsed was .35 (*SE* = .03). Observed scores ranged from 0 to 8 with most participants endorsing no symptoms (75.4%). A modest portion of the sample endorsed one symptom (18.8%), a small portion of the sample endorsed two symptoms (3.7%), and a small portion endorsed three or more symptoms (2.2%). Using the conventional criteria of five or more symptoms, 5 (0.6%) probands met criteria for BPD. When weighting for T_3 participation, the mean number of symptoms was .29 (*SE* = .08). We assessed whether BPD symptoms measured a unitary construct by fitting a one-factor confirmatory factor analysis model. The nine BPD symptoms were entered as observed, binary indicators and we found that the model fit the data very well using the WLSMV estimator ($\chi^2[27] = 31.66$, $p = .24$, CFI = .98, RMSEA = .01 [.00-.03]). Table 1 displays the unweighted and weighted prevalence rates of adolescent and parental psychopathology.

Poisson regression models examined bivariate associations between our predictor variables and proband BPD symptoms at age 30 (Table 2). We did not find a significant gender difference in BPD symptoms. Family cohesion, maternal support, and maternal-child discord at T_2 were associated with later BPD symptoms, such that higher levels of dysfunction were associated with higher levels of BPD symptoms. All forms of maternal psychopathology and paternal anxiety, SUD, and ASPD were associated with higher levels of proband BPD symptoms. All forms of proband psychopathology through T_2 were associated with higher levels of BPD symptoms.

The first multivariate model (Table 2) included proband sex, parental education, and all adolescent reports of family functioning. This model did not identify any family functioning measures that uniquely predicted proband BPD symptoms. The second multivariate model included proband sex, parental education, and maternal and paternal psychopathology. As only a very small number of mothers had a lifetime ASPD diagnosis, this was not included in analyses. This model found that maternal SUD and BPD uniquely predicted proband BPD symptoms. However, maternal depression and anxiety and paternal anxiety, SUD, and ASPD were no longer associated with proband BPD symptoms. The third multivariate model included proband sex, parental education, and proband psychopathology through T_2. This model found that proband depression, SUD, CD/ODD, and suicidality were associated with later BPD symptoms. However, proband ADHD was no longer associated with proband BPD symptoms. Our final model included all indicators of family functioning, parental psychopathology, and early proband psychopathology. This final model found that maternal-child discord, maternal BPD, paternal SUD, and proband depression, SUD, and suicidality continued to predict BPD symptoms at age 30. We examined whether sex moderated the relationship between adolescent psychopathology at T1 and later BPD

symptomatology beyond the influence of the full set of predictors in Model 5 (Table 2). However, no interaction effects were significantly associated with BPD symptomatology. Finally, to determine the impact of MDD on our findings, we re-ran the final model, including T_3/T_4 MDD as a covariate. All the conclusions remain the same. However, the magnitude of the maternal BPD to offspring BPD symptoms association is reduced to a trend ($B = .68$, $SE = .37$, $p = .069$).

Discussion

As expected, there were many significant univariate associations between each set of early risk factors and later BPD symptoms during adulthood, which provides some support for many of the prominent theories of BPD (Bateman & Fonagy, 2003; Kernberg, 1984; Linehan, 1993). Consistent with previous empirical work, maladaptive family functioning, including low family cohesion, low maternal support, and high levels of maternal-child discord, were associated with later BPD symptoms (Fruzzetti et al., 2005; Johnson et al., 2006). Consistent with previous family and family history studies of probands with BPD, we found that maternal and paternal Anxiety disorders, SUD, and ASPD predicted proband BPD symptoms in adulthood (White et al., 2003). Additionally, maternal history of MDD and BPD predicted proband BPD symptoms. Proband early-onset psychiatric diagnoses, including Depression, Anxiety, SUD, CD/ODD, ADHD, and suicidality were significantly related to BPD symptoms in adulthood. This is also similar to previous findings from longitudinal studies of community and psychiatric youth (Fischer et al., 2002; Kasen et al., 1999; Lewinsohn et al., 1997; Rhode, Lewinsohn, Kahler, Seeley, & Brown, 2001; Thatcher et al., 2005).

In the multivariate analysis, many of the relationships between early risk factors and BPD symptoms in adulthood were no longer significant. These findings highlight the interrelationship among many early risk factors commonly examined in the prediction of BPD. To reduce this methodological confound and understand the unique effects of these early risk factors on the development of BPD, it is important to assess the family environment. In the final model, maternal-child discord, maternal history of BPD, paternal history of SUD, and proband early-onset depression, SUD, and suicidality uniquely predicted in the prediction of BPD symptoms in adulthood. Each of these qualitatively distinct variables demonstrated direct and unique contributions to the development of BPD over and above all other variables included in the model.

Finding that maternal-child discord predicted BPD symptoms in adulthood suggests that children who are at risk for BPD may be sensitive to maternal communication and forms of control (Fruzzetti et al., 2005). Maternal psychological control has been linked to psychopathology in children (El-Sheikh, Hinnant, Kelly, & Erath, 2010) and may be proxy for a larger context of ‘invalidation’; that the child experiences, consistent with Linehan's (1993) biosocial theory. However, this is the first study to demonstrate such a prospective effect for BPD in adults. The unique association between maternal BPD and proband BPD symptoms documents the familiality of this disorder. Additionally, the association of paternal history of SUD with proband BPD symptoms is consistent with the notion that impulsivity may aggregate individually in family members of those with BPD (Silverman et al., 1991). The progression of early-onset Depression and SUD to later BPD for some youth suggests that a common set of traits, perhaps emotion dysregulation and impulsivity, may give rise to these disorders in childhood and BPD in adulthood, consistent with recent adaptions of the biosocial theory of BPD (Crowell, Beauchaine, & Linehan, 2009). Additionally, early-onset psychiatric diagnoses, including Depression and SUD put children at risk for interpersonal and academic impairments that persist even into young adulthood (Flory, Lynam, Milich, Leukefeld, & Clayton, 2004; Lewinsohn, Rohde, Seeley, Klein, &

Gotlib, 2003), increasing the likelihood of these functional impairments becoming more entrenched, resulting in the emergence of personality disorder in adulthood. The clusters of predictors chosen for this study (i.e., family functioning, parental psychopathology, and proband psychopathology) are proxies for more complex developmental processes. Our findings suggest that parenting and the broader family environment are important in the development of BPD in adulthood. Consistent with our finding regarding the negative relationship between parental education and BPD symptoms, low socioeconomic status has been demonstrated to put children at risk for BPD (Johnson, Cohen, Dohrenwend, Link, & Brook, 1999).

This study was not without limitations. We did not assess for proband personality pathology prior to adulthood so the impact of the associations between early-onset psychiatric diagnoses and adult BPD symptoms may be attenuated when early personality disorder symptoms are taken into account. Similarly, early personality disorder diagnosis and/or traits may predict later Axis I disorders or transactional relationships may exist between early-onset Axis I disorders and early-onset BPD. This study emphasizes identifying unique influences, possibly at the expense of common risk factors. Due to low base rates, we were not able to examine BPD diagnosis but instead utilized symptom counts as the dependent variable. Therefore, the conclusions that can be drawn from this study are regarding the relationship between early predictors and symptoms, not the disorder. The use of symptom counts may limit the generalizability of these findings to patient samples. It is important to note that individuals with elevated BPD symptoms have been shown to have clinical and functional impairments comparable to those individuals with a formal BPD diagnosis (Clifton & Pilkonis, 2007). Additionally, individuals with a history of disruptive behavior disorder at T_1 were more likely to drop-out of the study, which may have influenced our finding that ODD/CD was not related to BPD symptoms. Lastly, the full sample was not followed-up. The complex sampling design sought to follow-up all participants with psychopathology through T2, which may have over sampled for those at risk for later BPD pathology. However, we incorporated the sampling design into the analyses using a weighting procedure that appropriately adjusted analyses to provide generalizable estimated associations between risk factors and symptoms.

The strengths of the study included the use of clinical interviews to assess Axis I and II disorders in both the probands and parents when possible, the longitudinal design, and the use of community participants. This is the first longitudinal study to report the prospective associations between both mothers' and fathers' histories of psychiatric diagnoses and offspring BPD symptoms in adulthood. The use of community participants rather than patients ensures that the prospective associations observed are more representative of the development of BPD that unfolds in the general population.

The findings from this study have important implications for research and clinical practice. Multivariate assessment models are needed to determine unique associations between early life experiences and BPD in adulthood. Future work should employ these models and test interactions to better elucidate the transactions that may occur between child- and family-level characteristics in predicting the development of BPD. Identifying unique risk factors helps focus screening efforts. Our study identified maternal-child discord, maternal BPD, paternal SUD, and proband depression, SUD, and suicidality as specific risk factors for BPD symptoms in adulthood. Screening for a smaller set of risk factors, such as the ones we have identified, during routine evaluations, may results in an efficient early detection of risk. These findings may ultimately inform prevention efforts, such as targeting maternal-child discord, to prevent BPD.

Acknowledgments

This work was supported by National Institute of Mental Health Grants R01 MH40501, R01 MH50522, R01 MH52858, and National Institute of Drug Abuse Grant R01 DA012951 (Dr. Lewinsohn). Dr. Stepp's effort was supported by K01 MH086713 and Dr. Olino's effort was supported by K01 MH092603. The content is solely the responsibility of the authors and does not represent views of the National Institutes of Health.

References

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 3rd. Washington, DC: Author; 1987.

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th. Washington, DC: Author; 1994.

Barnow SS, Grabe HJ, Kessler C, Freyberger HJ. Individual characteristics, familial experience, and psychopathology in children of mothers with borderline personality disorder. Journal of the American Academy of Child & Adolescent Psychiatry. 2006; 45:965–72. [PubMed: 16865039]

Bateman AW, Fonagy P. The development of an attachment-based treatment program for borderline personality disorder. Bulletin of the Menninger Clinic. 2003; 67(3):187–211. [PubMed: 14621062]

Burke JD, Stepp SD. Adolescent disruptive behavior and borderline personality disorder symptoms in young adult men. Journal of Abnormal Child Psychology. 2012; 40:35–44. [PubMed: 21853377]

Clifton AP, Pilkonis PA. Evidence for a single latent class of Diagnostic and Statistical Manual of Mental Disorders borderline personality pathology. Comprehensive Psychiatry. 2007; 48:70–8. [PubMed: 17145285]

Cohen P, Crawford TN, Johnson JG, Kasen S. The Children in the Community Study of developmental course of personality disorder. Journal of Personality Disorders. 2005; 19:466–86. [PubMed: 16274277]

Crowell SE, Beauchaine TP, Linehan MM. A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. Psychological Bulletin. 2009; 135:495–510. [PubMed: 19379027]

El-Sheikh M, Hinnant JB, Kelly RJ, Erath S. Maternal psychological control and child internalizing symptoms: vulnerability and protective factors across bioregulatory and ecological domains. Journal of Child Psychology and Psychiatry. 2010; 51:188–98. [PubMed: 19703095]

First, MB.; Spitzer, RL.; Gibbons, M.; Williams, JBW. The Structured Clinical Interview for DSM-IV Axis I Disorders-Non-patient edition. New York: Biometrics Research Department, New York State Psychiatric Institute; 1996.

Fischer M, Barkley RA, Smallish L, Fletcher K. Young adult follow-up of hyperactive children: Self-reported psychiatric disorders, comorbidity, and the role of childhood conduct problems and teen CD. Journal of Abnormal Child Psychology. 2002; 30:463–75. [PubMed: 12403150]

Flory K, Lynam D, Milich R, Leukefeld C, Clayton R. Early adolescent through young adult alcohol and marijuana use trajectories: Early predictors, young adult outcomes, and predictive utility. Development and Psychopathology. 2004; 16:193–213. [PubMed: 15115071]

Fruzzetti AE, Shenk C, Hoffman PD. Family interaction and the development of borderline personality disorder: A transactional model. Development and Psychopathology. 2005; 17:1007–30. [PubMed: 16613428]

Gunderson JG, Lyoo IK. Family problems and relationships for adults with borderline personality disorder. Harvard Review of Psychiatry. 1997; 4:272–8. [PubMed: 9385003]

Gunderson JG, Zanarini MC, Choi-Kain LW, Mitchell KS, Jang KL, Hudson JI. Family study of borderline personality disorder and its sectors of psychopathology. Archives of General Psychiatry. 2011; 68:753–762. [PubMed: 21727257]

Johnson JG, Cohen P, Chen H, Kasen S, Brook JS. Parenting behaviors associated with risk for offspring personality disorder during adulthood. Archives of General Psychiatry. 2006; 63:579–87. [PubMed: 16651515]

Johnson JG, Cohen P, Dohrenwend BP, Link BG, Brook JS. A longitudinal investigation of social causation and social selection processes involved in the association between socioeconomic status

and psychiatric disorders. Journal of Abnormal Psychology. 1999; 108:490–9. [PubMed: 10466273]

Johnson JG, Cohen P, Kasen S, Smailes EM, Brook JS. Association of maladaptive parental behavior with psychiatric disorder among parents and their offspring. Archives of General Psychiatry. 2001; 58:453–60. [PubMed: 11343524]

Kasen S, Cohen P, Skodol AE, Johnson JG, Brook JS. Influence of child and adolescent psychiatric disorders on young adult personality disorder. American Journal of Psychiatry. 1999; 156:1529–35. [PubMed: 10518162]

Kasen S, Cohen P, Skodol AE, Johnson JG, Smailes E, Brook JS. Childhood depression and adult personality disorder. Archives of General Psychiatry. 2001; 58:231–6. [PubMed: 11231829]

Keller MB, Lavori PW, Friedman B, Nielsen E, Endicott J, McDonald-Scott P, Andreasen NC. The Longitudinal Interval Follow-up Evaluation. A comprehensive method for assessing outcome in prosepective longitudinal studies. Archives of General Psychiatry. 1987; 44:540–8. [PubMed: 3579500]

Kernberg, O. Severe personality disorders: Psychotherapeutic strategies. New Haven, CT: Yale University Press; 1984.

Leckman JF, Sholomskas D, Thompson WD, Belanger A, Weissman MM. Best Estimate of Lifetime Psychiatric-Diagnosis-a Methodological Study. Archives of General Psychiatry. 1982; 39:879–83. [PubMed: 7103676]

Lewinsohn PM, Hops H, Roberts RE, Seeley JR, Andrews JA. Adolescent psychopathology: I. Prevalence and incidence of depression and other DSM-III--R disorders in high school students. Journal of Abnormal Psychology. 1993; 102:133–44. [PubMed: 8436689]

Lewinsohn PM, Rohde P, Seeley JR, Klein DN. Axis II psychopathology as a function of Axis I disorders in childhood and adolescence. Journal of the American Academy of Child & Adolescent Psychiatry. 1997; 36:1752–9. [PubMed: 9401337]

Lewinsohn PM, Rohde P, Seeley JR, Klein DN, Gotlib IH. Psychosocial functioning of young adults who have experienced and recovered from major depressive disorder during adolescence. Journal of Abnormal Psychology. 2003; 112:353–63. [PubMed: 12943014]

Linehan, MM. Cognitive Behavioral Treatment of Borderline Personality Disorder. New York: Guilford Press; 1993.

Loranger AW, Lenzenweger MF, Gartner AF, Susman VL, Herzig J, Zammit GK, Gartner JD, Abrams RC, Young RC. Trait-state artifacts and the diagnosis of personality disorders. Archives of General Psychiatry. 1991; 48:720–8. [PubMed: 1883255]

Loranger AW, Sartorius N, Andreoli A, Berger P, Buchheim P, Channabasavanna SM, Coid B, Dahl A, Diekstra RFW, Ferguson B, Jacobsberg LB, Mombour W, Pull C, Ono Y, Regier DA. The International Personality Disorder Examination: The World Health Organization/Alchohol, Drug Abuse, and Mental Health Administration international pilot study of personality disorders. Archives of General Psychiatry. 1994; 51:215–24. [PubMed: 8122958]

Mannuzza, S.; Fyer, AJ. Family Informant Schedule and Criteria (FISC), July 1990 Revision. New York: Anxiety Disorders Clinic, New York State Psychiatric Institute; 1990.

Moos, RH. Family Environment Scale and Preliminary Manual. Palo Alto, CA: Consulting Psychologist Press; 1974.

Muthen, LK.; Muthen, BO. Mplus User's Guide. Sixth. Los Angeles, CA: Author; 1998-2010.

Olino TM, Klein DN, Lewinsohn PM, Rohde P, Seeley JR. Longitudinal associations between depressive and anxiety disorders: A comparison of two trait models. Psychological Medicine. 2008; 38:353–63. [PubMed: 17803836]

Orvaschel H, Puig-Antich J, Chambers WJ, Tabrizi MA, Johnson R. Retrospective assessment of prepubertal major depression with the Kiddie-SADS-E. Journal of the American Academy of Child & Adolescent Psychiatry. 1982; 21:392–7.

Prinz RJ, Foster S, Kent RN, O'Leary KD. Multivariate assessment of conflict in distressed and nondistressed mother-adolescent dyads. Journal of Applied Behavior Analysis. 1979; 12:691–700. [PubMed: 541311]

Riso LP, Klein DN, Anderson RL, Ouimette PC. A family study of outpatients with borderline personality disorder and no history of mood disorder. Journal of Personality Disorders. 2000; 14:208–17. [PubMed: 11019745]

Rohde P, Lewinsohn PM, Kahler CW, Seeley JR, Brown RA. Natural course of alcohol use disorders from adolescence to young adulthood. Journal of the American Academy of Child & Adolescent Psychiatry. 2001; 40:83–90. [PubMed: 11195569]

Rohde P, Lewinsohn PM, Seeley JR. Comparability of telephone and face-to-face interviews in assessing axis I and II disorders. American Journal of Psychiatry. 1997; 154:1593–8. [PubMed: 9356570]

Schaefer ES. Children's report of parental behavior: An inventory. Child Development. 1965; 36:413–24. [PubMed: 14300862]

Silverman JM, Pinkham L, Horvath TB, Coccaro EF, Klar H, Schear S, Apter S, Davidson M, Mohs RC, Siever LJ. Affective and impulsive personality disorder traits in the relatives of patients with borderline personality disorder. American Journal of Psychiatry. 1991; 148:1378–85. [PubMed: 1897620]

Thatcher DL, Cornelius JR, Clark DB. Adolescent alcohol use disorders predict adult borderline personality. Addictive Behaviors. 2005; 30:1709–24. [PubMed: 16095845]

Weaver TL, Clum GA. Early family environments and traumatic experiences associated with borderline personality disorder. Journal of Consulting and Clinical Psychology. 1993; 61:1068–75. [PubMed: 8113485]

White CN, Gunderson JG, Zanarini MC, Hudson JI. Family studies of borderline personality disorder: A review. Harvard Review of Psychiatry. 2003; 11:8–19. [PubMed: 12866737]

Zanarini MC, Frankenburg FR, Reich DB, Marino MF, Lewis RE, Williams AA, Khera GS. Biparental failure in the childhood experiences of borderline patients. Journal of Personality Disorders. 2000; 14:264–73. [PubMed: 11019749]

Zanarini MC, Frankenburg FR, Ridolfi ME, Jager-Hyman S, Hennen J, Gunderson JG. Reported childhood onset of self-mutilation among borderline patients. Journal of Personality Disorders. 2006; 20:9–15. [PubMed: 16563075]

Zanarini MC, Williams AA, Lewis RE, Reich RB, Vera SC, Marino MF, Levin A, Yong L, Frankenburg FR. Reported pathological childhood experiences associated with the development of borderline personality disorder. American Journal of Psychiatry. 1997; 154:1101–6. [PubMed: 9247396]

Table 1

Weighted and Unweighted Rates of Psychopathology in Probands, Mothers, and Fathers

	Proband		Maternal		Paternal	
	Unweighted % (n)	Weighted %	Unweighted % (n)	Weighted %	Unweighted % (n)	Weighted %
Depression	34.8 (284)	24.9	37.1 (260)	35.3	23.2 (160)	21.3
Anxiety	13.8 (113)	10.2	15.8 (111)	15.5	7.7 (53)	6.7
SUD	13.8 (113)	10.0	20.8 (146)	18.6	45.4 (314)	44.0
CD/ODD	5.9 (48)	4.3	--		--	
ADHD	3.2 (26)	2.3	--		--	
BPD	--		1.3 (9)	1.1	3.8 (26)	3.7
ASPD	--		0.4 (3)	0.3	8.1 (56)	7.3

SUD = Substance Use Disorder; CD/ODD = Conduct Disorder/Oppositional Defiant Disorder; ADHD = Attention Deficit/Hyperactivity Disorder; BPD = Borderline Personality Disorder; ASPD = Antisocial Personality Disorder. Proband diagnoses are lifetime through the T_2 assessment. Maternal and Paternal diagnoses are lifetime through the family history assessment at T_3. Due to low base rates of maternal ASPD, maternal ASPD is not considered in predictor models.

Table 2

Univariate and Multivariate Models Predicting Offspring BPD Symptoms at Age 30

	Model 1 B (SE)	Model 2 B (SE)	Model 3 B (SE)	Model 4 B (SE)	Model 5 B (SE)
Parental Education	-.35 (.16)*	-.27 (.16)	-.21 (.18)	-.16 (.15)	.03 (.18)
Male	.08 (.15)	.18 (.16)	.19 (.19)	.13 (.15)	.22 (.18)
Proband Report of Family Functioning at T_2					
Family Cohesion	-.13 (.04)***	-.04 (.05)			-.05 (.05)
Maternal Support	.06 (.02)***	.03 (.03)			-.03 (.02)
Paternal Support	.03 (.02)	.02 (.02)			-.02 (.02)
Maternal-Child Discord	.08 (.02)***	.05 (.03)			.06 (.03)*
Maternal Psychopathology					
Depression	.54 (.17) ***		.29 (.19)		.20 (.19)
Anxiety	.50 (.21)*		.06 (.24)		.15 (.22)
SUD	.74 (.19) ***		.42 (.18)*		.20 (.19)
BPD	1.09 (.36) ***		1.23 (.04)*		.95 (.37)*
ASPD	1.81 (.82)*		--		---
Paternal Psychopathology					
Depression	.28 (.22)		.01 (.22)		-.14 (.22)
Anxiety	.69 (.28)*		.33 (.33)		.46 (.33)
SUD	.65 (.17) ***		.34 (.20)		.43 (.17)*
BPD	.42 (.31)		.16 (.36)		-.33 (.41)
ASPD	.76 (.22)***		.30 (.27)		.48 (.34)
Proband T_2Psychopathology					
Depression	.81 (.15) ***			.39 (.16)*	.36 (.18)*
Anxiety	.67 (.20) ***			.28 (.19)	-.06 (.25)
SUD	1.36 (.16) ***			1.02 (.18)***	1.11 (.20)***
CD/ODD	1.4 (.23) ***			.54 (.26)**	.14 (.28)

Personal Disord. Author manuscript; available in PMC 2014 July 01.

	Model 1	Model 2	Model 3	Model 4	Model 5
	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)
ADHD	1.21 (.38) ***			.55 (.35)	.41 (.57)
Suicidality	.14 (.04) ***			.07 (.03) *	.09 (.04) *

* $p < .05$;

** $p < .01$;

*** $p < .001$. BPD = Borderline Personality Disorder; SUD = Substance Use Disorder; CD/ODD = Conduct Disorder and/or Oppositional Defiant Disorder; ADHD = Attention Deficit-Hyperactivity Disorder; ASPD = Antisocial Personality Disorder. Parental education is operationalized as having at least one parent who completed a BS/BA program. Models are estimated using Poisson regression methods.