Published in final edited form as:

J Abnorm Child Psychol. 2017 August ; 45(6): 1157–1167. doi:10.1007/s10802-016-0225-x.

A Within-Person Analysis of the Association Between Borderline Personality Disorder and Alcohol use in Adolescents

Sophie A. Lazarus¹, Jordan Beardslee¹, Sarah L. Pedersen¹, and Stephanie D. Stepp¹

Department of Psychiatry, University of Pittsburgh School of Medicine

Abstract

Many studies examining the association between borderline personality disorder (BPD) and alcohol use during adolescence have focused on between-individual differences (rank order stability), comparing whether adolescents with elevated rates of alcohol use have higher BPD symptoms than those with lower rates of alcohol use. As such, the extent to which an individual's alcohol use is associated with concurrent and future BPD symptoms has been relatively unstudied. The current study assessed year-to-year fluctuations in alcohol use and BPD symptoms in a large urban sample of girls from age 14 to age 17 ($N = 2450$). The primary aim was to examine whether increases in alcohol use were associated with increases in adolescent girls' BPD symptoms in the same year and in the following year. Results of fixed-effects (within-individual) models revealed that even while controlling for the time-varying impact of symptoms of both internalizing and externalizing disorders, prior and concurrent other substance use, and all time invariant, pre-existing differences between individuals, higher past-year alcohol use was associated with higher levels of BPD symptoms. Furthermore, this association did not vary by age, or by sociodemographic factors, including child race and socioeconomic status of the family. The results of this study indicate heightened risk for the exacerbation of BPD symptoms following increases in alcohol use frequency and highlight the potential utility of interventions targeting drinking behavior for preventing escalations in BPD symptoms.

Keywords

Borderline personality disorder; Adolescence; Alcohol use

While borderline personality disorder (BPD) is known to have high rates of comorbidity with alcohol use disorder (AUD) during adulthood, (see Trull, Sher, Minks-Brown, Durbin, & Burr, 2000 for review), accumulating research indicates that this association is also present during adolescence (Cohen, Chen, Crawford, Brook, & Gordon, 2007; Stepp, Trull, & Sher, 2005; Trull, Waudby, & Sher, 2004). Furthermore, adolescent alcohol use predicts BPD symptoms and diagnosis in adulthood (Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001; Thatcher, Cornelius, & Clark, 2005), suggesting that adolescence is a potentially critical time to understand how alcohol use may increase risk for developing BPD. The existing research has exclusively examined the relationship between alcohol use and BPD

symptoms at the between-person level (i.e., do those with higher levels of alcohol use on average also have higher levels of BPD at the next time-point) of analysis, limiting our understanding of how and whether alcohol use affects BPD symptoms within individuals as these symptoms emerge and fluctuate throughout adolescence. To our knowledge, no research has assessed how shifts in alcohol use during adolescence may exacerbate BPD symptoms. In order to understand these risk processes at the within-individual level, the focus must move towards understanding the developmental associations between these symptoms and behaviors as they unfold, year-to-year, during adolescence. This type of analysis can identify both concurrent and predictive associations that may ultimately aid in understanding how the emergence of alcohol use affects risk for BPD. This work is needed so that interventions can occur before these problems can canalize, and become more difficult to treat.

BPD is a severe mental illness that is characterized by unstable relationships, impulsivity, fears of abandonment, suicidal behavior, and difficulties with emotion regulation (American Psychiatric Association, 2013). BPD is found at higher rates among women compared to men in community and outpatient samples (Korzekwa, Dell, Links, Thabane, & Webb, 2008; Trull, Jahng, Tomko, Wood, & Sher, 2010), and related to greater distress and disability in this population compared to men (Grant et al., 2008). There is also growing consensus that symptoms of BPD are present by adolescence (Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Chanen & Kaess, 2011) and the discrepancy in rates of diagnosis across gender is already present at this time (Eppright, Kashani, Robison, & Reid, 1993; Grilo et al., 1996), suggesting a need to more closely examine predictors of the disorder in females. Furthermore, BPD symptoms put adolescents at risk for multiple adult disorders, including a BPD diagnosis (Johnson et al., 2000). Importantly, even subclinical levels of BPD are related to clinically significant impairment in psychiatric outpatients (Zimmerman, Chelminski, & Young, 2011) as well as impaired social, emotional, and academic functioning among young adults (Trull, Useda, Conforti, & Doan, 1997). Thus, BPD symptoms during adolescence are related to significant distress and put one at risk for BPD and other serious psychiatric comorbidities.

Studies examining the course of BPD symptoms point to adolescence as a key period in the development of the disorder, as well as an important time to intervene to mitigate future impairment. First, dimensional measures of BPD symptoms show considerable stability during childhood and adolescence (Chanen et al., 2004; Johnson et al., 2000). Other studies have found that BPD symptoms peak between approximately ages 14–17 and decline thereafter (Bornoalova, Hicks, Patrick, Iacono, & McGue, 2011; Stepp et al., 2014b). This research indicates that mid-to late-adolescence represents a time of increased risk for BPD symptoms, but also highlights the occurrence of a normative decline in symptoms during the transition to adulthood. As a result, one way to understand risk for the disorder is to identify behaviors during adolescence that may exacerbate BPD symptoms and potentially interfere with the age-related decline in symptoms. Thus, focusing on this developmental stage in adolescent girls may help elucidate factors that lead to the escalation and persistence of BPD symptoms.

One particularly problematic correlate of BPD symptoms is elevated rates of alcohol use. Adolescence is also a time when alcohol use emerges and increases in frequency, with 10% of 8th graders and 35% of 12th graders reporting alcohol use in the past 30 days (Johnston, O'Malley, Miech, Bachman, & Schulenberg, 2016). While alcohol use is problematic for adolescents in general because of its association with risk-taking behaviors, emotional distress, and later alcohol, mood, and personality disorders (Colder, Campbell, Ruel, Richardson, & Flay, 2002; Rohde et al., 2001), it may be especially harmful in the context of BPD symptoms. This is worrisome given the frequent co-occurrence of BPD symptoms and alcohol use. Specifically, BPD during adolescence is associated with elevated symptoms of alcohol abuse (Cohen et al., 2007) and adolescents with BPD diagnosis have higher rates of binge drinking compared to those without the diagnosis (Serman, Johnson, Geller, Kanost, & Zacharapoulou, 2002). Finally, there is evidence of a longitudinal association such that adolescent alcohol use disorder predicts elevated levels of BPD symptoms in adulthood (Rohde et al., 2001; Thatcher et al., 2005). Together, this research points to alcohol use as a behavior that may serve to ensnare adolescents in a context that is ripe for the development of BPD.

First, alcohol use may set the stage for the development of BPD symptoms by exacerbating or evoking a multitude of BPD-related problems. Specifically, alcohol use is related to impulsive and risk-taking behavior (DuRant, Smith, Kreiter, & Krowchuk, 1999; Farley & Kim-Spoon, 2015; Tapert, Aarons, Sedlar, & Brown, 2001), elevations in negative affect (Hussong, Gould, & Hersh, 2008), and suicidality (Galaif, Sussman, Newcomb, & Locke, 2007; Schilling, Aseltine, Glanovsky, James, & Jacobs, 2009), all of which characterize BPD. Consistent with theories of the development of BPD, behaviors that are learned and reinforced during this time may be especially problematic for those who are emotionally sensitive (Linehan, 1993). For example, involvement in deviant peer groups over time may decrease opportunities for the modeling of effective emotional and behavioral regulation (Allen, Porter, McFarland, Marsh, & McElhaney, 2005; Simons-Morton & Chen, 2006) while increasing opportunities for victimization and exposure to trauma (Parker, Debnam, Pas, & Bradshaw, 2015). Similarly, the use of alcohol may reinforce maladaptive means of coping with aversive emotions and increase risk for suicide attempts, especially when drinking occurs in the context of emotional distress (Schilling et al., 2009). Finally, adolescence is a time of rapid neurological growth and activity in regions affecting emotion, reinforcement, and reactivity, which also underlie BPD, and may be impacted by substance use during this period (Chambers, Taylor, & Potenza, 2003; Squeglia et al., 2015; Steinberg, 2010). Thus, adolescence represents a critical window for potential interventions; however, a better understanding of the concurrent and predictive associations between alcohol and BPD symptoms during this sensitive time is required.

One barrier to understanding how alcohol use may affect BPD symptoms is the focus of the existing research on between-individual differences and rank-order stability that may obscure meaningful changes in alcohol use that occur within individuals over time (i.e., does fluctuation in alcohol use within an individual predict increases in that person's BPD symptoms). An added benefit of focusing on change occurring within individuals is that all stable and pre-existing potential confounding variables are controlled for, eliminating many potential third variables explaining this relationship. This is an especially useful technique

for examining the highly comorbid problems, such as BPD and substance use, which are sometimes thought to be a consequence of common risk factors (Bornova, Hicks, Iacono, & McGue, 2013). For example, genetic predisposition or family mental health history might explain why some individuals are more likely than others to initially engage alcohol use or develop BPD symptoms, but these are time-invariant factors and thus, cannot explain why BPD symptoms might fluctuate in unison with alcohol use from year-to-year. Thus, this type of analysis directly contributes to our understanding of how shifts in alcohol use frequency during adolescence track with BPD symptoms both concurrently and prospectively while accounting for all pre-existing time-stable factors that may explain the overlap between alcohol use and BPD symptoms.

The present study builds upon prior work by capitalizing on repeated measurement of alcohol use and BPD symptoms in a large sample of adolescent girls to examine how changes in alcohol use frequency are associated with concurrent and future BPD symptoms. Furthermore, our analytic strategy overcomes many shortcomings of the existing research and increases the specificity of our conclusions by simultaneously controlling for the time-varying impact of symptoms of both internalizing and externalizing disorders, prior and concurrent other substance use, and all time-invariant, pre-existing factors that may explain differences between individuals. The inclusion of these time-varying covariates can increase our confidence that any increases in BPD symptoms are not due to changes in psychopathology more generally, and can be attributed to changes in alcohol use frequency, rather than other substances with which alcohol is highly correlated (e.g., marijuana, or tobacco use). By including both concurrent and year-prior levels of our predictor variable, we are able to examine whether alcohol use has a lasting or sustained effect on BPD symptoms, over and above the effect of concurrent alcohol use. The diverse community sample presents considerable variability in socioeconomic status (SES) and race, which are often neglected in clinical studies but have implications for more culturally informed theory and practice. Finally, it is important to acknowledge the possibility of reverse causation. While not focusing exclusively on adolescence, several studies do find evidence in support of the opposite path (Cohen et al., 2007; Stepp et al., 2005; Trull et al., 2004). Given this possibility, we controlled for prior BPD symptoms in all models and also examined the reverse direction: whether BPD symptoms predicted alcohol use in the same year as well as the following year.

Therefore, the primary hypothesis for the current study was that year-to-year fluctuations in alcohol use frequency would predict concurrent and prospective increases in BPD symptoms. We also tested the opposite pathway to examine whether the associations observed in the primary models were entirely due to reverse causation. Finally, in order to fully consider the diverse study sample, and because previous studies have demonstrated associations between socioeconomic factors such as low SES and BPD (Stepp, Keenan, Hipwell, & Krueger, 2014a), we also explored whether race, low SES, or age moderated the relationship between year-to-year fluctuations in alcohol use and increases in BPD symptoms.

Method

Participants and Procedures

Data for the present study were obtained from a large, urban-dwelling sample of girls (Pittsburgh Girls Study; PGS). Informed consent was obtained from all individual participants included in the study. All procedures performed in the study were in accordance with the ethical standards of the University of Pittsburgh Institutional Review Board. In 1998–1999, 103,238 households in Pittsburgh neighborhoods were identified. Households with a girl between the ages of 5 and 8 years were invited to participate in a longitudinal study. In order to oversample low-income families, all households in neighborhoods where at least 25% of families were living at or below poverty were screened for eligibility, and 50% of households in the remaining neighborhoods were screened. Girls were grouped into four age-based cohorts (cohorts 5, 6, 7, and 8 included girls who were recruited at ages 5, 6, 7, and 8, respectively). The final sample included 2,540 girls and their primary caregivers (82% of target population). The sample was racially diverse (41.25% White; 58.75% Black) and approximately 39% of the sample was receiving public assistance (i.e., food stamps, supplemental nutrition program or women, infants, and children, welfare) at study initiation. Girls and primary caregivers participated in an initial interview immediately after study enrollment, as well as a series of annual follow-up interviews. The last annual follow-up was conducted when girls were between 19 and 22 years old.¹ Analyses presented here utilized the girls' self-reported assessments from age 14 to 17, when both BPD symptoms and alcohol use were assessed. Sample retention for the assessments used in the present study ranged from 85.7% to 90.1%. Girls who missed any of the four measurement occasions used in the present study ($N = 542$) were more likely to be White ($OR = 1.40$; $p < .01$) and less likely to have been receiving public assistance at study initiation ($OR = 0.70$, $p = .01$) than girls with complete data. The cohorts did not differ in likelihood of having any missing data ($\chi^2 = 3$; $p = .87$). All primary caregivers provided informed consent and girls provided assent prior to study enrollment (Keenan et al., 2010).

Measures

BPD symptoms—During annual interviews beginning at age 14, girls self-reported the presence of BPD symptoms using the International Personality Disorder Examinations-Screen (IPDE-S; Lenzenweger, Loranger, Korfine, & Neff, 1997). The IPDE-BOR is a nine-item questionnaire which assesses the primary *DSM-IV* diagnostic criteria of BPD (American Psychiatric Association, 2000). Sample items include: “I’m very moody” and “I often feel empty inside.” Girls responded to each item by reporting whether they experienced each symptom during the most recent year (0 = *false*; 1 = *true*). One item: “I show my feelings for all to see,” was excluded because of relatively high base rates (43% to 49% at each age; 76% of girls endorsed at least once during the study) and low point-biserial correlations with the total BPD score (r s from .01 to .03). Additionally, an item reflecting the core BPD symptom of identity disturbance, “The way I feel about myself changes a lot,” was created for this study in year nine (when girls were between 13 and 16). Because this item was added after study initiation, it had planned missingness at ages 14 and 15 for two

¹Data collection is ongoing.

cohorts (i.e., cohorts 7 and 8). All nine items were summed together to create a total score that ranged from 0 to 9. The scale demonstrated acceptable internal consistency at each age ($\alpha = .71 - .72$). The mean number of total BPD symptoms ranged from 2.03 to 2.24 at each age. The intraclass correlation coefficient for BPD symptom total across adolescence was .55, indicating variability in the presence of symptoms from year to year (see Table 1). Chanen et al. (2008) demonstrated adequate sensitivity and specificity of BPD symptoms on the IPDE-BOR to BPD diagnosis in a sample of youth.

Furthermore, a clinical cut-off of greater than six symptoms has been suggested for a BPD diagnosis (Chanen et al., 2008) and a score of greater than three symptoms may be indicative of clinically significant impairment/distress (Smith, Muir, & Blackwood, 2005; Stepp, Burke, Hipwell, & Loeber, 2012). In the present sample, 9.61% ($n = 214$) of girls reported seven or more symptoms of BPD at some point during the study. Of the girls reporting seven or more symptoms during the study period, only 1.40% continued to report symptoms at this level of severity or greater at all four assessments ($M = 1.35$ years, $SD = 0.64$). Additionally, 43.94% ($n = 979$) of girls reported four or more symptoms of BPD at least once during the study period. Of the girls who reported four or more symptoms at least once, 11.24% continued to report symptoms at this level of severity or greater at all four assessments ($M = 2.00$ years; $SD = 1.03$). See Table 1 for additional descriptive statistics regarding BPD symptoms.

Alcohol use—Girls self-reported frequency of past-year alcohol use using items from The Nicotine, Alcohol, and Drug Substance Use measure (Pandina, Labouvie, & White, 1984). At each annual assessment, girls were asked to state how often they had consumed beer, wine, and liquor using an 8-point scale that ranged from 0 (*no use within past year*) to 7 (*used every day or more than once per day*). Because each type of liquor was asked in its own item and approximately 32% of girls reported using more than one type of alcohol per year during the study period, we used the maximum frequency across the three types (see Hansell & White, 1991). More than half (55.86%) of the sample used alcohol during the study period. Additionally, 8.17% of girls reported a maximum of monthly use, 9.03% reported a maximum of weekly use, and 0.80% reported a maximum of near daily or daily use. See Table 1 for additional descriptive statistics regarding alcohol use. Past year rates of alcohol use in PGS are generally consistent with rates found in national surveys (Johnston et al., 2016).

Time-varying covariates—Symptoms of depression and conduct problems were assessed using the Adolescent Symptom Inventory-4 (Gadow & Sprafkin, 1998). Fourteen items assessed conduct problems (e.g., “In the past year, how often have you started physical fights?”) and 11 items assessed symptoms of depression (e.g., “In the past year, have you been depressed for most of the day?”). Girls responded to the 25 items using a 4-point scale (0 = *never* to 3 = *often*). The items from each of the two scales were summed to reflect severity of conduct problems and symptoms of depression. The ASI-4 depression and conduct problems scales have demonstrated adequate concurrent validity, sensitivity, and specificity with clinician-rated diagnoses (Gadow & Sprafkin, 1998). Cronbach’s alphas for the depression and conduct scales were greater than .72 and .79, respectively, at all

measurement occasions used in the present analysis. Co-occurring concurrent and prior (T-1) tobacco and marijuana use were also assessed with the previously described Nicotine, Alcohol, and Drug Substance Use measure (Pandina et al., 1984). Because tobacco use was bimodal, it was coded as a binary variable to index whether girls were daily or near-daily tobacco smokers in the current year (concurrent tobacco use) and whether they were near-daily/daily smokers in the prior year (T-1; 0 = *not daily or near daily smoker*, 1 = *daily or near-daily smoker*). Concurrent and prior (T-1) marijuana use was coded using the same scale that was used to measure alcohol use (eight-point scale that ranged from 0 = *no use within the past year* to 7 = *used every day or more than once per day*). Prior BPD symptoms were the symptom count of BPD symptoms in the year prior (T-1). Age and age² were also included as time-varying covariates in all models to account for developmental change in the outcome variables. See Table 1 for descriptive statistics regarding the covariates and Table 2 for correlations.

Demographic moderators—Age (both linear and non-linear), race (coded 1 = *Black or African American*, 0 = *White or Caucasian*), and low SES were examined as potential moderators. We used the receipt of public assistance as a marker of low SES (0 = *not receiving public assistance*; 1 = *receiving public assistance*). Because low SES could change from year to year, we examined interactions with both a time-invariant version (poverty status at analysis Time 1—age 14), and a time-varying version (annual indicator of low SES during each year of the study period).

Analytic Strategy

Fixed effects (within-individual) Poisson regressions were used to examine the extent to which BPD symptoms fluctuated based on concurrent and prior alcohol use. Because these models focus exclusively on change within individuals, fixed effects regressions effectively use individuals as their own “controls” by essentially including a series of dummy coded variables (less 1) to represent each participant. This enables the estimation of the effects of time-varying variables (e.g., alcohol use) while accounting for all unchanging characteristics of the individual—regardless of whether the time-invariant characteristics are measured or not (Allison, 2009). Only confounding variables that change year-to-year need to be directly modeled in the analysis.

We first examined the effects of concurrent and prior alcohol use on BPD symptoms after controlling for age (linear and quadratic) and prior BPD symptoms only. Second, the remaining time-varying covariates were added to the model to control for potential confounds (i.e., concurrent symptoms of conduct disorder and depression; concurrent and prior marijuana use, concurrent and prior tobacco use).² Furthermore, in order to assess whether the associations observed in our primary model varied as a function of demographic factors, we included interactions between current and prior alcohol use with race, low SES (both at age 14 and annual status), and age in separate models (after adjusting for all of the previously described time-varying confounding variables).

²We also examined the time-varying effect of annual low SES for the adolescent, but this variable was not associated with either outcome and its inclusion did not change the pattern of results. As such, for the sake of model parsimony, low SES was not included in the final models.

Finally, the reverse model was tested by examining the effects of concurrent and prior BPD symptoms on alcohol use frequency in reduced models (only controlling for age and prior alcohol use) as well as fully-adjusted models (controlling for all time-varying covariates: age, prior alcohol use, concurrent conduct problems, concurrent depressive symptoms; concurrent and prior marijuana use, and concurrent and prior tobacco use).

Results

Missing data

Nearly 78% of the initial sample provided data all four assessments used in the present analyses. Approximately 7% of girls were missing one year of data, approximately 3% were missing 2 years of data, approximately 2% were missing three years of data, and approximately 9% of the initial sample was missing all four years of data (i.e., over 90% of initial sample provided data between ages 14 and 17). Girls who missed any of the four annual assessments ($N = 542$) did not differ from those with all years of data ($N = 1908$) on total BPD symptoms, depressive symptoms, or tobacco use at any age (all p s $> .10$). Girls with any missing data reported slightly more alcohol and marijuana use at age 14 (OR = 1.19; $p = .02$; OR = 1.11; $p = .04$, respectively) and conduct problems at ages 15 and 17 (OR = 1.09, $p < .01$; OR = 1.09, $p = .17$, respectively). Models were estimated with maximum likelihood estimation, which utilized all available data to generate parameters.

BPD Symptoms as a Function of Alcohol Use Frequency

Prior to covariate adjustment, both concurrent and prior alcohol use were associated with increases in BPD symptoms (see Table 3). Specifically, for every 1-point increase in alcohol use, an adolescent girl's BPD symptoms systematically increased by 5% in the same year and her symptoms remained elevated by 3% in the following year (p s $< .05$). When concurrent and prior marijuana use, concurrent and prior tobacco use, concurrent depressive symptoms, and concurrent conduct problems were included in the analysis, the effect of prior alcohol use remained significant but the effect of concurrent alcohol use did not (see Table 3). Increases in concurrent depressive symptoms and conduct problems were also associated with increases in BPD symptoms (see Table 3).

None of the interactions between concurrent and prior alcohol use with race, low SES (both at age 14 and annual status), and age were significant (all p s $> .05$; more information available from the authors).

Alcohol Use as a Function of BPD Symptoms

In both the reduced and the fully adjusted models, increases in BPD symptoms were associated with simultaneous increases in concurrent alcohol use, although the lagged or sustained effect of BPD symptoms was not significant in either model (see Table 4). Increases in concurrent marijuana use, prior marijuana use, and concurrent conduct problems use were also associated with increases in alcohol use.

Discussion

Using annual assessments from a large, diverse sample of adolescent girls, the current study sought to examine the extent to which BPD symptoms varied as a function of concurrent and prior alcohol use—net of other substance use and co-morbid internalizing and externalizing symptoms— during adolescence. We found that within-person increases in alcohol use frequency prospectively predicted increases in girls' BPD symptoms, suggesting that alcohol use has a lasting impact on the development of BPD symptoms. In other words, even when accounting for girls concurrent alcohol use, their alcohol use in the previous year continued to have an effect on their concurrent BPD symptoms. The enduring nature of this effect suggests that there is something insidious about alcohol use, where the complete impact of this behavior on BPD symptoms may not be immediately apparent. Importantly, the reverse model indicated that the sustained nature of the effect was not bidirectional. Within-person increases in BPD symptoms were only concurrently related to elevations in alcohol use frequency (BPD symptoms in the prior year were not associated with girls' alcohol use). Together, these results suggest that beyond their tendency to be mutually elevated, alcohol use may place adolescents at risk for future increases in their BPD symptoms.

The findings from the present study help clarify and extend the existing literature on BPD and alcohol use in adolescence. For example, in another community-based study, Rohde and colleagues (2001) found that adolescent AUD predicted a diagnosis of BPD in young adulthood. In fact 13% of adolescents diagnosed with an AUD at baseline had a BPD diagnosis when assessed during young adulthood. This number stands in contrast to approximately 2.7% of adults diagnosed with the BPD in the general population (Tomko, Trull, Wood, & Sher, 2014). In a sample recruited from clinical treatment resources, Thatcher and colleagues (2005) found consistent results, indicating that adolescent AUDs prospectively predicted severity of adult BPD. These studies suggest that alcohol use during adolescence may be an important indicator of risk for later BPD. The present study expands upon these findings by showing that in a high-risk community sample of female youth, this process operates at the level of the individual and across a range of severity of alcohol use and BPD symptoms. This means that even among those without an AUD, increases in frequency of alcohol use may be a clinically noteworthy indicator of increased risk for the development of BPD symptoms.

The lasting influence of alcohol use on BPD symptoms may occur through various pathways. One possibility is that alcohol use leads to the reinforcement of emotional avoidance in a manner that fuels the development of BPD symptoms (Cooper, Frone, Russell, & Mudar, 1995; Sinha et al., 2008). In other words, adolescents may experience relief from negative affective states while using alcohol, which not only reinforces the use of alcohol, but also increases the likelihood of engaging in this ineffective means of emotion regulation through other strategies. Avoidance of painful emotions may take other forms, including non-suicidal self-injury, impulsive behavior, or dissociation. This process may interfere with adolescents' comfort experiencing and understanding their emotions, and lead to a failure to develop effective emotion regulation capacities and a cohesive sense of self. Alternatively (or in conjunction with this), increases in alcohol use frequency may leave adolescents more vulnerable to experiences that can exacerbate BPD symptoms over time,

such as victimization by dating partners (Parker et al., 2015) while also potentially representing a transition to new and more deviant peers, and subsequent exposure to the effects of more substance using friends (Farley & Kim-Spoon, 2015; Simons-Morton & Chen, 2006). Finally, our moderation analyses indicated that the effect of girls' alcohol use on in BPD symptoms did not depend of her race, low SES, or age. Future research should examine other person-specific factors that might identify individuals for whom the associations presented here are magnified.

The inclusion of the reverse model helps contextualize these findings by clarifying the directionality of the association between BPD symptoms and alcohol use during adolescence. Specifically, the "reverse model" indicated that during years when a girl experiences an increase in her BPD symptoms, she is also likely to concurrently report elevated rates of alcohol use. While this relationship is not a prospective one, it does have implications for understanding alcohol use in the context of BPD symptoms. First, elevated alcohol rates of alcohol use may be a reflection of the BPD symptom of impulsivity. Excessive consumption of alcohol and other risky behaviors, such as driving while intoxicated, are part of the impulsivity criterion for BPD. Alternatively, this association is consistent with self-medication theories of alcohol use and may suggest that increases in alcohol use during years of elevated BPD symptoms reflect a maladaptive effort at dealing with BPD-related emotional distress (Gould, Hussong, & Hersh, 2012; Jahng et al., 2011). This hypothesis also fits with research using experience sampling methodology to examine the co-variation of alcohol use with mood, which has found that certain subgroups of adolescents (Hersh & Hussong, 2009; Hussong et al., 2008; Reimuller, Shadur, & Hussong, 2011) as well as those who experience more anger (Gould et al., 2012), and higher levels of within-individual variability in affect (Gottfredson & Hussong, 2013) tend to show greater evidence of self-medication. Thus, this association in our analyses may reflect a means of coping with distress related to spikes in BPD symptoms, rather than a lasting effect of BPD symptoms that continues to affect alcohol use to the following year.

This study highlights some of the strengths of using within-individual approaches to the examination of developmental processes (Allison, 2009). First, to strengthen causal inference the analyses were designed to rule out all time-invariant and pre-existing factors as possible confounds, in addition to the time-varying factors included in the model. This means that the association between alcohol use and BPD symptoms cannot be explained by other factors that are typically difficult to account for, such as genetics or early life experiences. Furthermore, relevant time-varying symptoms and behaviors were accounted for in the models, including concurrent levels of depressive symptoms and conduct disorder symptoms, both prior and concurrent levels of tobacco and marijuana use, as well as prior levels of BPD. Their inclusion in our models can increase our confidence in the specificity of these findings to alcohol use, rather than these other common comorbidities or substance use more generally.

The current study is not without limitations. First, the study was limited to self-reported symptoms by adolescent girls, and our findings can only speak to the relations among BPD symptoms and alcohol use in this population. It is important to examine whether boys show similar increases in BPD symptoms in the context of increases in alcohol use frequency. In

addition, the current study assessed symptoms of BPD in a community sample rather than treatment seeking individuals or those diagnosed with the disorder. While studying community samples has utility for understanding broader targets for prevention and early intervention, the low mean level of BPD symptoms present throughout the study (approximately two), makes it difficult to generalize these findings to those with a diagnosis of BPD. However, approximately 9% of girls reported seven or greater symptoms of BPD during the course of the study and even low levels of BPD severity are related to poor psychiatric outcomes and impairment (Zimmerman et al., 2011). Also, given that girls with missing data were more likely to be White and not receiving public assistance, our data may reflect an even more high-risk sample than originally intended. Finally, the relationship between BPD and alcohol use is a complex one that remains present (Trull et al., 2000) and related to poor treatment and symptom outcomes (Bornovalova & Daughters, 2007; Hasin, 2011; Linehan et al., 1999; Links, Heslegrave, Mitton, Van Reekum, & Patrick, 1995; Martínez-Raga, Marshall, Keaney, Ball, & Strang, 2002; van den Bosch, Verheul, Schippers, & van den Brink, 2002; Yen et al., 2003) into adulthood. It is important to note that the direction of this association may not remain constant across the lifespan. In fact, research by Bornovalova and colleagues (2013) showed that the association between BPD and substance use was significantly weaker at age 18 compared to age 14. Future research should expand this work to understand how this process unfolds during young adulthood.

Furthermore, while within-individual effects were found using the current study design, it would be useful to capture these processes as they occur in real-life. As mentioned earlier, using an experience sampling or event-contingent recording design may provide a more fine-grained picture of the relation between alcohol use and processes that may ultimately lead to the worsening of BPD symptoms over time. Such a design could test potential mechanisms by which alcohol use affects the development of BPD symptoms, such as reinforcement of emotional avoidance, in real time. While work like this exists using healthy adolescent populations (Gould et al., 2012; Hussong et al., 2008) and in adults with BPD (Jahng et al., 2011), it should be extended to adolescents with BPD symptoms.

This research suggests directions for future research on interventions designed to reduce the burden of BPD in adolescence. For example, among those displaying early signs and symptoms of the disorder, interventions that focus on preventing or reducing alcohol use may help mitigate the risk of developing the BPD. While the current study did not find that increases in BPD symptoms have a lasting effect on alcohol use, it does shed light on a potentially dangerous cycle where increases in BPD symptoms are accompanied by elevated rates of alcohol use, which then set the adolescent up for future increases in BPD symptoms. Thus, while we did not test why alcohol use is elevated in years when girls experience increases in BPD symptoms, research that does this may help refine such interventions by allowing clinicians to focus on deterring alcohol use and disrupting this cycle. Finally, existing interventions may benefit from more explicitly highlighting the risks associated with alcohol use for future mental health outcomes, especially in those already displaying symptoms of BPD.

Together, our findings support the presence of a relationship between within-individual changes in alcohol use and the concurrent and subsequent escalation of BPD symptoms and

represent a step towards delineating their pattern of co-development during adolescence. Capturing these symptoms during adolescence is a significant strength of the current study, as it may represent a critical period for the development of BPD, when symptoms of the disorder are particularly malleable (Lenzenweger & Castro, 2005). Specifically, while symptoms of BPD are shown to decrease during the transition to adulthood, it is important to work towards identifying the subgroup of individuals for whom this does not occur. Finally, research that examines this process at the level of the individual can complement our knowledge of how this association plays out across individuals, to help hone interventions and treatment for BPD.

Acknowledgments

This research was supported by grants from the National Institute of Mental Health (MH056639) and the National Institute on Drug Abuse (DA012237). Dr. Pedersen's effort was supported by K01AA0211535.

References

- Allen JP, Porter MR, McFarland FC, Marsh P, McElhaney KB. The two faces of adolescents' success with peers: Adolescent popularity, social adaptation, and deviant behavior. *Child Development*. 2005; 76:747–760. <http://doi.org/10.1111/j.1467-8624.2005.00875.x>. [PubMed: 15892790]
- Allison, PD. *Fixed Effects Regression Models*. Thousand Oaks, CA: SAGE Publications; 2009.
- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4. Washington, DC: Author; 2000. (text rev.)
- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5. Washington, DC: Author; 2013.
- Beauchaine TP, Klein DN, Crowell SE, Derbidge C, Gatzke-Kopp L. Multifinality in the development of personality disorders: A Biology $\times$ Sex $\times$ Environment interaction model of antisocial and borderline traits. *Development and Psychopathology*. 2009; 21:735–36. <http://doi.org/10.1017/S0954579409000418>. [PubMed: 19583882]
- Bornoalova MA, Daughters SB. How does Dialectical Behavior Therapy facilitate treatment retention among individuals with comorbid borderline personality disorder and substance use disorders? *Clinical Psychology Review*. 2007; 27:923–943. <http://doi.org/10.1016/j.cpr.2007.01.013>. [PubMed: 17376574]
- Bornoalova MA, Hicks BM, Iacono WG, McGue M. Longitudinal twin study of borderline personality disorder traits and substance use in adolescence: Developmental change, reciprocal effects, and genetic and environmental influences. *Personality Disorders*. 2013; 4:23–32. <http://doi.org/10.1037/a0027178>. [PubMed: 22642461]
- Bornoalova MA, Hicks BM, Patrick CJ, Iacono WG, McGue M. Development and validation of the Minnesota borderline personality disorder scale. *Assessment*. 2011; 18:234–252. <http://doi.org/10.1177/1073191111398320>. [PubMed: 21467094]
- Chambers RA, Taylor JR, Potenza MN. Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *The American Journal of Psychiatry*. 2003; 160:1041–1052. <http://doi.org/10.1176/appi.ajp.160.6.1041>. [PubMed: 12777258]
- Chanen AM, Jackson HJ, McGorry PD, Allot KA, Clarkson V, Yuen HP. Two-year stability of personality disorder in older adolescent outpatients. *Journal of Personality Disorders*. 2004; 18:526–541. <http://doi.org/10.1521/pedi.18.6.526.54798>. [PubMed: 15615665]
- Chanen AM, Jovev M, Djaja D, McDougall E, Yuen HP, Rawlings D, Jackson HJ. Screening for borderline personality disorder in outpatient youth. *Journal of Personality Disorders*. 2008; 22:353–364. <http://doi.org/10.1521/pedi.2008.22.4.353>.
- Chanen AM, Kaess M. Developmental pathways to borderline personality disorder. *Current Psychiatry Reports*. 2011; 14:45–53. <http://doi.org/10.1007/s11920-011-0242-y>.

- Cohen PR, Chen H, Crawford TN, Brook JS, Gordon K. Personality disorders in early adolescence and the development of later substance use disorders in the general population. *Drug and Alcohol Dependence*. 2007; 88(Suppl 1):S71–84. <http://doi.org/10.1016/j.drugalcdep.2006.12.012>. [PubMed: 17227697]
- Colder CR, Campbell RT, Ruel E, Richardson JL, Flay BR. A finite mixture model of growth trajectories of adolescent alcohol use: Predictors and consequences. *Journal of Consulting and Clinical Psychology*. 2002; 70:976–985. <http://doi.org/10.1037//0022-006X.70.4.976>. [PubMed: 12182281]
- Cooper ML, Frone MR, Russell M, Mudar P. Drinking to regulate positive and negative emotions: A motivational model of alcohol use. *Journal of Personality and Social Psychology*. 1995; 69:990–1005. <http://doi.org/10.1037/0022-3514.69.5.990>. [PubMed: 7473043]
- DuRant RH, Smith JA, Kreiter SR, Krowchuk DP. The relationship between early age of onset of initial substance use and engaging in multiple health risk behaviors among young adolescents. *Archives of Pediatrics & Adolescent Medicine*. 1999; 153:286–291. <http://doi.org/10.1001/archpedi.153.3.286>. [PubMed: 10086407]
- Eppright TD, Kashani JH, Robison BD, Reid JC. Comorbidity of conduct disorder and personality disorders in an incarcerated juvenile population. *The American Journal of Psychiatry*. 1993; 150:1233–1236. <http://doi.org/10.1176/ajp.150.8.1233>. [PubMed: 8328569]
- Farley JP, Kim-Spoon J. Longitudinal associations among impulsivity, friend substance use, and adolescent substance use. *Journal of Addiction Research & Therapy*. 2015; 6 <http://doi.org/10.4172/2155-6105.1000220>.
- Gadow, KD., Sprafkin, J. Adolescent symptom inventory-4 norms manual. Stonybrook, NY: Checkmate Plus; 1998.
- Galaif ER, Sussman S, Newcomb MD, Locke TF. Suicidality, depression, and alcohol use among adolescents: A review of empirical findings. *International Journal of Adolescent Medicine and Health*. 2007; 19:27–35. <http://dx.doi.org/10.1515/IJAMH.2007.19.1.27>. [PubMed: 17458321]
- Gottfredson NC, Hussong AM. Drinking to dampen affect variability: Findings from a college student sample. *Journal of Studies on Alcohol and Drugs*. 2013; 74:576–583. <http://dx.doi.org/10.15288/jsad.2013.74.576>. [PubMed: 23739021]
- Gould LF, Hussong AM, Hersh MA. Emotional distress may increase risk for self-medication and lower risk for mood-related drinking consequences in adolescents. *The International Journal of Emotional Education*. 2012; 4:6–24. [PubMed: 24358457]
- Grant BF, Chou SP, Goldstein RB, Huang B, Stinson FS, Saha TD, Ruan WJ. Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: Results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *The Journal of Clinical Psychiatry*. 2008; 69:533–545. <http://dx.doi.org/10.4088/JCP.v69n0404>. [PubMed: 18426259]
- Grilo CM, Beeker DF, Fehon DC, Walker ML, Edell WS, McGlashan TH. Gender differences in personality disorders in psychiatrically hospitalized adolescents. *American Journal of Psychiatry*. 1996; 153:1089–1091. [PubMed: 8678180]
- Hansell S, White HR. Adolescent drug use, psychological distress, and physical symptoms. *Journal of Health and Social Behavior*. 1991; 32:288–301. <http://dx.doi.org/10.2307/2136809>. [PubMed: 1940211]
- Hasin DS. Personality disorders and the 3-year course of alcohol, drug, and nicotine use disorders. *Archives of General Psychiatry*. 2011; 68:1158–19. <http://doi.org/10.1001/archgenpsychiatry.2011.136>. [PubMed: 22065531]
- Hersh MA, Hussong AM. The association between observed parental emotion socialization and adolescent self-medication. *Journal of Abnormal Child Psychology*. 2009; 37:493–506. <http://doi.org/10.1007/s10802-008-9291-z>. [PubMed: 19137423]
- Hussong AM, Gould LF, Hersh MA. Conduct problems moderate self-medication and mood-related drinking consequences in adolescents. *Journal of Studies on Alcohol and Drugs*. 2008; 69:296–307. <http://dx.doi.org/10.15288/jsad.2008.69.296>. [PubMed: 18299772]
- Jahng S, Solhan MB, Tomko RL, Wood PK, Piasecki TM, Trull TJ. Affect and alcohol use: An ecological momentary assessment study of outpatients with borderline personality disorder.

- Journal of Abnormal Psychology. 2011; 120:572–584. <http://doi.org/10.1037/a0024686>. [PubMed: 21823761]
- Johnson JG, Cohen PR, Kasen S, Skodol AE, Hamagami F, Brook JS. Age-related change in personality disorder trait levels between early adolescence and adulthood: a community-based longitudinal investigation. *Acta Psychiatrica Scandinavica*. 2000; 102:265–275. <http://doi.org/10.1034/j.1600-0447.2000.102004265.x>. [PubMed: 11089726]
- Johnston, LD., O'Malley, PM., Miech, RA., Bachman, JG., Schulenberg, JE. Monitoring the Future national survey results on drug use, 1975–2015: Overview, key findings on adolescent drug use. Ann Arbor: Institute for Social Research, The University of Michigan; 2016.
- Keenan K, Hipwell AE, Chung T, Stepp SD, Stouthamer-Loeber M, Loeber R, McTigue K. The Pittsburgh Girls Study: Overview and initial findings. *Journal of Clinical Child & Adolescent Psychology*. 2010; 39:506–521. <http://doi.org/10.1080/15374416.2010.486320>. [PubMed: 20589562]
- Korzekwa MI, Dell PF, Links PS, Thabane L, Webb SP. Estimating the prevalence of borderline personality disorder in psychiatric outpatients using a two-phase procedure. *Comprehensive Psychiatry*. 2008; 49:380–386. <http://doi.org/10.1016/j.comppsy.2008.01.007>. [PubMed: 18555059]
- Lenzenweger MF, Castro DD. Predicting change in borderline personality: Using neurobehavioral systems indicators within an individual growth framework. *Developmental Psychopathology*. 2005; 17:1207–1237. <http://doi.org/10.1017/S0954579405050571>.
- Lenzenweger MF, Loranger AW, Korfine L, Neff C. Detecting personality disorders in a nonclinical population. Application of a 2-stage procedure for case identification. *Archives of General Psychiatry*. 1997; 54:345–351. <http://dx.doi.org/10.1001/archpsyc.1997.01830160073010>. [PubMed: 9107151]
- Linehan, MM. Cognitive-behavioral Treatment of Borderline Personality Disorder. New York, NY: Guilford Press; 1993.
- Linehan MM, Schmidt H, Dimeff LA, Craft JC, Kanter J, Comtois KAK. Dialectical Behavior Therapy for patients with borderline personality disorder and drug-dependence. *The American Journal on Addictions*. 1999; 8:279–292. <http://doi.org/10.1080/105504999305686>. [PubMed: 10598211]
- Links PS, Heslegrave RJ, Mitton JE, Van Reekum R, Patrick J. Borderline personality disorder and substance abuse: Consequences of comorbidity. *The Canadian Journal of Psychiatry*. 1995; 40:9–14. [PubMed: 7874683]
- Martínez-Raga J, Marshall EJ, Keaney F, Ball D, Strang J. Unplanned versus planned discharges from in-patient alcohol detoxification: Retrospective analysis of 470 first-episode admissions. *Alcohol and Alcoholism*. 2002; 37:277–281. <http://doi.org/10.1093/alcalc/37.3.277>. [PubMed: 12003918]
- Pandina RJ, Labouvie EW, White HR. Potential contributions of the life span developmental approach to the study of adolescent alcohol and drug use: The Rutgers Health and Human Development Project, a working model. *Journal of Drug Issues*. 1984; 14:253–268. <http://doi.org/10.1177/002204268401400206>.
- Parker EM, Debnam K, Pas ET, Bradshaw CP. Exploring the link between alcohol and marijuana use and teen dating violence victimization among high school students: The influence of school context. *Health Education & Behavior*. 2015; 43:1–9. <http://doi.org/10.1177/1090198115605308>.
- Reimuller A, Shadur J, Hussong AM. Parental social support as a moderator of self-medication in adolescents. *Addictive Behaviors*. 2011; 36:203–208. <http://doi.org/10.1016/j.addbeh.2010.10.006>. [PubMed: 21146938]
- 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 and Adolescent Psychiatry*. 2001; 40:83–90. <http://doi.org/10.1097/00004583-200101000-00020>. [PubMed: 11195569]
- Schilling EA, Aseltine RH, Glanovsky JL, James A, Jacobs D. Adolescent alcohol use, suicidal ideation, and suicide attempts. *The Journal of Adolescent Health : Official Publication of the Society for Adolescent Medicine*. 2009; 44:335–341. <http://doi.org/10.1016/j.jadohealth.2008.08.006>. [PubMed: 19306791]

- Serman N, Johnson JG, Geller PA, Kanost RE, Zacharapoulou H. Personality disorders associated with substance use among American and Greek adolescents. *Adolescence*. 2002; 37:841–854. [PubMed: 12564834]
- Simons-Morton B, Chen RS. Over time relationships between early adolescent and peer substance use. *Addictive Behaviors*. 2006; 31:1211–1223. <http://doi.org/10.1016/j.addbeh.2005.09.006>. [PubMed: 16229958]
- Sinha R, Fox HC, Hong KA, Bergquist K, Bhagwagar Z, Siedlarz KM. Enhanced negative emotion and alcohol craving, and altered physiological responses following stress and cue exposure in alcohol dependent individuals. *Neuropsychopharmacology*. 2008; 34:1198–1208. <http://doi.org/10.1038/npp.2008.78>. [PubMed: 18563062]
- Smith DJ, Muir WJ, Blackwood DH. Borderline personality disorder characteristics in young adults with recurrent mood disorders: A comparison of bipolar and unipolar depression. *Journal of Affective Disorders*. 2005; 87:17–23. <http://doi.org/10.1016/j.jad.2005.02.019>. [PubMed: 15967232]
- Squeglia LM, Tapert SF, Sullivan EV, Jacobus J, Meloy MJ, Rohlfing T, Pfefferbaum A. Brain development in heavy-drinking adolescents. *The American Journal of Psychiatry*. 2015; 172:531–542. <http://doi.org/10.1176/appi.ajp.2015.14101249>. [PubMed: 25982660]
- Steinberg L. A behavioral scientist looks at the science of adolescent brain development. *Brain and Cognition*. 2010; 72:160–164. <http://doi.org/10.1016/j.bandc.2009.11.003>. [PubMed: 19963311]
- Stapp SD, Burke JD, Hipwell AE, Loeber R. Trajectories of attention deficit hyperactivity disorder and oppositional defiant disorder symptoms as precursors of borderline personality disorder symptoms in adolescent girls. *Journal of Abnormal Child Psychology*. 2012; 40:7–20. <http://doi.org/10.1007/s10802-011-9530-6>. [PubMed: 21671009]
- Stapp SD, Keenan K, Hipwell AE, Krueger RF. The impact of childhood temperament on the development of borderline personality disorder symptoms over the course of adolescence. *Borderline Personality Disorder and Emotion Dysregulation*. 2014a; 1:18–10. <http://doi.org/10.1186/2051-6673-1-18>. [PubMed: 26064524]
- Stapp SD, Trull TJ, Sher KJ. Borderline personality features predict alcohol use problems. *Journal of Personality Disorders*. 2005; 19:711–722. <http://doi.org/10.1521/pedi.2005.19.6.711>. [PubMed: 16553564]
- Stapp SD, Whalen DJ, Scott LN, Zalewski M, Loeber R, Hipwell AE. Reciprocal effects of parenting and borderline personality disorder symptoms in adolescent girls. *Development and Psychopathology*. 2014b; 26:361–378. <http://doi.org/10.1017/S0954579413001041>. [PubMed: 24443951]
- Tapert SF, Aarons GA, Sedlar GR, Brown SA. Adolescent substance use and sexual risk-taking behavior. *The Journal of Adolescent Health : Official Publication of the Society for Adolescent Medicine*. 2001; 28:181–189. [http://dx.doi.org/10.1016/S1054-139X\(00\)00169-5](http://dx.doi.org/10.1016/S1054-139X(00)00169-5). [PubMed: 11226840]
- Thatcher DL, Cornelius JR, Clark DB. Adolescent alcohol use disorders predict adult borderline personality. *Addictive Behaviors*. 2005; 30:1709–1724. <http://doi.org/10.1016/j.addbeh.2005.07.008>. [PubMed: 16095845]
- Tomko RL, Trull TJ, Wood PK, Sher KJ. Characteristics of borderline personality disorder in a community sample: comorbidity, treatment utilization, and general functioning. *Journal of Personality Disorders*. 2014; 28:734–750. http://doi.org/10.1521/pedi_2012_26_093. [PubMed: 25248122]
- Trull TJ, Jahng S, Tomko RL, Wood PK, Sher KJ. Revised NESARC personality disorder diagnoses: Gender, prevalence, and comorbidity with substance dependence disorders. *Journal of Personality Disorders*. 2010; 24:412–426. <http://doi.org/10.1521/pedi.2010.24.4.412>. [PubMed: 20695803]
- Trull TJ, Sher KJ, Minks-Brown C, Durbin J, Burr R. Borderline personality disorder and substance use disorders: a review and integration. *Clinical Psychology Review*. 2000; 20:235–253. [http://doi.org/10.1016/S0272-7358\(99\)00028-8](http://doi.org/10.1016/S0272-7358(99)00028-8). [PubMed: 10721499]
- Trull TJ, Useda D, Conforti K, Doan B-T. Borderline personality disorder features in nonclinical young adults: 2. Two-year outcome. *Journal of Abnormal Psychology*. 1997; 106:307–314. <http://doi.org/10.1037/0021-843X.106.2.307>. [PubMed: 9131850]

- Trull TJ, Waudby CJ, Sher KJ. Alcohol, tobacco, and drug use disorders and personality disorder symptoms. *Experimental and Clinical Psychopharmacology*. 2004; 12:65–75. <http://doi.org/10.1037/1064-1297.12.1.65>. [PubMed: 14769101]
- van den Bosch LMC, Verheul R, Schippers GM, van den Brink W. Dialectical Behavior Therapy of borderline patients with and without substance use problems. *Addictive Behaviors*. 2002; 27:911–923. [http://doi.org/10.1016/S0306-4603\(02\)00293-9](http://doi.org/10.1016/S0306-4603(02)00293-9). [PubMed: 12369475]
- Yen S, Shea T, Pagano M, Sanislow CA, Grilo CM, McGlashan TH, et al. Axis I and Axis II disorders as predictors of prospective suicide attempts: Findings from the Collaborative Longitudinal Personality Disorders Study. *Journal of Abnormal Psychology*. 2003; 112:375–381. <http://doi.org/10.1037/0021-843X.112.3.375>. [PubMed: 12943016]
- Zimmerman M, Chelminski I, Young D. Does the presence of one feature of borderline personality disorder have clinical significance? Implications for dimensional ratings of personality disorders. *The Journal of Clinical Psychiatry*. 2011; 73:8–12. <http://doi.org/10.4088/JCP.10m06784>. [PubMed: 22054015]

Table 1

Descriptive Statistics for Total Borderline Symptoms and Study Variables

	ICC	95% CI	Age 14 <i>M (SD)</i>	Age 15 <i>M (SD)</i>	Age 16 <i>M (SD)</i>	Age 17 <i>M (SD)</i>
Total BPD symptoms	0.55	[0.53, 0.58]	2.11 (1.93)	2.24 (2.00)	2.22 (2.07)	2.03 (2.01)
Depression	0.52	[0.49, 0.54]	7.57 (4.88)	7.25 (4.86)	6.89 (4.84)	6.61 (4.99)
Conduct	0.46	[0.44, 0.49]	1.29 (2.10)	1.42 (2.12)	1.29 (2.07)	1.17 (1.94)
Substance use						
Alcohol use	0.35	[0.32, 0.39]	0.33 (0.80)	0.45 (0.93)	0.60 (1.07)	0.78 (1.22)
Marijuana use	0.40	[0.38, 0.43]	0.31 (1.11)	0.48 (1.34)	0.62 (1.54)	0.79 (1.74)
Tobacco use ^a	0.48	[0.45, 0.50]	0.03 (0.16)	0.05 (0.22)	0.08 (0.26)	0.11 (0.31)

Notes. *N* = 2,149 (age 14); *N* = 2,115 (age 15); *N* = 2,073 (age 16); *N* = 2,055 (age 17);

ICC = intraclass correlation coefficients (average correlations across the 4 time-points within individuals), BPD = borderline personality disorder;

^aBecause tobacco use was a binary indicator of “nearly every day” or more often, the means in the table represent the prevalence (%) of near daily use at each age.

Table 2
Mean Correlations Among Borderline Symptoms and Study Variables from Ages 14–17

Measures	BPD	Conduct	Depression	Alcohol use	Marijuana use	Tobacco use
BPD						
Conduct	0.41					
Range	(0.39–0.42)					
Depression	0.60	0.32				
Range	(0.57–0.61)	(0.30–0.34)				
Alcohol use	0.18	0.37	0.17			
Range	(0.16–0.21)	(0.32–0.40)	(0.14–0.23)			
Marijuana use	0.21	0.43	0.12	0.47		
Range	(0.19–0.24)	(0.38–0.46)	(0.11–0.14)	(0.43–0.52)		
Tobacco use	0.21	0.36	0.14	0.42	0.49	
Range	(0.18–0.26)	(0.29–0.41)	(0.12–0.18)	(0.39–0.45)	(0.43–0.55)	

Notes. BPD = borderline personality disorder. Correlation represents mean correlation from ages 14–17. All correlations were significant at $p < .01$ at all ages.

Table 3

Concurrent and Prior Alcohol Use Predicting Total Borderline Personality Disorder Symptoms

	<u>Reduced Model</u> (<i>N</i> =1,800; obs.=5,316)		<u>Fully Adjusted Model</u> (<i>N</i> =1,800; obs.=5,316)	
	IRR	95% CI	IRR	95% CI
Concurrent alcohol use	1.05 **	[1.03,1.08]	1.02	[0.99,1.05]
Prior year alcohol use	1.03 *	[1.01,1.06]	1.03 *	[1.00,1.06]
Prior year BPD	0.93 **	[0.92,0.94]	0.94 **	[0.93,0.95]
Concurrent tobacco use			1.05	[0.94,1.18]
Prior year tobacco use			1.02	[0.91,1.15]
Concurrent marijuana use			1.02	[1.00,1.03]
Prior year marijuana use			1.00	[0.98,1.03]
Concurrent depressive symptoms			1.04 **	[1.04,1.05]
Concurrent conduct problems			1.04 **	[1.02,1.05]

Notes. IRR = incidence rate ratio; BPD = borderline personality disorder; obs. = observations.

All models estimated with fixed effects Poisson regressions. Linear and quadratic age included in all models. Analytic sample only includes participants who had at least two years of data and at least one year with a non-zero value on the outcome (model default).

* $p < .05$;

** $p < .001$

Table 4

Total Borderline Personality Disorder Symptoms Predicting Alcohol Use

	<u>Reduced Model</u> (<i>N</i> =1,072; obs.=3,174)		<u>Fully Adjusted Model</u> (<i>N</i> =1,072; obs.=3,174)	
	IRR	95% CI	IRR	95% CI
Concurrent BPD	1.08 ^{**}	[1.05,1.12]	1.04 [*]	[1.00,1.07]
Prior year BPD	1.00	[0.97,1.03]	0.99	[0.96,1.02]
Prior year alcohol use	0.82 ^{**}	[0.79,0.85]	0.81 ^{**}	[0.78,0.85]
Concurrent tobacco use			1.19	[0.98,1.44]
Prior year tobacco use			1.10	[0.91,1.32]
Concurrent marijuana use			1.22 ^{**}	[1.18,1.25]
Prior year marijuana use			1.04 [*]	[1.00,1.07]
Concurrent depressive symptoms			1.01	[0.99,1.02]
Concurrent conduct problems			1.06 ^{**}	[1.04,1.09]

Notes. IRR = Incidence rate ratio; BPD = borderline personality disorder; obs. = observations.

All models estimated with fixed effects Poisson regressions. Linear and quadratic age included in all models. Analytic sample only includes participants who had at least two years of data and at least one year with a non-zero value on the outcome (model default).

* $p < .05$;

** $p < .001$