Developmental Trajectories of Borderline Personality Disorder Symptoms and Psychosocial Functioning in Adolescence

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Abstract

Major gains toward understanding the emergence of borderline personality disorder (BPD) pathology, which is typically first noted during adolescence, have been made. The present study addresses a gap in our understanding of within-person change in BPD symptoms across adolescence and contributes to the limited literature on outcomes associated with adolescent BPD. Using an at-risk community sample of girls (N=2,450), bivariate latent growth curve models were used to analyze the co-development of BPD symptoms with eight domains of psychosocial functioning (e.g., social skills, sexual behavior) across ages 14-17. Findings revealed moderate to strong effect sizes for the associations between BPD symptoms and every domain of psychosocial functioning, suggesting that the development of BPD was coupled with poorer outcomes across development. These results highlight the increased need for extending advancements in the adult PD literature to research on PDs in adolescence, and for greater recognition of adolescent BPD in clinical settings.

Keywords: Borderline Personality Disorder; Psychosocial Functioning; Adolescence; Growth Curve Modeling; Developmental Trajectories
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Diagnosing personality disorders (PDs) in adolescents remains controversial, as many practitioners believe that personality, not fully developed, cannot yet be deemed aberrant (Hutsebaut, Feenstra, & Luyten, 2013). However, the clinical need to intervene is becoming undeniable (for a recent review, see Courtney-Seidler, Klein, & Miller, 2013), particularly when adolescents exhibit features of borderline personality disorder (BPD). On average, treatment seeking for BPD begins during late adolescence (Zanarini et al., 2006) and, similar to adults, adolescents with BPD are overrepresented in clinical settings. Specifically, adolescents with BPD constitute approximately 30% of clinical samples (Glenn & Klonsky, 2013; Miller, Muehlenkamp, & Jacobson, 2008), and experience high levels of distress and co-morbidity with other disorders (Ha, Balderas, Zanarini, Oldham, & Sharp, 2014). Adolescents with BPD often engage in deliberate self-harm (Jacobson, Muehlenkamp, Miller, & Turner, 2008), which constitutes a serious public health concern as suicide is the third leading cause of death in 15-19 year olds (Crowell & Yaptangco, 2013; Youth Suicide Prevention, CDC). However, as noted by others (Stepp, Pilkonis, Hipwell, Loeber, & Stouthamer-Loeber, 2010), surprisingly little is known about adolescents who are diagnosed with BPD given the manifest clinical severity and the potential dire outcomes. The present study examined a relatively unknown aspect of adolescent BPD—its relation to psychosocial functioning, such as academic, social, and health-related behaviors. Specifically, we tested whether developmental trajectories of BPD symptoms and impairments in psychosocial functioning track together during adolescence.

BPD debut: Targeting adolescence

By enlisting prospective study designs, the development and psychopathology literature
has made critical advances examining the etiological mechanisms by which BPD develops (Courtney-Seidler et al., 2013; Crowell, Beauchaine, & Linehan, 2009). Transactions between biological vulnerabilities (Crowell et al., 2012) and environmental risk-factors (Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009) are predictive of BPD symptoms, such as affective instability, impulsivity, and non-suicidal self injury. Both theory and empirical research point to adolescence as a key developmental period within which to study the onset of BPD. While these advancements have contributed much toward our understanding of how BPD develops, there are few data to speak to the functional impact of BPD symptoms in adolescence. By diagnostic definition, BPD disturbs one’s sense of identity and disrupts interpersonal functioning. Processes of identity formation and understanding self in relation to peer and romantic partners have long been considered key developmental foci during the adolescent period (Hill et al., 2013; Kerpelman et al., 2012). Thus, challenges within these developmental tasks or the expression of BPD symptoms during adolescence are ripe for transactional processes that perpetuate both pathology and functional impairment.

**Individual variability in personality pathology**

Prior to reviewing what is known about the links between BPD and psychosocial functioning during adolescence, it is important to review what is known about the variability of BPD symptoms over time. In general, a large body of research now shows that personality disorders are not as stable as previously assumed, highlighting that the severity of pathology varies within afflicted individuals in adulthood (see Morey & Hopwood, 2013 for a review). Moreover, there are individual differences in trajectories of personality pathology, such that individuals vary in both the direction (i.e., some decrease and others increase) and rate of change over time (Lenzenweger, Johnson, & Willett, 2004). More specifically, these general patterns are observed in BPD, highlighting that the severity of BPD naturally vacillates within individuals
in adulthood (Choi-Kain, Zanarini, Frankenburg, Fitzmaurice, & Reich, 2010; Wright, Hopwood, & Zanarini, in press; Wright, Pincus, & Lenzenweger, 2010).

Taking a dynamic rather than static view of personality pathology during adulthood helped shift the expectation that rates would be stable during adolescence. In studies that have assessed BPD with dimensional assessments, adolescent BPD actually demonstrates moderate rank-order stability across time (Crick, Murray-Close, & Woods, 2005; de Clercq, van Leeuwen, van den Noortgate, de Bolle, & de Fruyt, 2009), yet individuals differ in trajectories of change over time (de Clercq et al., 2009). This suggests that adolescent and adult BPD may share similar dynamic characteristics, in that there will be significant between-person variability in the within-person trajectories of BPD symptoms over time.

**Personality pathology and psychosocial functioning**

Personality pathology is related to a host of poor psychosocial outcomes, including aspects of social functioning, occupational functioning, and health related outcomes (Smith & Benjamin, 2002). By definition, individuals with BPD are likely to struggle with complex social interactions, which require them to repeatedly modulate their emotional arousal to communicate more effectively and relate to another person. In some ways, the distinction between what is a feature, versus a consequence, of BPD is unclear as some symptoms of BPD are in fact defined by the individual’s endorsement of being challenged in various social relations (Ro & Clark, 2013). Although we recognize this circularity, it must be also noted that personality pathology and psychosocial functional impairment may not have a 1:1 correspondence. In other words, we cannot assume that improvement in BPD equates to improvement in psychosocial functioning or even vice versa. In fact, findings from a 2-year study demonstrated that social functioning impairment remained, even after PD pathology remitted (Skodol et al., 2005). Subsequently, the same pattern of remitting BPD symptomatology in the face of enduring impairments was found
over the course of 10 (Gunderson et al., 2011) and 16 years (Zanarini et al., 2012). These findings suggest that at least for some individuals, the psychosocial consequences of BPD may be enduring, which likely confirms clinical observations that even when crises cease, core aspects of personality pathology persist. Furthermore, the presence of enduring psychosocial impairment may exacerbate BPD symptoms. We now turn to reviewing the limited research available on the relationships between adolescent BPD symptoms and psychosocial functioning.

**BPD and psychosocial functioning**

Only a small number of studies have reported on psychosocial functioning subsequent to adolescent BPD. Long-term follow-up data obtained from the Children in the Community cohort study found that greater symptoms of BPD at age 14 predicted poorer academic and employment status, less partner involvement, and greater likelihood of needing services 20 years later (Winograd, Cohen, & Chen, 2008). Evidence of poor functioning was also found when using a diagnostic approach. Chanen, Jovev, and Jackson (2007) found that adolescents with BPD concurrently had worse relations with peers, family, and had overall poorer self-care compared to adolescents with another PD or those with no personality pathology (Chanen et al., 2007).

When considering the potential co-development of BPD symptoms with domains of psychosocial functioning, it is helpful to consider how psychosocial functioning relates to mental health concerns, more broadly defined, during adolescence. Prevention and intervention models have frequently demonstrated that poor social, school, and health functioning are detrimental to mental health during adolescence. For instance, less school connectedness has been prospectively associated with depression and anxiety (Bond et al., 2007), and participation in certain extracurricular activities predicts lower rates of drinking and drug use (Eccles, Barber, Stone, & Hunt, 2003). Engaging in sexual and romantic relations during early adolescence predicts greater depression (Starr, Davila, et al., 2012) with one study demonstrating that sexual
activity and mental health symptoms were bi-directionally associated in African-American adolescent girls (Starr, Donenberg, & Emerson, 2012). When further considering the transactional relation between functional impairment and developmental psychopathology, there is also evidence that early externalizing problems can negatively affect later identity formation (Crocetti, Klimstra, Hale, Koot, & Meeus, 2013). Despite the ample evidence that social, school, and health functioning exhibits reciprocal relations with mental health problems, to date, no studies have examined the prospective links between changes in psychosocial functioning and BPD symptoms in adolescents.

Thus, there is some concurrent evidence to hint that a shared association between trajectories of BPD symptoms and psychosocial functioning likely exists. However, studies have not yet examined whether the trajectories of psychosocial indicators track with development of BPD symptoms throughout adolescence. Furthermore, no work has examined whether domains of adolescent psychosocial functioning may be unique to BPD, beyond an indication of overall poorer mental health. Indeed, BPD significantly covaries with both internalizing and externalizing disorders in epidemiological (Eaton et al., 2011) and clinical samples (Zimmerman et al., 2005) in adulthood. As such, determining if there are any unique links, by parsing out other forms of psychopathology, may provide valuable clinical insight as to whether particular domains of psychosocial functioning are developmentally linked to BPD in adolescence.

Understanding the psychosocial concomitants of adolescent BPD development, including the trajectory of academic functioning, peer relations, and other aspects of health, would provide a window into the impact of BPD symptoms during adolescence. In the current study we address these questions by modeling trajectories of BPD symptom development in a large sample of adolescent girls between the ages of 14 and 17, and concurrent development of academic performance, extracurricular involvement, global assessment of functioning, mental health
treatment utilization, self perception, social skills, and sexual activity. We estimate latent growth models for BPD symptoms and each of the psychosocial functioning constructs and correlate individual differences in initial values and rates of change between BPD symptoms and functioning. We then examine the specificity of these associations with BPD by controlling for depression and conduct disorder features.

**Method**

**Sample Description**

The Pittsburgh Girls Study (PGS; \( N = 2,450 \)) involves an urban community sample of four cohorts of girls, ages 5-8 at the first assessment, and their primary caretaker, followed annually according to an accelerated longitudinal design. To identify the study sample, low income neighborhoods were oversampled, such that neighborhoods in which at least 25% of families were living at or below poverty level were fully enumerated and a random selection of 50% of households in all other neighborhoods were enumerated (see Hipwell et al., 2002 for full study recruitment details). The analyses presented here cover ages 14 to 17 years. African American girls made up slightly more than half of the sample (53%), while 41.2% were Caucasian. Most of the remaining 5.8% of girls were described as multi-racial. The overwhelming majority of caretakers were parents (92.0% biological/birth parents; 0.7% stepparents; 2.8% adoptive parents; and 0.3% foster parents). Thus, we refer to caregivers as parents. Most parents (57%) were cohabiting with a spouse or domestic partner; and 50% completed >12 years of education.

**Data Collection**

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

**Measures**
**BPD symptoms.** BPD symptoms were assessed with girls’ reports when they were 14-17 years-old using the questions from the screening questionnaire of the International Personality Disorders Examination (IPDE-BOR; Loranger et al., 1994). Parent-report was not obtained. The IPDE-BOR consists of nine items (e.g., *I get into very intense relationships that don’t last*) scored either “true” or “false.” Adequate concurrent validity, and sensitivity and specificity of BPD symptom scores to clinicians’ diagnosis have been demonstrated for the IPDE-BOR in a sample of youth and a score of 4.0 or greater may be considered in the clinically significant range (Smith, Muir, & Blackwood, 2005). To demonstrate the level of severity in this sample, the upper quartile had an average score of 5.1 at ages 14-15, 5.0 at age 16, and 4.4 at age 17. The internal consistency for BPD symptoms ranged from $\alpha = .68$ to .69 across time-points.

**Academic Performance.** Parents rated the girls’ academic achievement on a 5-point scale ranging from 1 (far above average) to 5 (failing). Achievement was rated separately for reading, writing, and arithmetic. Items were averaged so that higher scores indicated better academic achievement. Internal consistencies were uniformly $\alpha = .85$ across time points.

**Activity Involvement.** Activities assessed included: Extra-curricular school activities; non-school clubs; fun family activities, volunteering, and religious activities. Time spent in each activity was rated by the girl on a 6-point scale, ranging from 1 (just about every day) to 6 (never). Thus higher-scores indicated lower activity involvement. Internal consistency for these five activities ranged from $\alpha = .64$ to $\alpha = .68$ across time points.

**Mental Health Treatment Utilization.** Girls were asked to report on whether they received the following mental health treatment from a therapist, hospital, community agency, or whether they were institutionalized in the last year. Internal consistency for these four questions ranged from $\alpha = .51$ to $\alpha = .55$ across time points.

**Global Assessment of Functioning.** The child’s level of global impairment was rated by
the parent using the Children’s Global Assessment Scale (Setterberg, Bird, Gould, Shaffer, & Fisher, 1992). This scale assessed the child’s lowest level of functioning across four domains (home, school, with friends, and duration leisure time) during the past six months. A continuous 100-point scale with descriptive anchors ranges from 1-10 (extremely impaired) to 91-100 (doing very well). Scores of lower than or equal to 60 represents significant impairment of functioning.

Self-Perception. Self-perception was measured using girls’ report on the Perceptions of Peers and Self inventory (POPS; Rudolph et al., 1995). The POPS was included in order to capture social-cognitive perceptions of the self and self in relation to peers. Three scales scored from the child-report of the POPS were used as the observed measures of self-perception. These include Social Self-Worth, Self-Competence, and Peer-Victimization. In contrast to all other study measures, the POPS was only administered at ages 14, 15, 16, but not 17. Internal consistency for these three scales ranged from $\alpha = .70$ to $\alpha = .71$ across time points.

Social Skills. Girls and their parents were each asked about their interpersonal/social functioning using the Assertion, Cooperation, Self-Control, and Empathy scales of the Social Skills Rating Scale (Gresham & Elliot, 1990). Internal consistency for these four scales ranged from $\alpha = .81$ to $\alpha = .84$ across time points for child report, and from $\alpha = .82$ to $\alpha = .85$ across time points for parent report.

Sexual Behavior. Girls were asked about sexually intimate behaviors in the past year using 4 items adapted from the 13-item Adolescent Sexual Activity Index (ASAI; Hansen, Paskett, & Carter, 1999). The ASAI has been validated on a large community sample of male and female adolescents (ages 12-19), and has demonstrated high internal consistency and reliability. Items were scored as either 0 (no) or 1 (yes), and included: Boy’s hands under
clothes; Undressed with organs exposed; Intercourse with boy; and Oral sex with boy. The adapted measure used in the current study utilized a ‘past year’ timeframe instead of the ‘past 30 days’ of the original measure to enable low frequency behaviors to be captured. Internal consistency for these four sexual behaviors ranged from $\alpha = .85$ to $\alpha = .89$ across time points.

**Depression.** Girls reported on symptoms of depression using the Adolescent Symptom Inventory—Fourth Edition (ASI-4; Gadow & Sprafkin, 1998). This measure includes the nine symptoms of DSM-IV major depression disorder. Endorsed items were summed to generate symptom counts ranging from 0 to 9.

**Conduct Disorder.** Girls reported on symptoms of conduct disorder using the ASI-4 (Gadow & Sprafkin, 1998). This measure includes the 15 symptoms of DSM-IV conduct disorder. Endorsed items were summed to generate symptom counts.

**Analysis**

We developed nine domain-specific second-order latent growth curve models (LGMs described below) to establish mean trajectories and individual variability, followed by estimating parallel process (i.e., multivariate) LGMs to test the covariation among initial levels and rates of change between BPD and functioning domains. All models were estimated in Mplus 7.11 (Muthén & Muthén, 2012), using a robust weighted least squares estimator (WLSMV) and the theta parameterization in models with categorical items.

With the exception of our global assessment of functioning, all of the functioning measures (e.g., mental health utilization, social skills, etc.) described above included multiple items or scales. Accordingly, to account for measurement error and improve reliability of estimates, we modeled BPD and each of the functioning scales as latent variables at each time

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1 An additional item reflecting *Girls hands underneath a boy’s clothes* was nearly perfectly correlated with the *Boy’s hands under clothes* item, which caused problems in estimation when including both. Therefore it was
point. An important issue in longitudinal developmental research is whether the measurement of a construct of interest is consistent across time (Widaman, Ferrer, & Conger, 2010). In particular, for a latent variable to represent the same construct at each measurement occasion, at least the factor loadings, if not indicator intercepts or thresholds, must be invariant over time (Meredith, 1993). Therefore prior to modeling any LGM, we ran a series of confirmatory factor analysis (CFA) models for each construct between ages 14 and 17, testing for measurement invariance across all four ages. For each construct, we first tested a model that imposed the same structure but allowed factor loadings and thresholds were free to vary at each age, which was then compared to a model in which factor loadings were fixed to equality across all ages, followed by a model with equal intercepts at each year, and finally a model with equality of factor variances across all years using the appropriate chi-squared difference tests (e.g., DIFFTEST option in MPlus when using WLSMV). Error variances for each indicator were freely estimated across time points. Additionally, error variances from corresponding items were allowed to covary across waves, and when indicated based on fit tests, comparable distances were fixed to equality (e.g., the covariance between affective lability item at time 1 and time 2 was held equivalent to the corresponding covariance between time 2 and time 3, etc.).

We then used these time-invariant latent variables as the observations for the LGMs. That is to say, we modeled growth in latent constructs as opposed to basic scale scores (this type of model is sometimes referred to as a second-order LGM; see e.g., Hancock, Kuo, & Lawrence, 2001). LGMs offer a flexible approach to estimating the mean shape of change (i.e., level and rate), and individual heterogeneity around the average trajectory (i.e., growth factor variances), in a given psychological construct (e.g., BPD, psychosocial functioning), using specific parameterizations of latent factors. In a standard approach, latent intercept and slope factors capture the level and linear rate of change in a given construct, respectively. Individual
heterogeneity in the shape and rate of change can be quantified by the estimated variances of the growth factors. LGMs can also be treated in a multivariate fashion, capturing growth in more than one psychological construct simultaneously (i.e., parallel processes), and assessing the associations (i.e., covariances) between growth factors across systems. Parallel process (alternatively multivariate or associative) LGMs can therefore test whether the trajectories in BPD symptoms trajectories track with the trajectories in psychosocial functioning domains, offering a direct test of our hypotheses. Figure 1 provides a conceptual diagram of these models as they were fitted in this study. Of primary interest are the covariation between the intercept factors (i.e., initial level of growth trajectory) for BPD symptoms and functioning, and the covariation between slope factors (i.e., rates of change). In order to protect against the potential for spurious associations between slopes that merely reflect correlated regression to the mean, we regressed each slope factor on the same construct’s intercept factor (e.g., BPD slope was regressed on BPD intercept). In other words, this controls for initial values when estimating the slope correlations. BPD and functioning intercepts and slopes were allowed to correlate freely so as not to bias estimates of slope covariation.

Finally, in order to determine whether the associations between BPD and functioning were specific as opposed to reflective of poor-mental health generally, we re-estimated all of the parallel process LGMs but now controlling for child reported depression and conduct disorder symptoms at each time point. In each model latent BPD and functioning variables at each time point were regressed on concurrently assessed child-reported depression and conduct disorder symptoms.

Results

Measurement Invariance

All levels of measurement invariance held for the CFAs for each construct at each time-
point. We additionally freed corresponding item residual covariances across time-points to account for shared content, and these were constrained to equality for equal temporal distances. For sexual behavior, adding item residual covariances resulted in problems with estimation so they were excluded, yet model fit remained excellent. Due to space considerations we do not present detailed results of these tests, but they are available from the first author on request.

**LGMs**

We next fitted a series of LGMs to each construct. With the exception of the LGM fitted to global functioning (which was assessed with only one item), these were second-order LGMs, such that growth was modeled as a latent trait comprised of items/subscales at each age. These models were adapted from the strict measurement invariance CFAs described above. For each construct we fitted a linear LGM, with an intercept and linear slope factor capturing mean and individual variability in trajectories over time. The resulting model fit criteria are summarized in Table 1. As can be seen, all growth models provided an excellent fit to the data (i.e., non-significant RMSEA, CFI and TLI > .95; Hu & Bentler, 1999) suggesting that linear growth curves provide a close fit to the observed growth trajectories.²

Table 2 contains the key parameter estimates from the growth factors, including initial level (i.e., intercept factor means), rate of change (i.e., slope factor means), and individual variability in initial levels and rates of change (i.e., factor variances). For all second-order LGMs, the initial value (i.e., intercept) of the growth trajectory is fixed to 0.0 because it is associated with the latent factor mean, and therefore represents the initial average in the sample. The slope mean reflects direction and rate of change per year from this initial average. For BPD

² We additionally estimated and considered quadratic LGMs. In some instances the addition of a quadratic factor significantly improved fit despite the fact that even in those cases there was generally non-significant variance in the quadratic growth factor, and the curvature was modest. Thus, we elected to retain the linear models. We note, however, that we tested parallel process quadratic models when it was potentially indicated and the conclusions related to cross-variable associations in growth remain identical.
symptoms, mental health treatment, and self-perception we observed no change, on average, across assessment waves. Global functioning, parent and child reported social skills, problems with academic performance, and sexual activity all demonstrated increases across the course of adolescence. On the other hand, participation in extracurricular activities decreased on average over the course of the study, indicating an overall lower involvement with age (where higher scores reflect less involvement). Importantly, regardless of the degree of mean change, there was significant individual variability in initial value and rate of change across the study for all study variables. Thus, individuals started at different levels and changed at varying directions and rates across adolescence. In Figure 2 we provide a diagram of the observed heterogeneity in estimated BPD growth trajectories. As is readily seen, some individuals remained flat, consistent with the mean, whereas other increase and decrease dramatically.\(^3\) This type of rich heterogeneity in growth is a prerequisite for examining covariation in the developmental trajectories of BPD symptoms and psychosocial functioning.

The observed variability in trajectories supported parallel process or bivariate LGMs to establish the associations between individual trajectories in BPD growth and psychosocial functioning. Table 3 contains the correlations between intercept and growth factors for each of these models, which are the primary parameters of interest for study hypotheses. Individual differences in initial levels (i.e., intercept factors) uniformly correlated positively across domains, such that age 14 BPD symptoms were associated with worse psychosocial functioning. All correlations were positive and the point estimates ranged from modest to strong \((Mdn \ r = .50; \ range = .21-.61)\). A similar pattern of associations was observed for the growth factors \((Mdn \ r = .44, \ range = .29-.74)\). This indicates that as BPD symptoms increase in adolescence, so

\(^3\) Due to space limitations, we only provide a figure for BPD growth trajectories. However, plotting the remaining study constructs generate similarly diverse patterns of growth.
does psychosocial dysfunction, and as BPD symptoms decline, psychosocial functioning improves. The strongest links in the trajectories of BPD symptoms and functioning were for self-reported social skills, sexual activity, and self-perception.

Finally, given that BPD shares features of both internalizing and externalizing pathology, we estimated a series of LGMs that controlled for contemporaneous depression and conduct disorder symptoms. Results are presented in Table 4. Although BPD intercepts uniformly demonstrated significant associations with each of the functioning factors when controlling for depression and conduct disorder, the only slope associations that remained significant were child-rated social skills, self-perception, and sexual activity.

**Discussion**

Given the clinical severity of BPD, it is concerning that so little is known about how BPD tracks with key indicators of social, academic, and health functioning during adolescence. This study examined how BPD symptoms and psychosocial functioning co-developed across adolescence in a large sample of girls. Using a series of bivariate LGMs, we found consistent evidence that individual differences in BPD symptoms tracked with changes in seven different domains of adolescent psychosocial functioning. In support of our hypotheses, increasing BPD symptoms were coupled with worsening social, academic, and mental health outcomes throughout the adolescent period, and amelioration of symptoms is associated with gains in functioning. Furthermore, analyses controlling for both internalizing and externalizing pathology suggested specific developmental associations of BPD with social-skills, self-perception, and sexual activity that go beyond general associations with psychopathology.

The results of the present analyses underscore the pervasive nature of personality pathology, as every psychosocial domain at least modestly tracked with initial levels and changes of BPD symptoms, and many exhibited strong links in changes over time. Furthermore, these
results broaden our understanding of why BPD is so concerning in this age group. Perhaps because suicidal behaviors and treatment difficulties elicit so much fear and frustration in parents and providers (Bourke & Grenyer, 2013; Hoffman, Buteau, Hooley, Fruzzetti, & Bruce, 2003; Miller et al., 2010), these clinically-relevant behaviors may inadvertently overshadow other critical difficulties that warrant attention in BPD as they may have long term consequences, both for girls as well as for society. Across individuals, there were strong effect sizes for the links between BPD symptoms with social skills and self-perception, which if compromised, threaten key developmental tasks during this period of peer friendship and identity formation (Roisman, Masten, Coatsworth, & Tellegen, 2004). Failure to effectively cultivate these skills during this developmental period may ultimately result in the severe impairments in self and interpersonal functioning recognized as the hallmark of personality pathology in adulthood (Luyten & Blatt, 2013; Bender et al., 2011; Hopwood et al., 2013; Kernberg, 1985). As an example of a broader societal outcome, poorer performance in high school academics could threaten long-term academic achievement, limiting employment and economic stability across the lifespan (French, Homer, Popovici, & Robins, 2014). In the health domain, early initiation of sexual activity may increase lifetime sexual partners, the likelihood of contracting sexually-transmitted diseases, or early and unplanned pregnancies (Kaestle, Halpern, Miller, & Ford, 2005; Sneed, 2009).

These findings have important implications for clinical intervention as well as educational and public health policy as they heighten the importance of why acknowledging the existence of and treating personality pathology during adolescence is critical. In other words, the cost of not acknowledging or treating BPD during adolescence goes beyond the confines of psychological or psychiatric treatment. The presence of BPD symptoms during adolescence may negatively affect the individual, the family, peers, school systems, and could have implications for public health. While many providers are either concerned with the stigma associated with a
BPD diagnosis, or unaware that a diagnosis of BPD is permissible by DSM standards (Laurensen, Hutsebaut, Feenstra, Van Busschbach, & Luyten, 2013), providers should also consider the costs associated with not assessing and treating salient BPD symptoms.

While poorer psychosocial outcomes are concerning in their own right, they are additionally problematic because they ripen the possibility of continued or exacerbated mental health problems in the future (Boden, Fergusson, & Horwood, 2008). Developmental psychopathologists have long been aware of the ‘canalization process’ by which compounding risk factors over development increase the probability of the emergence of psychopathology (Blair & Cybele, 2012). A tenet of developmental psychopathology also emphasizes viewing development as ‘probabilistic’ and not ‘deterministic’ (Sroufe, 1997). While the present results were certainly consistent across the various domains, sometimes with large effect sizes, it is important to remember that there were significant individual differences in the correspondence between BPD symptoms and psychosocial functioning. This suggests that some adolescent girls endorsing features of BPD may be doing well with academics, peer groups, or aspects of health related functioning. While it is possible that some of these girls were performing normatively compared to peers across these domains, despite emerging BPD symptoms, it may also be that these girls were having difficulties in domains not assessed (e.g., family conflict).

In addition to understanding the broad patterns between BPD symptoms and psychosocial functioning, it was also important to examine tests of specificity given the high degree of covariation of BPD and both internalizing and externalizing psychopathology. After controlling for internalizing and externalizing psychopathology, relations between self, interpersonal, sexual functioning and BPD symptoms all retained significant links across the adolescent period. It may not be surprising that these domains of functioning were uniquely linked to BPD symptoms, as challenges within the areas of identity (self) and interpersonal relationships (others) are central
to BPD pathology (Fonagy & Leuyten, 2009; Kernberg, 1984; Linehan, 1993). Furthermore, while all the psychosocial domains selected for this study were included due to their relevance within the adolescent period, aspects of identity formation and forming significant peer and romantic relationships are perhaps the most central tasks of adolescent development (Blakemore & Mills, 2014). This is consistent with the pattern of a pernicious cycle in which BPD symptoms interrupt core developmental processes, which if impaired or delayed, exacerbate key features of BPD.

Both the broad and unique links between psychosocial functioning and BPD symptoms are important to consider. The broad based associations are a reminder that adolescents with BPD symptoms will most likely exhibit features of internalizing and externalizing psychopathology, which negatively affect areas of psychosocial functioning very generally. The domains of most concern, though, are the unique associations with relating to self and others that affect central aspects of BPD and adolescent development.

In addition to testing whether adolescent girls’ BPD symptoms tracked with psychosocial functioning, these findings contribute to the literature on individual differences in BPD across adolescence. Similar to adult personality pathology studies, we found significant variability in adolescent BPD symptoms across time. Some girls exhibited stable BPD characteristics over time, some girls increased in severity, and some girls’ BPD symptoms abated. This finding is critically important for several reasons. First, it is in line with recent findings from the adult literature (Morey & Hopwood, 2013) demonstrating that personality pathology is not inherently “enduring” or a “pattern that is stable and of long duration” (American Psychiatric Association, 2013; pp. 646-647). We are careful to point out that these findings do not, however, give credence to the idea that it is inappropriate to diagnose personality pathology in adolescents because it is viewed as ‘transient’ (Laurensen et al., 2013). Rather, our findings reinforce the
view that personality disorders are dynamic phenomena that wax and wane in harmony with other aspects of functioning as an individual matures across the life span (Vachon et al., 2013; Wright et al., 2011). Secondly, these results further support the argument that there are many similarities between personality pathology in adolescence and adulthood, and sharply distinguishing between the two is inadvisable, even though developmental considerations are warranted (Tackett, Balsis, Oltmanns, & Krueger, 2009). Third, these findings may assure clinicians who have assessed for BPD that giving a diagnosis is not equivalent to giving a life sentence. Knowing that there is great variability with which adolescents continue to exhibit BPD symptoms may also combat stigma that results from the belief that the adolescent’s prognosis is bleak. Given the emerging evidence that BPD varies over time in adulthood has led to a shift in stance on the treatability and prognosis for borderline personality disorder (Gunderson, 2009). Similarly, clinicians may need to find a new stance by which to view personality pathology when treating adolescents. Such a stance could include the notion that personality pathology in adolescent populations is a clinically important problem that impacts several domains of functioning and as such, it should be assessed, diagnosed, and treated, if appropriate. Additionally, a clinical stance should recognize that there is a wide range for how personality pathology endures or desists. Clinical interventions that help adolescents improve in their academics, peer relations, and health-related behaviors may ultimately contribute to remitting BPD symptoms over time.

This is the first study to examine how adolescent BPD and a comprehensive battery of psychosocial domains are linked over time, thus contributing to our understanding of the seriousness of BPD features in the adolescent period. Strengths of this study include using a longitudinal, at-risk community sample, measuring an array of adolescent psychosocial domains, and using a rigorous empirical test to examine whether BPD symptoms and psychosocial
functioning are linked during adolescence. Although this study broadened our understanding of why adolescent BPD is serious, there are several questions that cannot be answered with the current sample. First and most obvious, these findings are only generalizable to girls. As studies measuring BPD using community data have revealed that BPD equally affects both genders (Lenzenweger et al., 2007; Zimmerman & Coryell, 1989), future studies should sample and test similar hypotheses with male adolescents. Our finding that BPD was stable across these years is somewhat inconsistent with prior findings (e.g., Cohen et al., 2005), and may reflect the strong representation of disadvantaged girls in the study. It is conceivable that different samples may lead to different results for the associations found here. Also, results should be interpreted in the context of the measure of BPD used, which uses binary items to assess BPD as defined in the diagnostic nomenclature. Other measures that provide a broader and more textured conceptualization of the construct may reveal additional insight (e.g., Zanarini et al., 1989).

This study is also limited by some of the methodologies employed, such as the heavy reliance on self-report and the correlational nature of the analyses that cannot establish predictive primacy among the measures. Shared reporter variance may account for why the findings were highly consistent across psychosocial functioning domains. Despite acknowledging this, there is some evidence to suggest that the findings did not result solely because girls reported on most of the measures. For instance, mothers’ reported on girls social skills and these findings were quite similar to what girls themselves reported, albeit with more modest effect sizes. Additionally, BPD symptoms and psychosocial functioning did not correspond perfectly, suggesting that these findings did not result from girls rating things entirely negatively or positively. However, the correlational properties of the LGM’s cannot rule out the possibility that other variables contributed to links between BPD and psychosocial functioning. Relatedly, cleanly differentiating between measures of psychosocial functioning and mental disorder per se can be
difficult, and in the case of personality pathology may reflect arbitrary distinctions to some degree (e.g., Clark & Ro, 2014). These lines are blurry and may better reflect conventions of measurement as opposed to meaningful psychological distinctions, especially when considering the measures of self and interpersonal functioning that exhibited developmental relationships with BPD even when controlling for internalizing and externalizing pathology. Finally, we note that for some measures used here the internal consistency was lower than desirable. Nevertheless, our analytic approach guards against this impacting our results because we based our growth estimates on latent factors that offer error free estimates of an individual’s standing on a particular construct.

The current findings on the links between adolescent girls’ BPD symptoms and psychosocial outcomes provide a comprehensive depiction of the challenges a teenager with BPD symptoms might face throughout adolescence. To date, the developmental psychopathology literature has tested critical prospective examination on the etiology of BPD, and clinical and intervention trials have documented the clinical course and co-morbid presentations associated with the diagnosis. The present work should serve as a logical complement by adding to the field’s understanding of expected and natural variation in BPD features across adolescence, and provide a more person-centered view of the problems confronting girls with features of BPD. Ultimately, we hope this work adds to the growing literature demonstrating that the emergence of BPD during adolescence is valid and while a simple treatment plan may not suffice, it is not an immutable disorder, and change is possible. Perhaps adolescence may ultimately prove to be the most fruitful age for intervention.
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Table 1. Summary of Growth Model Fit Statistics

<table>
<thead>
<tr>
<th>Domain</th>
<th>$\chi^2$</th>
<th>df</th>
<th>RMSEA</th>
<th>RMSEA 90% CI</th>
<th>RMSEA p</th>
<th>CFI</th>
<th>TLI</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD Symptoms</td>
<td>943.79</td>
<td>475</td>
<td>.021</td>
<td>.019-.023</td>
<td>1.00</td>
<td>0.98</td>
<td>0.97</td>
</tr>
<tr>
<td>Academic Performance</td>
<td>161.54</td>
<td>74</td>
<td>.023</td>
<td>.018-.028</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Extracurricular Activities</td>
<td>666.40</td>
<td>236</td>
<td>.029</td>
<td>.026-.031</td>
<td>1.00</td>
<td>0.98</td>
<td>0.98</td>
</tr>
<tr>
<td>Mental Health Treatment</td>
<td>148.00</td>
<td>108</td>
<td>.013</td>
<td>.007-.018</td>
<td>1.00</td>
<td>0.99</td>
<td>0.99</td>
</tr>
<tr>
<td>Global Functioning</td>
<td>5.47</td>
<td>5</td>
<td>.007</td>
<td>.000-.031</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Self Perception</td>
<td>101.29</td>
<td>32</td>
<td>.031</td>
<td>.025-.038</td>
<td>1.00</td>
<td>0.99</td>
<td>0.99</td>
</tr>
<tr>
<td>Social Skills (Child report)</td>
<td>217.73</td>
<td>65</td>
<td>.033</td>
<td>.028-.037</td>
<td>1.00</td>
<td>0.99</td>
<td>0.99</td>
</tr>
<tr>
<td>Social Skills (Parent report)</td>
<td>247.23</td>
<td>65</td>
<td>.036</td>
<td>.031-.040</td>
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<tr>
<td>Sexual Activity</td>
<td>265.55</td>
<td>112</td>
<td>.025</td>
<td>.021-.029</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Note. BPD = borderline personality disorder. RMSEA = Root Mean Square Error of Approximation; CI = Confidence Interval; CFI = Comparative Fit Index; TLI = Tucker Lewis Index.
Table 2. Individual Latent Growth Curve Model Parameters

<table>
<thead>
<tr>
<th>Domain</th>
<th>Intercept Mean (Fixed Effect)</th>
<th>Intercept Variance (Random Effect)</th>
<th>Slope Mean (Fixed Effect)</th>
<th>Slope Variance (Random Effect)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coeff</td>
<td>95% CI</td>
<td>p</td>
<td>Coeff</td>
</tr>
<tr>
<td>BPD Symptoms</td>
<td>0.00</td>
<td>--</td>
<td>--</td>
<td>-0.01</td>
</tr>
<tr>
<td>Academic Performance</td>
<td>0.00</td>
<td>--</td>
<td>--</td>
<td><strong>0.09</strong></td>
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<tr>
<td>Extracurricular Activities</td>
<td>0.00</td>
<td>--</td>
<td>--</td>
<td>0.08</td>
</tr>
<tr>
<td>Mental Health Treatment</td>
<td>0.00</td>
<td>--</td>
<td>--</td>
<td>-0.11</td>
</tr>
<tr>
<td>Global Functioning</td>
<td><strong>78.69</strong></td>
<td>78.12-79.26</td>
<td>&lt;.001</td>
<td><strong>0.41</strong></td>
</tr>
<tr>
<td>Self Perception</td>
<td>0.00</td>
<td>--</td>
<td>--</td>
<td>-0.01</td>
</tr>
<tr>
<td>Social Skills (Child report)</td>
<td>0.00</td>
<td>--</td>
<td>--</td>
<td><strong>0.16</strong></td>
</tr>
<tr>
<td>Social Skills (Parent report)</td>
<td>0.00</td>
<td>--</td>
<td>--</td>
<td>0.08</td>
</tr>
<tr>
<td>Sexual Activity</td>
<td>0.00</td>
<td>--</td>
<td>--</td>
<td><strong>1.19</strong></td>
</tr>
</tbody>
</table>

Note. BPD = borderline personality disorder. All coefficients presented in unstandardized format. Mean/Fixed effect for intercept reflects initial value at age 14; Mean/Fixed effect for Slope reflects rate of change per year. Underlined coefficients were fixed to zero in the model for identification purposes.
Table 3. Associations between intercept and slope parameters from parallel process growth curve models of functioning and BPD.

<table>
<thead>
<tr>
<th>Domain of Functioning</th>
<th>Intercept</th>
<th>Slope</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coeff. 95% CI</td>
<td>Coeff. 95% CI p</td>
</tr>
<tr>
<td>Academic Performance</td>
<td>.34 .28-.40 &lt; .001</td>
<td>.33 .06-.60 .017</td>
</tr>
<tr>
<td>Extracurricular Activities</td>
<td>.21 .14-.28 &lt; .001</td>
<td>.29 .11-.47 .002</td>
</tr>
<tr>
<td>Mental Health Treatment</td>
<td>.55 .47-.64 &lt; .001</td>
<td>.47 .18-.75 .011</td>
</tr>
<tr>
<td>Global Functioning</td>
<td>.50 .55-.44 &lt; .001</td>
<td>.41 .23-.59 &lt; .001</td>
</tr>
<tr>
<td>Self Perception</td>
<td>.49 .43-.55 &lt; .001</td>
<td>.50 .29-.71 &lt; .001</td>
</tr>
<tr>
<td>Social Skills (Child report)</td>
<td>.61 .67-.55 &lt; .001</td>
<td>.74 .54-.95 &lt; .001</td>
</tr>
<tr>
<td>Social Skills (Parent report)</td>
<td>.39 .45-.32 &lt; .001</td>
<td>.33 .11-.54 .003</td>
</tr>
<tr>
<td>Sexual Activity</td>
<td>.60 .52-.69 &lt; .001</td>
<td>.54 .32-.76 &lt; .001</td>
</tr>
</tbody>
</table>

Note. BPD = borderline personality disorder. All coefficients (Coeff.) standardized. Scores for Global Functioning (GAF) and Social Skills (both child and parent report) scales have been reversed so that higher scores on all scales reflect more impairment. Thus, coefficients across scales are directly comparable.
Table 4. Associations between intercept and slope parameters from parallel process growth curve models of functioning and BPD controlling for child-reported conduct disorder and major depressive symptoms at each time point.

<table>
<thead>
<tr>
<th>Domain of Functioning</th>
<th>Intercept</th>
<th></th>
<th></th>
<th>Slope</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coeff.</td>
<td>95% CI</td>
<td>p</td>
<td>Coeff.</td>
<td>95% CI</td>
<td>p</td>
</tr>
<tr>
<td>Academic Performance</td>
<td>0.29</td>
<td>0.14-.33</td>
<td>&lt; .001</td>
<td>0.04</td>
<td>-0.02-.02</td>
<td>.875</td>
</tr>
<tr>
<td>Extracurricular Activities</td>
<td>0.12</td>
<td>0.03-.22</td>
<td>.012</td>
<td>0.19</td>
<td>-0.11-.49</td>
<td>.213</td>
</tr>
<tr>
<td>Mental Health Treatment</td>
<td>0.35</td>
<td>0.23-.47</td>
<td>&lt; .001</td>
<td>0.05</td>
<td>-0.45-.55</td>
<td>.836</td>
</tr>
<tr>
<td>Global Functioning</td>
<td>0.31</td>
<td>0.22-.39</td>
<td>&lt; .001</td>
<td>0.07</td>
<td>-0.24-.39</td>
<td>.455</td>
</tr>
<tr>
<td>Self Perception</td>
<td>0.32</td>
<td>0.23-.40</td>
<td>&lt; .001</td>
<td>0.36</td>
<td>0.15-.58</td>
<td>.001</td>
</tr>
<tr>
<td>Social Skills (Child report)</td>
<td>0.38</td>
<td>0.28-.48</td>
<td>&lt; .001</td>
<td>0.39</td>
<td>0.02-.76</td>
<td>.039</td>
</tr>
<tr>
<td>Social Skills (Parent report)</td>
<td>0.30</td>
<td>0.21-.38</td>
<td>&lt; .001</td>
<td>0.05</td>
<td>-0.30-.41</td>
<td>.766</td>
</tr>
<tr>
<td>Sexual Activity</td>
<td>0.54</td>
<td>0.40-.68</td>
<td>&lt; .001</td>
<td>0.65</td>
<td>0.29-1.00</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

Note. BPD = borderline personality disorder. Significant values bolded. All coefficients (Coeff.) standardized. Scores for Global Functioning (GAF) and Social Skills (both child and parent report) scales have been reversed so that higher scores on all scales reflect more impairment. Thus, coefficients across scales are directly comparable.
Figure 1. Conceptual diagram of second-order parallel process latent growth curve model as estimated in this study. Ovals represent latent variables, numbers represent growth factor loadings, curved errors represent covariances among growth factors, straight arrows between latent factors represent regression paths controlling for initial values. bpd = borderline personality disorder symptoms; BPD = borderline personality disorder latent factor; func = functioning items; FUNC = functioning latent factors.
Figure 2. Diagram of individual estimated growth curve trajectories for one random sixth of the sample. A sub-sample was randomly selected to better illustrate growth heterogeneity.