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Trajectories of Attention Deficit Hyperactivity Disorder and Oppositional Defiant Disorder Symptoms as Precursors of Borderline Personality Disorder Symptoms in Adolescent Girls

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Abstract

Little empirical evidence exists regarding the developmental links between childhood psychopathology and borderline personality disorder (BPD) in adolescence. The current study addresses this gap by examining symptoms of attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) as potential precursors. ADHD and BPD share clinical features of impulsivity, poor self-regulation, and executive dysfunction, while ODD and BPD share features of anger and interpersonal turmoil. The study is based on annual, longitudinal data from the two oldest cohorts in the Pittsburgh Girls Study (N = 1233). We used piecewise latent growth curve models of ADHD and ODD scores from age 8–10 and 10–13 years to examine the prospective associations between dual trajectories of ADHD and ODD symptom severity and later BPD symptoms at age 14 in girls. To examine the specificity of these associations, we also included conduct disorder (CD) and depression symptom severity at age 14 as additional outcomes. We found that higher levels of ADHD and ODD scores at age 8 uniquely predicted BPD symptoms at age 14. Additionally, the rate of growth in ADHD scores from age 10–13 and the rate of growth in ODD scores from 8–10 uniquely predicted higher BPD symptoms at age 14. This study adds to the literature on the early development of BPD by providing the first longitudinal study to examine ADHD and ODD symptom trajectories as specific childhood precursors of BPD symptoms in adolescent girls.

Keywords

Borderline personality disorder; female children and adolescents; attention deficit hyperactivity disorder; oppositional defiant disorder; development

Symptom Severity Trajectories of Attention Deficit Hyperactivity Disorder and Oppositional Defiant Disorder as Precursors of Borderline Personality Disorder Symptoms in Adolescent Girls

Borderline Personality Disorder (BPD) is a heterogeneous condition characterized by impulsivity, affect dysregulation, and dysfunctional interpersonal relationships (American Psychiatric Association, 2000). Adolescent BPD symptoms are associated with poor functioning in adulthood, including lower educational and occupational achievement as well as less satisfaction in social and romantic relationships (Winograd, Cohen, & Chen, 2008). Individuals with BPD experience chronic negative mood which is a common feature of many mood and anxiety disorders (e.g., Trull et al., 2008). However, affective instability

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and particularly poorly controlled anger distinguishes the affective topography of BPD from these disorders (Henry, Mitropoulou, New, Koenigsberg, Silverman, & Siever, 2001; Trull et al., 2008), suggesting a unique affective component of this disorder. This anger is often manifested as hostility in day-to-day social exchanges and underscores much of the interpersonal turmoil characteristic of this disorder (Gunderson, 2007; Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007; Stepp, Pilkonis, Yaggi, Morse, & Feske, 2009). Impulsivity and poor self-regulation are also key features and may manifest as risky decision making, self-harm, and substance use (Siever & Davis, 1991; Trull, 2001).

According to the biosocial theory, the development of BPD is driven by an interaction between impulsivity and affective dysregulation (Crowell, Beauchaine, & Linehan, 2009; Linehan, 1993). The inter- and intra-personal features of the disorder are thought to emerge as a result of transactions between these biological vulnerabilities and invalidating environmental influences. We think it is plausible that childhood disorders that are precursors of BPD would share the underlying features of impulsivity, affective dysregulation, and interpersonal difficulties that are hallmark of BPD. It is also plausible that these childhood disorders may develop into BPD during adolescence. Although it is possible that the processes underlying the expression of any one childhood disorder may increase the likelihood of developing BPD in adolescence and young adulthood, there are unique areas of overlap between core features of BPD and the childhood disorders Oppositional Defiant Disorder (ODD) and Attention Deficit Hyperactivity Disorder (ADHD) which warrant further investigation as to the putative developmental links between these conditions.

Attention Deficit Hyperactivity Disorder and Borderline Personality Disorder

ADHD and BPD share common behavioral and neuropsychological impairments, which may explain the putative developmental links between these two disorders. Impulsivity, a key feature in both the clinical manifestation and development of BPD, is also a core deficit in ADHD (e.g., Barkley, 1997). Additional shared features of ADHD and BPD include poor self-regulation, executive function, and inhibitory control (Daruna & Barnes, 1993; Dowson et al., 2004; Evenden, 1999; Philipsen, 2006). Dysfunction in the prefrontal cortex cuts across ADHD and BPD, which implicates overlapping neurological and behavioral mechanisms for these two disorders (Spencer, Biederman, Wilens, & Faraone, 2002; Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). Both disorders are characterized by impairment in executive functioning processes, such as working memory and attentional regulation (Barkley, 1997; Nigg et al., 2005; Willcut, Pennington, Olson, Chhabildas, & Hulslander, 2005). Lampe and colleagues (2007) found that patients with ADHD and BPD did not differ in behavioral aspects of impulsivity, including angry temperament and non-planning impulsiveness. These impairments may be due to reduced cortical inhibition, which characterizes patients with BPD (Barnow et al., 2009) and ADHD (Gilbert et al., 2004; Richter et al., 2007). Despite a significant association between ADHD and BPD, the developmental continuity between childhood ADHD and later BPD has not been established due to the lack of prospective studies measuring ADHD and BPD.

Several studies have found that ADHD and BPD often co-occur. Coolidge and colleagues (2000) reported higher ADHD scores among children aged 5–17 years who had BPD features compared to children with other personality disorder features. Similar findings have been reported in adult populations. Black and colleagues (2007) reported that for adult male and female prisoners with BPD the odds of currently having ADHD are 3.4 times larger than the odds for prisoners without BPD. Conversely, Rösler and colleagues (2009) reported that for female prisoners with ADHD the odds of having BPD are 5.2 times larger than the odds for female prisoners without ADHD.

In support of a developmental link, research has demonstrated associations between ADHD in childhood and BPD in adulthood. Fischer and colleagues (2002) were the first to demonstrate a prospective link between ADHD and BPD. They found that children with ADHD (age at first assessment ranged from 4–12 years) had higher rates of BPD as young adults at follow-up (mean length of follow-up was 13.8 years, mean age at follow-up=20–21 years) compared to community controls. Retrospective research with adult samples has found a similar pattern of results. Adrulonis (1991) was among the first to propose that ADHD and BPD may both be caused by overlapping neurological mechanisms and found that 50% of adult males with BPD met criteria for learning disabilities and ADHD in childhood. Fossati and colleagues (2002) found higher mean scores of childhood ADHD in a group of adult patients with BPD compared to those with other personality disorders and controls. Similarly, Philipsen and colleagues (2008) reported a significant association between childhood ADHD and severity of BPD in an adult treatment-seeking sample. Participants reporting childhood ADHD were more likely to endorse BPD criterion 8 (‘difficulty controlling anger’) and criterion 9 (‘stress-related dissociative symptoms/ paranoid ideations’) compared to those not endorsing childhood ADHD.

Oppositional Defiant Disorder and Borderline Personality Disorder

The affective and interpersonal terrain of ODD is similar to that of BPD. Specifically, ODD and BPD are both characterized by poorly controlled anger and hostility (Burke, Hipwell, & Loeber, 2010). Children with ODD have been found to have chronic interpersonal problems characterized by hostile and defiant behaviors that extend beyond authority figures to include peer networks (Loeber, Burke, Lahey, Winters, & Zera, 2000; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). Dysfunction in frontolimbic regions, including regions of the amygdala, is common for both ODD and BPD, suggesting a shared neurobiological pathway for problems with emotion regulation (Burke, Loeber, Birmaher, 2002; Lieb et al., 2004). Considering the phenomenological associations between ODD and BPD, it is plausible that this childhood disorder may be a precursor of BPD.

ODD is described in the DSM-IV TR (APA, 2000) as a pattern of negativistic, defiant and hostile behaviors towards adults and authority figures. It includes both oppositional behaviors, such as arguing and defying directives, and symptoms of negative affect, specifically being touchy or angry. ODD is a strong predictor of later CD (Loeber et al., 2000) and of depression as well (Burke, Loeber, Lahey, & Rathouz, 2005; Copeland, Shanahan, Costello, & Angold, 2009). Furthermore, the behavioral and affective symptom dimensions of ODD have been shown to differentially predict subsequent behavioral versus affective disorders (Burke et al., 2010; Stringaris & Goodman, 2009). As a predictor of subsequent behavioral and mood disorders, ODD may play a pivotal role in the development of a variety of other types of psychopathology.

ODD is not only a disorder characterized by poor reaction to adult authority. It is also associated with significant impairments in peer interactions. Boys with ODD have been found to generate more aggressive solutions and show less accurate encoding of social information than boys without ODD (Coy, Speltz, DeKlyen, & Jones, 2001). Compared to children with other psychiatric disorders, children with ODD also show more problems with social behaviors (Greene et al., 2002). In particular, compared to children with ADHD, children with ODD show greater hostility towards peers and lower resistance to provocation from peers (Frankel & Feinberg, 2002).

Because ODD is associated with persisting difficulties in affective, especially anger, and social dysfunction, it would be reasonable to speculate that this disorder is associated with later BPD. However, few studies have examined this possibility. Becker and colleagues (2006) found that the affective instability and irritability components of BPD were

significantly associated with ODD among 123 adolescents admitted to a psychiatric hospital. This pattern of associations is important to establish in a non-treated sample. Coolidge and colleagues (2000) also found that ODD, as well as CD, was associated with BPD features in contrast to other personality disorders in a small, cross-sectional study of youth between age 5 and 17. It is unknown whether CD was related to BPD due to the shared variance between ODD and CD in this sample.

Very little other direct examination of the relationships between ODD and BPD has been undertaken. Several studies have examined the relationship between CD and BPD without considering ODD as well. Ceballos and colleagues (2006) found distinct patterns of brain function, suggestive of delayed maturation, in adolescents with CD with and without BPD, compared to those with BPD without CD and those with neither disorder. CD symptoms assessed by retrospective report differentially predicted BPD in contrast to Avoidant Personality Disorder in a clinical sample (Joyce et al., 2003). Overall, the available literature suffers from an absence of longitudinal evidence to test hypotheses about the relationship from early ADHD and ODD to later BPD, controlling for the presence of commonly comorbid conditions and associated factors, such as CD and depression. Because these disorders are commonly comorbid, but also differentially related to other psychopathology, while also each potentially reflecting distinct aspects of BPD, it is important that they be simultaneously accounted for to ascertain their relative contributions to the prediction of BPD.

Many factors have limited our understanding regarding childhood psychopathology precursors of BPD. First, by clinical convention, personality disorders are not often assessed in children and adolescents, and childhood disorders, such as ADHD and ODD, are not often assessed in adults, which has resulted in a dearth of data regarding these potential developmental links. Moreover, what is known about the associations between childhood disorders and later BPD relies heavily on retrospective reporting from adult patients with the disorder. Lastly, the specificity with which childhood precursors uniquely predict BPD remains unknown. We aim to extend beyond these limitations by using longitudinal data to examine the prospective associations between ADHD and ODD symptom severity in childhood and later BPD symptoms in adolescent girls. There is overlap between ADHD, ODD, depression, and CD and therefore, it is not known whether the apparent overlap between BPD and ADHD and ODD is unique or the result of general comorbidity. Thus, to examine the specificity of these relations, we control for associations between these predictors and overlapping adolescent outcomes, that is depression and conduct disorder (CD) symptom severity.

Our current study exclusively focuses on girls; thus, we will not be able to explore possible gender differences in the developmental course of these disorders. We will briefly review possible gender differences in the prevalence and overlap of these disorders. There is a higher prevalence of ODD and ADHD among boys compared to girls (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003) and a higher prevalence of BPD among women in clinical settings compared to men (Skodol & Bender, 2003). Therefore, much of the work on childhood ODD and ADHD has focused on boys while the majority of research on BPD has focused on girls and women resulting in little progress toward understanding the developmental overlap of BPD with ADHD and ODD. Lower prevalence rates of ODD and ADHD in girls compared to boys may indicate that girls that do have either or both of these disorders have especially severe cases given the thesis of the gender paradox theory (for a review see Loeber & Keenan, 1994). This theory postulates that when unequal prevalence rates of disorder by gender exist, the gender with the lower prevalence rate will have a more severe form of the disorder when it does manifest. Thus, the severity of ADHD and ODD may be greater in girls than in boys, which could put these girls at heightened risk for more

severe psychopathology, such as BPD, in adolescence and young adulthood. Based on this premise, understanding the precursors of BPD in girls will yield important information about the etiology and developmental course for those girls and women who appear to be at particularly high risk for functional impairments and high levels of treatment utilization in adulthood.

The goals of the current study were to examine the prospective associations between developmental trajectories of ADHD and ODD symptom severity from age 8–13 years and later BPD symptoms at age 14 in girls, a time when BPD starts to emerge (Zanarini, Frankenburg, Ridolfi, Jager-Hyman, Hennen, & Gunderson, 2006; Miller, Muehlenkamp, & Jacobson, 2008). By including symptom severity trajectories from age 8 to 13 years, we examined the impact of ADHD and ODD across the developmental periods of late childhood and early adolescence. Given that the lack of impulse control in BPD overlaps with CD (e.g., Coolidge et al., 2000), and the negative affectivity component of ODD is also related to depression (e.g., Burke et al., 2010), we also examined the specificity with which the combination of these two precursors uniquely predicted BPD symptoms. Thus, we included CD and depression symptom severity at age 14 as additional outcomes. We hypothesized that both symptom trajectories of ADHD and ODD would predict BPD symptoms even when simultaneously considering the relations between these precursors and CD and depression at age 14. This study adds to the literature on the early development of BPD by providing the first longitudinal study to examine symptom severity trajectories of ADHD and ODD as specific childhood precursors of BPD symptoms in adolescent girls.

Method

Sample Description

The Pittsburgh Girls Study (PGS; *N*=2,451) involves an urban community sample of four girl cohorts, ages 5–8 at the first assessment, and their primary caretaker, who have been 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 details on study design and recruitment). The analyses here use eight years of data collected in the two oldest cohorts (*N*=1233) during late childhood and early adolescence, covering ages 8 to 14. African American girls made up slightly more than half of the sample (52.4%), while 41.9% were Caucasian. Most of the remaining 5.7% of girls were described as multi-racial. At the age 8 assessment, the majority of caretakers were female (92.3%); most (58.3%) were cohabiting with a spouse or domestic partner; and about half (52.6%) completed >12 years of education. Caregivers' ages ranged from 20 to 75 years (M = 36.0, SD = 8.43). Retention over follow-up was high, with an average participation rate of 93.5% over the 8 years of data collection. There were no statistically significant differences between retained participants and those lost to follow-up on any variables used in this study (i.e., race, emotionality, CD, depression, ADHD, ODD, and BPD).

Data Collection

Separate in-home interviews for both the girl and caretaker were conducted annually by trained interviewers using a laptop computer. At each assessment, girls and caretakers reported on girls' symptoms in the past year. All study procedures were approved by the University of Pittsburgh Institutional Review Board. Families were compensated for their participation.

Measures

Symptom trajectories—The severity of ADHD and ODD symptoms were assessed using caretaker reports on the Child Symptom Inventory – 4th edition (CSI-4; Gadow & Sprafkin, 1994) when girls were 8–13 years-old. By using caretaker-report we were able to avoid shared method variance with girls' self-report of the severity of BPD, depression, and CD symptoms at age 14. The CSI-4 includes DSM-IV symptoms (e.g., difficulty waiting turn; often argues with adults) scored on a four-point scale (0=*never* to 3=*very often*). We used symptom severity scores rather than symptom counts to increase variability in outcomes. The mean for ADHD symptom severity ranged from 11.52 (*SD* = 7.80) at age 12 to 13.67 (*SD* = 7.28) at age 8. The mean for ODD symptom severity ranged from 5.27 (*SD*= 3.85) at age 10 to 5.60 (*SD* = 3.34) at age 8. Adequate concurrent validity, and sensitivity and specificity of ADHD and ODD symptom scores to clinicians' diagnoses have been reported for the CSI (Gadow & Sprafkin, 1994). In the present study, the average internal consistency coefficient across data from age 8 to 13 years was α=.91, with values ranging from α=.89 (age 8) to α=.92 (age 13) for ADHD symptoms. For ODD symptoms the average internal consistency was α=.87, with values ranging from α=.83 (age 8) to α=.89 (age 12).

We calculated the number of girls in this study who had ADHD and ODD scores in the clinical range. Based on CSI-4 *T* scores and standard deviations, ADHD scores ranging from 17–23 indicate moderate severity and ≥ 24 indicate high severity. ODD scores ranging from 9–12 indicate moderate severity and ≥ 13 indicate high severity. In a clinical sample, 94% of children clinically diagnosed with ADHD and 72% of children clinically diagnosed with ODD received CSI-4 symptom severity scores in at least the moderate severity range (Gadow & Sprafkin, 1994). The number of girls in the ADHD moderate severity category ranged from 161 (14.1%) at age 12 to 264 (21.7%) at age 8. The number of girls classified as high severity for ADHD ranged from 86 (7.3%) at age 10 to 109 (9.0%) at age 8. The number of girls in the ODD moderate severity classification ranged from 107 (9.7%) at age 12 to 133 (10.8%) at age 9. The number of girls in the ODD high severity category ranged from 46 (3.7%) at age 9 to 71 (5.8%) at age 12.

Outcomes—BPD symptoms were assessed with girls' reports when they were 14 years-old using the questions from the screening questionnaire of the International Personality Disorders Examination (IPDE-BOR; Loranger et al., 1994). 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 (Smith, Muir, & Blackwood, 2005). In the present study, the mean BPD score was 2.49 (*SD* = 1.92), the median score was 2.0, and scores ranged from 0 to 9. The upper quartile of our sample had an average score of 4.0, which is in the clinically significant range (Smith et al., 2005). The internal consistency for BPD symptoms was adequate, α=.64, (Nunnally & Bernstein, 1994) but relatively lower when compared to other symptom scores used in this study, which may reflect the fewer number of items used to create this construct (Kuder & Richardson, 1937) or that the items underlying this score may be less unidimensional comparatively. Lastly, we inspected the content of BPD, ADHD, and ODD items for overlap. This was our preliminary check to ensure that results were not simply an artifact of relabeling ADHD and ODD symptoms as BPD symptoms. Two ODD items were similar to the wording of two BPD items. Specifically, the ODD items of 'temper tantrums' and 'angry and resentful' appeared similar to two BPD items: 'temper tantrums or angry outbursts' and 'very moody.' There were no ADHD items that appeared similar to BPD items. We also empirically examined whether symptom overlap explained the predictive relations between childhood ADHD and ODD symptoms and later BPD symptoms. These findings are presented in the results section.

Depression and CD symptom severity at age 14 were assessed using girls' reports on the Adolescent Symptom Inventory – 4th edition (ASI-4, Gadow & Sprafkin, 1998). The ASI-4 includes DSM-IV symptoms (e.g., depressed mood, stealing) of depression and CD (American Psychiatric Association, 1994) scored on a four-point scale (0=*never* to 3=*very often*). Adequate concurrent validity, and sensitivity and specificity of depression and CD symptom severity scores to clinicians' diagnoses have been reported for the ASI-4 (Gadow & Sprafkin, 1998). In the present study, the mean depression score was 8.02 (*SD* = 4.78), and the mean CD score was 1.36 (*SD* = 2.06). The internal consistency was α=.82 for depression symptom severity and α=.73 for CD symptom severity.

Covariates—We included minority race (0=Caucasian, 1=Minority Race), the temperamental construct of negative emotionality from the first assessment, and severity of depression and CD symptoms based on age 8 caretaker reports as covariates. We included emotionality and severity of depression and CD symptoms to control for the relation between temperament and early symptoms of CD and depression on BPD in adolescence (e.g., Clarkin & Posner, 2005). Emotionality was measured by caretaker report using the Emotionality, Activity, and Sociability Temperament Survey (Buss & Plomin, 1984). The emotionality subscale consists of five items (e.g., "She cries easily") scored using a five-point scale (1=*a little* to 5=*a lot*). The mean emotionality score was 12.87 (*SD* = 4.95). The emotionality subscale has demonstrated construct validity in comparisons of girls with and without depression in a community sample (Goodyer, Ashby, Altham, Vize, & Cooper, 1993). In our study, the internal consistency coefficient for the emotionality scale was α=. 82. As with ADHD and ODD scores, we assessed severity of depression and CD symptoms using caretaker reports on the CSI-4 when the girls were 8 years-old. The mean depression score was 4.36 (*SD* = 2.72), and the mean CD score was 1.22 (*SD* = 1.89). The internal consistency was α=.68 for depression severity and α=.70 for CD symptom severity.

Data Analytic Plan

We conducted a series of latent growth curve models (LGCMs) to investigate the relation between trajectories of ADHD and ODD scores and BPD symptoms at age 14. We first employed a two-step procedure to create unconditional LGCMs separately for ADHD and ODD symptom severity trajectories: (1) identifying the unconditional functional form and significance of the growth parameters and (2) estimating the unconditional LGCMs within a multiple group context to examine whether the identified trajectories were similar across cohorts. Next, we modeled the ADHD and ODD symptom severity trajectories simultaneously to examine the associations between the growth processes. This allowed us to account for the shared overlap in ADHD and ODD symptom severity trajectories. We then tested conditional growth models in which predictors of these trajectories were examined. Lastly, we tested our substantive questions of interest using the conditional dual model of ADHD and ODD to predict BPD, CD, and depression scores at age 14.

Due to the sampling technique used at recruitment, which oversampled girls in low income neighborhoods, a weighting variable was applied to all analyses in order to obtain rates for the general population of girls in Pittsburgh. To handle non-normal distributions of study variables we used a robust maximum likelihood indicator. Missing data on dependent variables was handled through the use of the expectation maximization (EM) algorithm. Sample sizes for analyses varied between *n*=1220–1233 due to missing data. All models were estimated with Mplus 5.2 (Muthén & Muthén, 2008). Model fit was evaluated using the χ^2 goodness of fit test, comparative fit index (CFI), Tucker-Lewis index (TLI), and root-mean-square error of approximation (RMSEA). For CFI and TLI, we used the conventional cutoff ≥.90 for acceptable fit, and ≥.95 for good fit. RMSEA values between .05–.08 represent acceptable fit, while values <.05 indicate good fit (McDonald & Ho, 2002).

Results

Creating Latent Growth Curve Models

Zero-order correlations for all study variables are presented in Table 1. We first fit a series of unconditional LGCMs to determine the optimal form of growth across ages 8–13 years in symptom severity scores, separately for ADHD and ODD. The forms of growth we investigated included a linear model, a quadratic model, and a piecewise linear model. For both types of piecewise growth models we examined ages 10 and 11 years for the joint that connects the two pieces of the growth model (Bollen & Curran, 2006). These ages were the only possible joints in this model as three time points are needed to estimate a slope factor. Since these models were not nested, we compared fit by examining the match between the model estimated growth and the observed means of symptom scores at each age (c.f. Hussong, Flora, Curran, Chassin, & Zucker, 2008).

For ADHD symptom scores, a piecewise growth model with an estimated time score at age 9 on the first piece provided the best model fit, while providing a parsimonious interpretation, χ^2 (11, N = 1220) = 11.41, p = .41; CFI = 1.000 and RMSEA = .006.1 The first piece described change from age 8 to 10 years, while the second linear piece described change from 10 to 13 years. For ODD symptom scores, a linear piecewise growth model provided the best model fit and allowed for the most parsimonious interpretation, χ^2 (11, N = 1220) = 16.27, p = .13; CFI = .998 and RMSEA = .020. As with ADHD, the first linear piece described change from age 8 to 10 years and the second linear piece depicted change from age 10 to 13 years. The intercept factor in both growth models represented symptom scores at age 8 years to enable the initial effect of ADHD and ODD scores on BPD, CD, and depression during adolescence to be examined.

The unconditional piecewise LGCM for ADHD scores resulted in a mean intercept that differed significantly from zero, M_i = 13.64, z = 65.12, p <.001; slope for 8–10 years, M_{s1} = −.86, z = −9.95, p <.001; and non-significant slope for 10–13 years, M_{s2} = −.09, z = −1.45, p = .24.2 The variances were significant for the intercept, D_i = 48.01, z = 8.67, p <.001; slope for 8–10 years, D_{s1} = 5.50, z = 4.42, p <.001; and slope for 10–13 years, D_{s2} = 1.60, z = 5.03, p <.001, indicating substantial variation across girls in initial ADHD scores and the shape of the subsequent trajectories. The intercept was significantly correlated with both the slope for 8–10 years (R_{is1} = −6.60, p = .011) and the slope for 10–13 years (R_{is2} = −1.66, p = .002). The slopes were not significantly correlated.

The unconditional piecewise LGCM for ODD scores resulted in a significant mean intercept, M_i = 5.20, z = 59.47, p <.001; and slope for 8–10 years, M_{s1} = −.15, z = −3.40, p <.001; but a non-significant slope for 10–13 years, M_{s2} = .02, z = .68, p = .50. The variances were significant for the intercept, D_i = 7.49, z = 11.93, p <.001; slope for 8–10 years, D_{s1} = .65, z = 3.69, p <.001; and slope for 10–13 years, D_{s2} = .44, z = 4.97, p <.001, indicating substantial variation across girls in initial ODD symptoms and subsequent trajectories. The intercept was significantly correlated with the slope for 10–13 years (R_{is2} = −.45, p = .001) but not 8–10 years. As previously, there was no correlation between the slopes.

[1]The fit statistics for the alternative forms of growth were as follows: linear models of ADHD [χ^2 (16, N = 1220) = 69.50, p < .001; CFI = .98; RMSEA = .052] and ODD symptom scores [χ^2 (16, N = 1220) = 72.08, p < .001; CFI = .98; RMSEA = .054]; quadratic models of ADHD [χ^2 (12, N = 1220) = 11.91, p =.45; CFI = .99; RMSEA = .030] and ODD symptom scores [χ^2 (12, N = 1220) = 15.01, p = .24; CFI = .98; RMSEA = .034].

[2]Even without significant growth in the unconditional model in ADHD scores from age 10–13, including covariates and outcomes can influence the slope so that it may vary as a function of these additional variables (c.f. Fisher & Kim, 2007). Specifically, we were able to demonstrate the significant predictive effect of the development trajectory of ADHD from age 10–13 on BPD symptoms at age 14. These seemingly contradictory results may be due to increased power to detect slope variability when covariates and outcomes are included in the model.

Latent Growth Curve Models across Cohorts

The piecewise LGCMs for ADHD and ODD symptoms were then estimated within a multiple group context to examine whether the trajectories were similar across cohorts. Cross-group (cohort) equality constraints were specified for the latent intercept and slope means, variances, and covariances. To test for cohort differences on growth parameters, we examined a series of nested multiple group models. Each of these nested models constrained a single parameter (e.g., intercept variance) to be equal for Cohort 7 and Cohort 8 girls. We used a $\chi 2$ difference test to compare each nested model with the base model (i.e., the model that assumed the parameter to be unequal across cohorts). If the constraint did not result in a significantly worse fit over the base model, the parameter was assumed to be equal for both cohorts. Multiple group analyses for the unconditional LCGM of ADHD did not reveal any cohort differences for intercept mean [$\Delta\chi^2$ (1) = 3.60, p = .058], slope means [slope 1: $\Delta\chi^2$ (1) = 2.24, p = .135; slope 2: $\Delta\chi^2$ (1) = 1.61, p = .205], intercept variance [$\Delta\chi^2$ (1) = 1.39, p = .238], and slope variances [slope 1: $\Delta\chi^2$ (1) = 2.36, p = .125; slope 2: $\Delta\chi^2$ (1) = 0.20, p = .655], indicating that the mean growth as well as the intraindividual variability in initial and developmental course of ADHD symptoms scores did not differ substantially between Cohort 7 and Cohort 8 girls. Similarly, multiple group analyses for the unconditional LCGM of ODD symptom scores did not reveal any cohort differences for intercept mean [$\Delta\chi^2$ (1) = 2.86, p = .091], slope means [slope 1: $\Delta\chi^2$ (1) = 2.90, p = .089; slope 2: $\Delta\chi^2$ (1) = 0.69, p = .406], intercept variance [$\Delta\chi^2$ (1) = 2.99, p = .084], and slope variances [slope 1: $\Delta\chi^2$ (1) = 2.36, p =.125; slope 2: $\Delta\chi^2$ (1) = 0.31, p = .578].

Fitting a multiple group piecewise LGCM to the data for ADHD scores produced an acceptable Chi-square test statistic χ^2 (23, N = 1220) = 37.033, p = .12, and fit indices representing an acceptable model fit, CFI = .996 and RMSEA = .023. Fitting a multiple group piecewise LGCM to the data for ODD scores also produced an acceptable Chi-square test statistic χ^2 (23, N = 1220) = 20.761, p = .84, and fit indices representing an excellent model fit, CFI = 1.000 and RMSEA <.001. These fit statistics indicate that the LGCMs for ADHD and ODD scores were similar across the two cohorts, justifying our use of single LGCMs for the two cohorts.

Dual Process Latent Growth Curve Models

As an intermediate step, the ADHD and ODD trajectories were modeled simultaneously to estimate the relations among the latent growth processes. Fitting dual process piecewise LGCMs to model ADHD and ODD symptoms in parallel produced a non-significant Chi-square test statistic χ^2 (46, N = 1220) = 56.738, p = .13, and fit indices representing an acceptable model fit, CFI = .998 and RMSEA = .014. The ADHD intercept was significantly correlated with the ODD intercept (β = .62, p<.001) such that levels of ADHD symptoms and ODD symptoms were significantly associated at age 8 years. The slopes depicting change in ADHD and ODD symptoms from age 8–10 years (β = .78, p<.001) and the slopes capturing change in symptoms from age 10–13 (β = .62, p<.001) were also significantly correlated, indicating that growth rate in ADHD and ODD scores are positively related across late childhood and early adolescence.

Dual ADHD and ODD Model Conditioned on Minority Race, Emotionality and Severity of Depression and Conduct Disorder Symptoms at age 8

We examined the unique effects of minority race, negative emotionality at age 8, and severity of depression and CD symptoms at age 8 on our dual process piecewise LGCM. All covariates were entered simultaneously into the baseline model, allowing us to examine the effects of each covariate while controlling for all other covariates. Minority race was significantly associated with the intercept factor of ODD scores (β = −.26, p < .001) such that age 8 ODD scores were significantly higher for Caucasian girls than for minority girls.

The growth of ODD scores across childhood and early adolescence did not differ by minority race status. Minority race was also significantly associated with the first ADHD slope factor (β = .10, $p < .05$), indicating that minority girls showed greater increases in ADHD scores during late childhood. However, during early adolescence, their ADHD scores were similar to those of Caucasian girls.

Emotionality at age 8 was significantly and positively associated with the intercept factors of ADHD (β = .17, $p < .001$) and ODD symptoms (β = .23, $p < .001$) at age 8. However, emotionality at age 8 did not continue to exert an effect on the growth of ADHD and ODD symptoms across late childhood and early adolescence.

Similar to emotionality, depression and CD scores at age 8 were significantly associated with the intercept factors of ADHD (β = .23, $p < .001$) and ODD scores (β = .23, $p < .001$), such that higher depression and CD scores were related to higher ADHD and ODD intercept scores. However, depression and CD scores at age 8 did not significantly effect the growth of ADHD and ODD across ages 8–13.

Effect of the Conditioned Model on BPD symptoms at age 14, Including Severity of Depression and CD Symptoms as Additional Outcomes

To examine the unique effects of the conditioned baseline model on later borderline personality, BPD, CD, and depression scores at age 14 were regressed on the latent variable growth factors and covariates (Table 2). By including the three outcomes simultaneously, we tested for the prospective associations between our conditioned latent variable model and BPD symptoms at age 14, even after accounting for the relation that these predictors also have with CD and depression scores at age 14. Minority race was significantly associated with BPD symptoms at age 14 (β = .09, $p < .05$) such that BPD symptoms were significantly higher for girls of a minority background compared to Caucasian girls. A similar association with minority race was found for CD but not depression scores. Depression symptom severity at age 8 significantly predicted BPD symptoms (β = .08, $p < .05$) indicating that early depression severity was prospectively associated with higher BPD symptoms at age 14. A similar relation was found between depression scores at age 8 and later depression but not CD scores. Emotionality and CD scores at age 8 did not predict BPD, CD, or depression scores at age 14.

The intercept factors of ADHD (β = .14, $p < .05$) and ODD (β = .15, $p < .05$) scores were uniquely associated with BPD symptoms at age 14, such that higher levels of ADHD and ODD at age 8 predicted higher levels of BPD symptoms at age 14, even when accounting for the relations between the intercept factors and CD and depression scores at age 14. A similar association was found between initial levels of ODD and ADHD symptom severity predicting later CD symptom severity at age 14.

We also examined the unique effects of the latent slope variables on BPD symptoms at 14. The second ADHD slope was significantly associated with BPD symptoms (β = .17, $p < .05$), indicating that the rate of growth in ADHD symptoms from age 10–13 uniquely predicted higher levels of BPD symptoms at age 14. However, the rate of growth in ADHD scores from age 8–10 was not significantly associated with later BPD symptoms. The rate of growth in ADHD scores across late childhood and early adolescence was not specifically associated with either depression or CD scores at age 14. The first ODD slope was significantly associated with BPD symptoms (β = .24, $p < .01$) such that the rate of growth in ODD symptom severity from age 8–10 uniquely predicted BPD symptoms at age 14. The rate of growth in ODD scores from age 10–13 did not, however, uniquely predict later BPD symptoms. The rate of growth in ODD scores from 10–13 also uniquely predicted depression scores at age 14.3 These findings highlight the importance of timing in the

development of psychopathology leading to BPD symptoms. One possible developmental pathway to BPD for girls may start with ODD symptoms and then shift to ADHD symptoms.

Lastly, we ran a final set of analyses that allowed us to examine the effect of ADHD and ODD symptom trajectories from age 8–13 on BPD symptoms after controlling for ADHD and ODD symptoms at age 14. Importantly, results regarding the predictive associations between ADHD and ODD symptom trajectories and BPD symptoms at age 14 remained the same. Specifically, both the intercept of ADHD ($\beta = .15$, $p < .05$) and ODD ($\beta = .15$, $p < .05$) symptoms significantly predicted BPD symptoms at age 14. Additionally, the rate of growth in ADHD symptoms from 10–13 years ($\beta = .18$, $p < .05$) and the rate of growth in ODD symptoms from 8–10 ($\beta = .21$, $p < .05$) significantly predicted BPD symptoms. Thus, these results suggest that these findings are not an artifact of similar symptoms being labeled at one point in time as ADHD or ODD, and at another point in time as BPD.

Discussion

The current study rigorously examines ADHD and ODD severity as childhood psychopathology precursors of BPD symptoms in adolescent girls based on data drawn from a large, prospective study of girls' development. Results are consistent with previous work demonstrating retrospective associations between ADHD and BPD in adults (Fossati et al., 2008; Philipsen et al., 2008) and with hypotheses regarding the relation between ODD and BPD. These data offer an important demonstration of the prospective relation between childhood psychopathology and BPD in adolescent girls. Specifically, ADHD and ODD at age 8 predicted BPD symptoms at age 14. We also found evidence that the timing of growth in ADHD and ODD severity serves to predict BPD symptoms in 14 year-old girls. Growth in ODD severity from age 8–10, but not age 10–13, predicted BPD symptoms at age 14. Conversely, for ADHD, growth in scores from age 10–13, but not 8–10, predicted BPD symptoms at age 14. This suggests that for adolescent BPD symptoms, difficulties with emotion regulation and relationships may precede problems with impulse control. The effects of ADHD and ODD were independent of race, emotionality, CD, and depression. Such findings serve to highlight the course and timing of ADHD and ODD development that predicts BPD symptoms in adolescence, which suggests unique childhood psychopathology influences on this extremely debilitating problem in girls and young women. This is the first paper to demonstrate such prospective associations in a community sample of girls. These findings are consistent with previous research suggesting that the disruptive behavior disorders are predictive of Cluster B personality disorders (which includes BPD; Kasen, Cohen, Skodol, Johnson, & Brook, 1999), and with research demonstrating that inattention and relationship dysfunction during childhood and adolescence are predictors of later BPD (Carlson et al., 2009).

Even though the rate of ADHD and ODD is higher in boys compared to girls, girls with these disorders may have more extreme variants of the disorders and may have more comorbid conditions (Costello et al., 2003). Thus, it would seem that girls who do develop BPD may experience many features in common with girls who have ADHD and ODD, specifically anger, hostility and interpersonal problems, impulse control difficulties, and inattention.

[3]To determine the effect of the covariates on the final model of interest, we ran additional analyses without the covariates negative emotionality, conduct disorder symptoms, and depression symptoms at age 8. The pattern of results remained the same. The ADHD ($\beta = .16$, $p = .003$) and ODD ($\beta = .13$, $p = .020$) intercepts, as well as the ODD slope from 8–10 years ($\beta = .22$, $p = .022$) and the ADHD slope from 10–13 years ($\beta = .16$, $p = .037$) predicted BPD symptoms at age 14.

Of particular importance in our study, we controlled for the overlap between BPD, conduct problems, and depression by including these variables as additional age 8 covariates and age 14 outcomes in our model. Our findings are consistent with previous studies that demonstrated significant overlap between adolescents with conduct problems, depressed mood, and BPD in adolescence (Becker et al., 2006; Coolidge et al., 2006). By including these overlapping conditions in our study, we demonstrated prospective associations between ADHD, ODD, and BPD. By controlling for ADHD and ODD symptoms at age 14, we were also able to demonstrate that these findings cannot be accounted for by mere symptom overlap.

Girls of a minority background had significantly higher BPD symptoms and conduct disorder symptom severity compared to Caucasian girls. The higher rate, on average, of CD severity and BPD symptoms among minority girls, compared to Caucasian girls, likely reflects, in part, the greater proportion of African American families living in poverty in the study sample, given that poor neighborhood conditions are associated with conduct problems (Fontaine et al., 2009; Keenan et al., under review) and that family economic deprivation also predicts BPD symptoms over young adulthood (Cohen, Chen, Gordon, Johnson, Brook, & Kasen, 2008). Emotionality did not predict BPD symptoms at age 14, which is surprising given that Carlson and colleagues (2009) found a prospective association between this temperament variable and BPD in adults. This inconsistency could be due to the different populations used (adults versus adolescents), as well as additional covariates included, especially since emotionality was significantly associated with depression and CD severity at age 8.

These findings have implications for the research on the development of BPD during adolescence. Early childhood temperament and personality traits may put children and youth at risk for both Axis I and II disorders (De Clercq, De Fruyt, Ven Leeuwen, & Mervielde, 2006). The traits of affective dysregulation (especially anger and hostility) as well as impulsivity are hypothesized to be important drivers in the development of BPD (Crowell et al., 2009) and may put girls at risk for ADHD, ODD, and BPD. During adolescence, personality dimensions and personality disorder symptoms reach a moderate level of stability, similar to that during adulthood (e.g., Clark, 2007; Cohen, Crawford, Johnson, & Kasen, 2005; Grillo & McGlashan, 2005). Thus, adolescence may represent a time of critical importance for the development of personality disorders. Based on this evidence, Shiner (2009) posits that in early adolescence personality disorder symptoms may transact with Axis I disorders and negative or stressful life events to exacerbate personality disorder symptoms. Our findings provide initial support for the notion that BPD symptoms may emerge during adolescence as the result of previous ADHD and ODD symptoms. It is important to note that there is likely a transaction between vulnerabilities in emotion regulation, impulse control, and dysfunctional environments as well as traumatic events that must occur for BPD to emerge (e.g., Crowell et al, 2009). The current work highlights the importance of underlying vulnerabilities in this pathway but other aspects of youth's lives should be considered when developing a more comprehensive model of risk. Future work is needed to test whether these childhood disorders transact with stressful events or invalidating environments in the prediction of BPD during adolescence.

As determined from the dual process latent growth curve model, growth in ADHD and ODD scores was positively correlated across the developmental period under investigation. Our results on the dual trajectories of ADHD and ODD are also consistent with the numerous studies reporting comorbidity among these childhood disorders in both epidemiological and clinical samples of girls and boys (Angold, Costello & Erkanli, 1999; Biederman, Newcorn & Sprich, 1991; Maughan et al., 2004).

Strengths and Limitations

The findings presented here are significant in that they are the first to demonstrate a specific and prospective link between ADHD and ODD severity in childhood and BPD symptoms in adolescent girls. A major strength of this study is the longitudinal design, which decreases the reporting biases inherent when adults with BPD provide retrospective self-reports of childhood psychopathology. Furthermore, the repeated assessments of ADHD and ODD over 8 years increase the reliability and validity of these estimates. Data were collected from two informants, parent- and girl-report, which decreased shared method variance in the predictors and outcomes, providing a more conservative test of our hypotheses. Finally, to examine the specificity with which trajectories of ADHD and ODD predict BPD symptoms, we controlled for several variables. Specifically, we controlled for parent-reported temperament and age 8 depression and conduct disorder symptom severity as well as self-reported depression and conduct disorder symptom severity at age 14. Given the associations between BPD and conduct disorder problems and depression (e.g., Coolidge et al., 2000; Gunderson et al., 2008), this study was particularly stringent.

Certain study limitations warrant comment. Generalizability of study results may be limited to an urban community sample of girls in one particular geographic area. It is also of interest to examine the relation between these childhood precursors and BPD in boys, especially since BPD affects men and women equally in community samples (Torgersen, Kringlen, & Cramer, 2001). In addition, due to relatively low rates of full-blown disorders, we were not able to examine the prospective associations between childhood psychopathology and those girls who met criteria for a BPD diagnosis. Due to the nature of our study, we were not able to conduct semi-structured diagnostic interviews with study families, which would be the gold standard to determine the clinical severity of the symptoms that girls in our sample endorsed. Furthermore, the use of parental report regarding predictors and covariates underestimates certain covert conduct disordered behaviors and internalizing symptoms, but provides a way to reduce shared method variance with regard to report of girls' BPD symptoms and depression and CD symptom severity at age 14. It is also important to note that the findings presented here may not apply to all girls in our sample as different developmental trajectories may exist. For instance, some girls may have increasing ADHD scores and low and stable ODD scores, and this combination may not predict BPD symptoms at age 14. Now that we have determined the prospective relation between growth in ADHD and ODD and BPD symptoms in the aggregate for our sample, future work will examine classes of developmental trajectories over time. Examining the prospective relation between classes of joint developmental trajectories of ADHD and ODD and later BPD symptoms will provide a more fine-grained understanding of girls who are at particularly high risk (c.f. Nagin & Tremblay, 1999). Finally, this study focused on a relatively short developmental window (i.e., ages 8–14), and had only one time point assessing BPD symptoms. We did not assess BPD symptoms prior to age 14 and it is possible that BPD symptoms existed prior to this time point. BPD symptoms are being assessed in the ongoing study annually from age 14, and so a next step will be to examine predictors of this developmental trajectory. We also limited the number and type of covariates included to facilitate the interpretation of prospective effects. There probably are several factors, not included in the analyses, which may affect the association between ODD, ADHD, and later BPD symptoms, such as early maternal separation, childhood abuse, and parenting practices (Crawford, Cohen, Chen, Anglin, & Ehrensaft, 2009).

Implications for Clinical Practice

Girls who screen positive for ADHD and/or ODD symptoms should also be assessed for BPD symptoms that may also cause significant distress and impairment. Given that ADHD and ODD may be childhood precursors for adolescent BPD, children with ADHD and/or

ODD should be monitored for the emergence of BPD symptoms. Adolescents and youth with BPD should also be evaluated for ADHD and ODD as the presence of these Axis I disorders may inform treatment. Treatments for ADHD and ODD may hold promise for preventing and even treating BPD. For example, parent management training may be a useful treatment approach for BPD during adolescence. Lastly, treatments for BPD, such as Dialectical Behavior Therapy, may also hold promise in alleviating distress for some children with ODD and ADHD.

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Table 1

Zero-order correlations among all study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Covariates (Parent Report)																			
1. Minority race	1.00																		
2. Emotionality, Wave 1	0.03	1.00																	
3. CD, Age 8	0.13	0.26	1.00																
4. Dep, Age 8	0.08	0.25	0.32	1.00															
Observed ADHD and ODD symptom severity scores (Parent report)																			
5. ADHD, Age 8	0.07	0.28	0.45	0.34	1.00														
6. ODD, Age 8	−0.14	0.37	0.62	0.36	0.53	1.00													
7. ADHD, Age 9	0.08	0.28	0.39	0.29	0.68	0.41	1.00												
8. ODD, Age 9	−0.12	0.31	0.47	0.28	0.40	0.65	0.58	1.00											
9. ADHD, Age 10	0.12	0.26	0.41	0.30	0.63	0.41	0.67	0.48	1.00										
10. ODD, Age 10	−0.09	0.29	0.48	0.25	0.37	0.63	0.41	0.68	0.59	1.00									
11. ADHD, Age 11	0.11	0.22	0.37	0.27	0.59	0.38	0.64	0.44	0.72	0.46	1.00								
12. ODD, Age 11	−0.10	0.28	0.42	0.21	0.35	0.59	0.41	0.66	0.49	0.71	0.61	1.00							
13. ADHD, Age 12	0.12	0.26	0.38	0.25	0.55	0.39	0.60	0.44	0.68	0.47	0.72	0.49	1.00						
14. ODD, Age 12	−0.06	0.27	0.38	0.20	0.34	0.54	0.39	0.62	0.46	0.66	0.50	0.70	0.66	1.00					
15. ADHD, Age 13	0.14	0.21	0.35	0.22	0.52	0.32	0.56	0.37	0.61	0.39	0.67	0.43	0.71	0.46	1.00				
16. ODD, Age 13	−0.06	0.23	0.38	0.19	0.37	0.51	0.43	0.57	0.45	0.59	0.49	0.63	0.52	0.69	0.62	1.00			
Outcomes (Child Report)																			
17. BPD, Age 14	0.09	0.15	0.09	0.15	0.19	0.15	0.18	0.18	0.19	0.17	0.24	0.21	0.24	0.24	0.25	0.22	1.00		
18. Dep, Age 14	0.01	0.11	0.04	0.15	0.10	0.08	0.14	0.14	0.12	0.08	0.17	0.14	0.18	0.15	0.15	0.17	0.54	1.00	
19. CD, Age 14	0.13	0.02	0.08	0.04	0.10	0.08	0.15	0.13	0.13	0.11	0.20	0.14	0.21	0.15	0.21	0.19	0.38	0.34	1.00

Notes. $p < .05$ for rs ≥|.06|; $p < .01$ for rs ≥ |.08|; $p < .001$ for rs ≥ |.10|. CD = Conduct Disorder; DEP=Depression; BPD = Borderline Personality Disorder; ADHD=Attention Deficit Hyperactivity Disorder; ODD = Oppositional Defiant Disorder.

Table 2

Latent growth curve modeling results for the effect of Time 1 covariates and ADHD and ODD trajectories predicting BPD, CD, and Depression scores at Age 14

	Outcomes at Age 14 (Child Report)					
	Depression Severity		Borderline Personality Disorder Symptoms		Conduct Disorder Symptom Severity	
Covariate (Parent Report)	**β**	**95% CI**	**β**	**95% CI**	**β**	**95% CI**
Minority race	−.01	[−.09, .06]	.09*	[.02, .17]	.15***	[.08, .21]
Emotionality, Wave 1	.05	[−02, .12]	.07	[−01, .14]	−07	[−14, .01]
Conduct Disorder Severity, Age 8	−08	[−20, .04]	−09	[−22, .03]	−05	[−18, .07]
Depression Severity, Age 8	.11**	[.03, .19]	.08*	[.01, .16]	−04	[−11, .03]
ADHD Symptom Severity Trajectory (Parent Report)						
Intercept	.10	[−01, .21]	.14**	[.05, .26]	.11*	[.01, .21]
Slope Age 8–10	.12	[−06, .31]	−05	[−23, .16]	.11	[−06, .30]
Slope Age 10–13	.06	[−09, .23]	.17*	[.02, .33]	.11	[−03, .27]
ODD Symptom Severity Trajectory (Parent Report)						
Intercept	.07	[−09, .23]	.15*	[.01, .30]	.19*	[.03, .35]
Slope Age 8–10	.06	[−13, .24]	.24**	[.03, .39]	.08	[−.10, .24]
Slope Age 10–13	.18*	[.02, .34]	.08	[−.11, .22]	.12	[−04, .28]

Note:

* $p<.05$,

** $p<.01$,

*** $p<.001$