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Adolescent Disruptive Behavior and Borderline Personality Disorder Symptoms in Young Adult Men

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Abstract

Very few studies have prospective information, especially regarding males, on the prediction of Borderline Personality Disorder (BPD) in adulthood from psychiatric disorders in childhood. Certain childhood disorders, however, have notably similar features in common with BPD. In particular, the affective dysfunction, hostility and interpersonal conflict of Oppositional Defiant Disorder (ODD) and the impulsivity of Attention Deficit Hyperactivity Disorder (ADHD) in particular may be indicative of an early developmental path towards BPD. The present study uses longitudinal data from a clinical sample of 177 boys, initially between the ages of 7 and 12, who were followed up annually to age 18, and who were reassessed at age 24 (n=142). The study examines the prediction from repeated childhood measures of psychopathology measured annually through adolescence to BPD symptoms assessed at age 24, accounting for the effects of covariates including substance use, other personality disorders at age 24 and harsh physical punishment. The prevalence of BPD in this sample was consistent with other population estimates. Attention Deficit Hyperactivity Disorder (ADHD) and ODD were the only child psychiatric disorders to predict BPD symptoms, and the oppositional behavioral dimension of ODD was particularly predictive of BPD. These results indicate possible developmental links between early psychiatric disorders and BPD.

Borderline personality disorder (BPD) is a severe condition involving marked dysregulation of affect and behavioral control, along with instability in interpersonal relationships, difficulties with self-image and identity, and for some, suicidal behavior. Although the presentation of the disorder is heterogeneous and varies across individuals, those with BPD commonly suffer greatly in terms of functional impairments (Skodol et al. 2002, 2005) in social, occupational, and academic settings, and are at increased risk for severe outcomes including violence towards others (Newhill, Mack & Mulvey, 2009) and completed suicide (Fertuck, Makhija & Stanley, 2007). BPD has been shown in representative population studies to affect men and women at approximately equal rates (Grant et al., 2008), and men and women have been found to be more similar that dissimilar in terms of clinical presentations (Johnson et al., 2003; but see Bradley, Conklin & Westin, 2005). In clinical samples, BPD is one of the most prevalent personality disorders, with reported estimates ranging between 10% to 25% (Gratz et al., 2009; Johnson et al., 2003) relative to rates in general population studies ranging from 0% to 5.9% (Grant et al., 2008).

Both the level of severity and the relatively high prevalence rates of BPD in clinical populations highlight the importance of early identification. However, a ubiquitous observation among papers on the subject is the paucity of evidence from longitudinal studies of BPD, particularly those relying on prospective data, that might help to elucidate developmental pathways from childhood to adulthood (see e.g. Carlson, Egeland & Sroufe,

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2009; Cole, Llera & Pemberton, 2009; Gratz et al., 2009). When considering the amount of evidence of the developmental relationships between child psychopathology and other personality disorders, particularly APD (e.g. Burke, Waldman & Lahey, 2010), the dearth of prospective evidence regarding the degree to which distinct psychiatric disorders of childhood predict an onset of BPD in adulthood is striking (Crowell, Beauchaine & Linehan, 2009; Shiner, 2009). Not only does this gap in the literature have implications for etiological and developmental models of psychopathology from childhood which might help to resolve questions about the manifestation of BPD in adulthood, but it also conversely leaves an area of darkness in the ability to distinguish among multiple potential outcomes from childhood psychiatric disorders.

There is in fact some prospective evidence to provide guidance, albeit indirectly, regarding prediction from child psychiatric disorders to BPD. One large community study of links between childhood psychiatric disorders and adult personality disorders using prospective data examined broad composites of both childhood Axis I disorders (grouped by disruptive behavior disorders, depression and anxiety disorder) and adult personality disorder clusters (Kasen et al., 1999). Although this strategy, as might be expected, tended to find relatively few relationships that were not significant, it was observed that Cluster B personality disorders were predicted by both the disruptive disorders and major depression, and less so by anxiety disorders.

Carlson, Egeland and Sroufe (2009) provided a comprehensive examination of an array of potential child, family and contextual factors as predictors of BPD measured prospectively from birth to adulthood in a selected risk sample. Although they did not include childhood psychiatric disorders per se, these results do provide valuable evidence regarding processes that putatively underlie childhood psychopathology. In particular, the authors found that childhood measures of activity and emotionality, and adolescent measures of attention, emotion regulation and relationship functioning in adolescence were particularly predictive of BPD symptom levels in adulthood (Carlson, Egeland & Sroufe, 2009). Relatedly, Gratz et al. (2009) reviewed evidence of the roles of affective dysfunction and disinhibition in the development of BPD, noting that no studies have examined the ways that these traits interact to predict BPD.

Deficits in emotion regulation have long been regarded as a hallmark of BPD (e.g. Linehan, 1993), and a number of related constructs regarding affective dysfunction and emotion dysregulation have been discussed in regards to the affective and emotional difficulties that individuals with BPD experience. An influential model of the etiology of BPD is the biosocial theory of BPD (Crowell, Beauchaine & Linehan, 2009). This model describes emotional dysregulation as the primary characteristic of BPD, but elaborates on an etiological mechanism of transactions between individuals with biological vulnerabilities and environmental influences as a key process in the development towards the disorder. This summary description of the model is broadly similar to the transactional model of the development of oppositional defiant disorder (ODD) proposed by Greene (Greene & Doyle, 2000), in that the manifestation of ODD is believed to be the result of a transactional process between emotionally or behaviorally dysregulated children and maladaptive parenting practices. Key differences between these transactional models involve the specificity of the invalidating environment in the biosocial theory of BPD (Crowell, Beauchaine & Linehan, 2009).

Nevertheless, given recent evidence regarding distinctions between affective dysregulation and behavioral features within ODD (e.g. Burke & Loeber, 2010), it is intriguing to consider whether and how it might be related to later BPD. Certainly, it is not the only disorder of childhood that could be theoretically related to BPD, and from a diathesis-stress perspective,

one could expect that the processes which make individuals liable to the ultimate manifestation of BPD might express themselves in a variety of ways through childhood. Likewise, BPD is not the only personality disorder that involves affective dysfunction and interpersonal conflict, but there are several fairly clear and unique areas of overlap between core features of BPD and of ODD. Particularly, affective dysregulation, interpersonal conflict and hostility are prominent among those with BPD and are also key features of ODD, or perhaps of a subset of individuals with ODD (Burke & Loeber, 2010). ODD involves interpersonal hostility not only towards authority figures, but towards peers as well (Moura & Burns, 2010; Taylor, Burns, Rusby & Foster, 2006) which very likely either impairs the development of typical social skills or reflects the manifestation of more primary traits related to interpersonal conflict. On the other hand, distinctions between dimensions of affect dysfunction may suggest ways in which the development of BPD differs from ODD. For instance, Cole, Llera and Pemberton (2009) in discussing emotional instability (rapid, unexpected changes in emotion) versus emotional reactivity, suggested that BPD might be distinguished from ODD in particular in that ODD may reflect a greater level of reactivity rather that instability.

Crowell, Beauchaine & Linehan (2009) note that BPD is phenotypically heterogeneous; those meeting criteria for BPD may vary markedly in the symptoms that they present. It may thus be tempting to consider the possibility that a developmental course involving ODD may describe different etiological pathways for boys and girls towards BPD in adulthood. Consistent with such a hypothesis, some evidence suggests that the manifestation of BPD among adolescent boys compared to girls may be distinguished by a more aggressive, disruptive and antisocial presentation for boys, in contrast to greater problems with internalizing and emotionally dramatic features for girls (Bradley, Conklin & Westin, 2005). On the other hand, while ODD may typically be regarded as a male disorder, it is somewhat misleading to characterize it in such a fashion. Whereas in childhood, there is a clear gender bias towards greater prevalence of the disorder among males, by adolescence, these prevalence rate disparities diminish sharply (Boylan, Vaillancourt, Boyle, & Szatmari, 2007). Thus, while evidence for gender-based differences in the presentation of BPD symptoms is intriguing, it is safe to say that very little is known about developmental processes for boys regarding outcomes of BPD.

The aforementioned absence of a solid description in the extant literature of the development from childhood psychopathology to BPD in adulthood holds for men and women. Ultimately the development towards BPD needs to be understood for both genders, particularly in light of evidence of similar prevalence rates across genders. At the same time, however, the development of psychopathology more broadly is known to differ greatly across genders. As a result, even if men and women develop similar rates of BPD, it is important to carefully describe the routes by which each gender arrives at that outcome, since the means to intervene and potentially prevent BPD may be different for boys and girls. This may be especially true if there are observed differences by gender in the development of candidate precursor conditions which share similarities in the nature of the impairments that they effect.

Given areas of clear overlap in the nature of impairments in functioning, ODD should be evaluated as a specific candidate within a model of pathways from childhood to BPD in adulthood. In the empirical literature, however, greater attention has been given to examining conduct disorder (CD) as a correlate of BPD (e.g. Ceballos and colleagues, 2006; Coolidge and colleagues, 2000). Becker, McGlashan and Grilo (2006) conducted a factor analysis of BPD symptoms and tested the relationship between the factors and other psychopathology in a clinical sample of male and female adolescents. They found that CD was related to a factor consisting of identity disturbance and impulsiveness, while ODD was

related to a factor of affective instability and uncontrolled anger, and was inversely related to the identity disturbance/impulsiveness factor. This disparate attention is not warranted by the defining characteristics of CD in contrast to ODD. CD is developmentally associated with ODD, but is itself defined entirely by antisocial behaviors and not by problems of affect regulation.

In contrast, new evidence highlights the impact of the affective features of ODD on developmental psychopathology through adolescence. In a community sample of girls (Burke, Hipwell & Loeber, 2010), ODD symptoms loaded onto three factors, one of which was a dimension of negative affect, consisting of being angry, spiteful and irritable. Among the three factors, negative affect alone predicted subsequent depression. Similar results were found among a clinical sample of boys; the affective dimension of ODD predicted depression but not CD, and the behavioral dimensions predicted CD but not depression (Burke, in press). In a community samples of boys and girls, affective and behavioral dimensions of ODD symptoms, with slight differences in terms of which items load onto which factors, have similarly found that a subset of affectively oriented ODD items differentially relates to depression (Rowe, Costello, Angold & Copeland, 2010; Stringaris & Goodman, 2009). A related construct, irritability as indexed in childhood by parent report of the ODD symptoms of anger and temper tantrums, has also been reported to predict depression and dysthymia, as well as anxiety, measured in adulthood 20 years later (Stringaris, Cohen, Pine and Liebenluft (2009). Intriguingly, particularly in light of the prediction of Cole, Llera and Pemberton (2009), this measure of irritability did not predict BPD in adulthood.

To be sure, there are a number of features of BPD that are not found among the symptoms of ODD, such as impulsivity, concerns about abandonment, or identity problems. As such, it is important that studies examine child psychopathology using distinct constructs rather than combining across disorders. For instance, evidence of the role of activity and attention levels in childhood and adolescence (Carlson et al., 2009) or disinhibition (e.g. Gratz et al., 2009) suggest that from a childhood psychopathology perspective, ADHD represents difficulties or traits not found among symptoms of other disorders that could serve as potential predictors of later BPD. In fact, ADHD has also been identified as commonly comorbid with both ODD (Angold, Costello & Erkanli, 1999) and BPD (Davids & Gastpar (2005), and has also been associated with BPD via retrospective data collection regarding ADHD history among adults with BPD (e.g. Philipsen et al., 2008). Similarly, among adults with ADHD, a high rate of Cluster B personality disorders, and specifically BPD, has been observed (Miller, Nigg & Faraone, 2007).

The present study is an effort to test a hypothetical link between ODD, and particularly affect dysfunction within ODD, and later BPD using prospective measures of parent reported child psychopathology through adolescence, and the symptom counts of BPD as assessed via self-report at age 24. The effects of ODD will be tested in the context of the distinct effects of CD, ADHD, depression and anxiety. Additionally, since important contextual factors such as substance use and parenting behaviors, particularly abusive behaviors (see reviews by Crick, Woods, Murray-Close and Han, 2007 and Paris, 2000), have been implicated in regards to the development of BPD, the predictive effects from child psychopathology measures will be tested controlling for measures of demographic factors, abusive parenting behavior, and substance use. Finally, in order to identify whether child psychiatric disorders have a distinct relationship with later BPD symptoms, we will test the final model after controlling for APD and other personality disorders contemporaneously measured in adulthood.

Our study hypotheses are that that ODD will be predictive of BPD symptoms, whereas CD will not, and that the affective dimension of ODD will in particular be predictive of BPD symptoms, due to the affective dysfunction reflected in the two constructs. Because of the impulsivity reflected in both ADHD and BPD, we hypothesize that ADHD will be also be predictive of BPD symptoms. We anticipate that these relationships will remain after controlling for other child psychopathology, substance use, harsh parenting, demographic factors, and other contemporaneous personality disorders.

Method

Data for the present analyses come from the Developmental Trends Study (Loeber, Green, Lahey, Frick & McBurnett, 2000), a sample of 177 boys who were recruited from psychiatric clinics in Pittsburgh, Pennsylvania and Athens and Atlanta, Georgia. Boys were between the ages of 7 and 12 at recruitment, and were followed up, along with a parent, via annual (except for study Year 5) child assessments to age 17. At 18, 19 and 24, young adult assessments were conducted, using report of the young men alone. The sample was over-representative of ADHD, ODD and CD.

At enrollment, boys had to be living with at least one biological parent, and were excluded if they had a history of mental retardation or psychosis, inpatient psychiatric treatment within the last six months, or could not discontinue taking psychotropic medication for two days prior to their scheduled assessment. Further details regarding participant recruitment have been published previously (Loeber et al., 2000).

Retention rates for the childhood through adolescent data collection ranged from a high of 100% in Year 2 to 87.1% in Year 10, with an average across all years of 93.4%. There were 164 participants (93%) who completed at least one young adult follow up interview at 18, 19, or 24 years of age; with 162 assessed at age 18, 153 assessed at age 19, and 142 assessed at age 24. The sample was composed of non-Hispanic white (70%) and African-American boys (30%).

Measures

Child psychopathology—A modified version of the DISC (Costello et al., 1987) was developed based on DSM-III-R criteria (Loeber et al., 1989). Data for the present analyses come from the parent version, which was administered at each assessment wave for study years 1 through 10 to assess symptoms of CD, ODD, ADHD, depression and anxiety. Although child and teacher versions were also used, child report of ODD was discontinued after Year 2 of the study, and teacher report on the DISC was conducted only during Years 1–4.

We used parent report on the DISC to create a composite depression variable by combining reports of symptoms of Dysthymia and Major Depression. As specified in the DSM III-R (APA, 1987), Major Depression includes anhedonia, psychomotor agitation or retardation and recurrent thoughts of death, whereas Dysthymia does not. Dysthymia includes hopelessness, which was not an element of the criteria for Major Depression. To avoid counting certain symptoms twice, we summed across ten unique symptoms of depression from the two sets of criteria at each wave in order to operationalize depression for this study.

Dimensions of ODD—Based on previous factor analyses which identified separate dimensions of ODD symptoms, and their relationship to later psychopathology (Burke, Hipwell & Loeber, 2010; Burke, in press) we constructed three dimensions of ODD symptoms, using parent reported symptoms on the DISC as described above. The convergent validity of these scales has been demonstrated in that the negative affective

dimension predicted depression among a clinic sample of boys (Burke, in press) and a community sample of girls (Burke, Hipwell & Loeber, 2010), and similarly the oppositional behavior dimension was predictive of later conduct disorder in each study.

In the present analyses, the negative affective dimension was created by summing the three ODD symptoms of being angry, spiteful and touchy. We summed the three symptoms of loses temper, argues and defies to create the oppositional behavioral dimension. Finally, the items regarding blaming others and annoying others were summed to create the antagonistic dimension.

We examined the reliability of these scales at each assessment wave. For the oppositional behavioral dimension, the average alpha value across waves was .76, ranging from .57 in Year 1 to .85 in Year 9. For the negative affective dimension, the average alpha was .69, ranging from a low of .58 in Year 2 to .75 in Year 8. For the antagonistic dimension, the average alpha was .55, ranging from .39 in Year 1 to .75 in Year 10. A common value for acceptable indicators of reliability is .70, although factors such as sample size and the number of items will impact the measurement of inter-item consistency. Nevertheless, because of the overall low scores for the antagonistic behavior construct, we decided to exclude it from the analyses.

Borderline Personality Disorder—At age 24, assessment of Cluster B personality disorders, including BPD, was conducted using the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The SCID-II has demonstrated generally desirable psychometric properties in regards to assessing BPD, showing a kappa of .91 for inter-rater reliability for DSM-IV BPD (Maffei et al., 1997), and of .48 for test-retest reliability for DSM-III-R BPD (First et al., 1995). We created both a categorical BPD construct and a dimensional BPD construct reflecting the symptom count. Predictive models described here focus on BPD symptoms as the outcome.

Other Personality Disorders—At 24 years of age, participants were assessed using the Computerized Diagnostic Interview Schedule Revised (DIS; Robins & Helzer, 1988). The temporal stability of DIS-based measurement of APD has been shown to be good (kappas = .60 and .58 for 6 and 12 month follow up assessments; Vandiver & Sher, 1991). The assessment included DSM-III-R criteria for APD, whereas DSM-IV criteria for APD did not include items regarding ability to function as a responsible parent and sustaining a monogamous relationship for one year. Additionally, in DSM-IV, two items pertaining to inconsistent work behavior and failure to honor financial obligations were combined. We developed DSM-IV APD scoring by excluding DIS data on the two items that were dropped from DSM-IV, and defining the item on work behavior and financial obligations by combining items from the interview for those criteria. For the present analyses, we used only APD symptom reports measured at age 18 and 19.

At age 24, the SCID-II (First et al., 1997) was also used to assess the disorders of Histrionic Personality Disorder (HPD), Narcissistic Personality Disorder (NPD) and Paranoid Personality Disorder (PPD). For the present analyses, we combined these three disorders by summing the total number of symptoms reported for HPD, NPD and PPD. We refer to this construct hereafter as other personality disorder symptoms.

Other covariates—In study years 3 through 10, children were questioned about their alcohol, tobacco, marijuana and hard drug use (Elliott, Huizinga, & Ageton, 1985). Also, at each assessment wave through year 10, a construct of harsh discipline was measured by asking parents a single item (“If your son does something that he is not allowed to do or that

you don't like, do you slap or spank him, or hit him with something?"). Parents responded on a three point scale. Because of low endorsement of "often" (<5%) at each assessment, this item was dichotomized to contrast the response of "never" with the combination of the responses "sometimes" or "often." At baseline, 67% (n =118) reported sometimes or often engaging in harsh punishment. Also, at Year 1, demographic information was collected, including parental marital status, the age at which the participant's mother first gave birth, and the number of siblings of the study participant living in the household. Parent self-reported educational and occupational background at Year 1 were used to create an index of socioeconomic status (SES). Table 1 provides descriptive statistics for continuous variables in the sample. In addition, 67% of the sample was Caucasian and 33% was African American, by parent report at Year 1, and 61% of parents reported that they were married.

Analyses: The analyses were conducted using generalized linear regression (GLM) models. In each model, the data were clustered by participant to account for correlations within individuals across the waves of data. This allowed us to examine the associations across all 10 waves of data through adolescence as predictors of outcomes at 24. To examine the count data of symptoms of BPD we anticipated overdispersion and tested the appropriateness of models specifying negative binomial mean distribution versus poisson mean distribution. We examined predictors and covariates in separate models by domain and then by including those significant from within domains together in a final model. In each model, we examined variance inflation factor (VIF) and tolerance statistics in order to ensure that multicollinearity was not a threat to the integrity of models. These revealed no model mean VIF, nor individual predictor parameter, greater than 1.7. Further, no tolerance statistic fell below .51. Commonly accepted values that might indicate concerns regarding multicollinearity would be a tolerance statistic less than .2, or any VIF greater than 5 (see e.g. Menard, 1995). Correlations among psychopathology predictors also revealed no concerns regarding multicollinearity (see Table 2).

In each model, all available data for a given individual were used. Missing data for any independent variable in a model resulted in the deletion of that wave of data from the model, but all other available waves of data for an individual would be retained in order to develop parameter estimates. Previous analyses of missingness in this data set have indicated that those with ADHD were more likely to refuse to participate in later waves of the study (Cotter, Burke, Loeber & Mutchka, 2005). To address this potential systematic bias, we tested the interaction of ADHD by time in the prediction of BPD symptoms. The interaction was not significant, suggesting that the relationship between ADHD and BPD symptoms did not vary across waves, and was thus not likely influenced by a bias towards missingness in later waves among those with ADHD.

Results

Of the 142 young men assessed at age 24, 12 (8.5%) met symptom count criteria (five or more symptoms) for BPD. Although proportionally the prevalence of BPD in this sample is generally consistent with that reported in other clinical samples (see Johnson et al., 2003 for a review), the overall number of participants was relatively low. Examination of model stability when predicting to categorical BPD resulted in significantly poor fit and high influence for those coded as positive for BPD. As a result, we chose to use BPD symptom count as the outcome in order to increase variability and improve model stability. This dimensional approach is also consistent with that preferred in other recent prospective longitudinal analyses of BPD as an outcome (Carlson, Egeland & Sroufe, 2009). The symptom count for BPD ranged from 0 to 9, with a mean of 1.20 symptoms (sd = 1.95). The majority of the sample (54.4%) showed no symptoms of BPD. Because of the nature of the distribution of symptom counts, with a greater variance (3.81) than mean, we suspected

overdispersion might influence model stability. Testing as described in Hardin and Hilbe (2007) confirmed significant overdispersion and led to our specification of negative binomial distributions for the regression models.

First, we tested a model of child and adolescent psychopathology predicting BPD symptoms (see Table 3). After stepwise removal, only the symptoms of ADHD (Incident Rate Ratio (IRR) = 1.08, se = .03, p = .002) and ODD (IRR = 1.09, se = .05, p = 04) remained significant predictors of BPD.

We then tested a series of models of covariates, grouped by substance use, demographic factors and harsh discipline. To retain covariates for further analyses, a stepwise removal procedure, specifying a probability of $p < .10$, was employed. Age was included as a covariate in each model.

Among substance use variables (marijuana, tobacco, alcohol or other drug use), only marijuana use (IRR 1.00, se = .001, p = .014) was significant. At the point of their removal from the model, the statistics for the other substance use variables were: alcohol (IRR = 1.00, se = .001, p = 0.61); drug use (IRR = 0.99, se = .004, p = .12); and tobacco use (IRR = 1.00, se = .001, p = .11). Among demographic factors measured at baseline (marital status, maternal age at birth, SES, and the number of siblings in the household) only the number of siblings (IRR = 0.78, se = .11, p = .07) was retained for further analysis. Harsh physical punishment was not predictive of BPD (IRR = 1.16, se = .16, p = .30).

Subsequently, we examined a model of ODD, ADHD, marijuana use and number of siblings as predictors of BPD symptoms. Both ODD (IRR = 1.09, se = .05, p = .04) and ADHD (IRR = 1.08, se = .03, p = .002) remained predictive. Marijuana use (IRR = 1.00, se = .001, p = . 04) also remained predictive of BPD symptoms, while the number of siblings (IRR = 0.87, se = .10, p = 0.23) did not.

The results of the models suggest that, holding other predictors constant, for each single additional ADHD symptom through adolescence the number of symptoms of BPD in adulthood would be predicted to increase 8%. Given 18 symptoms of ADHD, this would suggest a potential increase of 144% in the number of BPD symptoms in young adulthood. For ODD, the IRR of 1.09 suggests an increase of 9% in the number of BPD symptoms in adulthood for each additional symptom of ODD through adolescence. Across the span of 8 ODD symptoms, one might thus predict a relative increase of 72% in the symptoms of BPD observed in young adulthood.

In order to determine which of the dimensions of ODD symptoms (negative affective symptoms and oppositional behaviors) were related to later BPD symptoms, we tested them together in a model with only age as an additional covariate. Only the oppositional behavioral dimension was predictive (IRR = 1.24, se = .11, p = .016) while the negative affect dimension (IRR = 1.04, se = .09, p = .65) was not. We thus tested a model with ADHD, marijuana use, and oppositional behavior in place of ODD. Oppositional behavior (IRR = 1.21, se = .11, p = .028) was predictive of BPD in this model, along with ADHD and marijuana use.

Finally, in order to test whether these relationships were specific to BPD symptoms over and above the potential relationships between ADHD and ODD with personality disorders more generally, we tested whether or not the relationships between the identified predictors and BPD symptoms at 24 remained after accounting for the number of symptoms of APD measured at 18 and 19, and the number of symptoms of other personality disorders at 24. Doing so resulted in the removal of marijuana use (IRR = 1.00, se = .001, p = .71) and ADHD (IRR = 1.04, se = .026, p = .09) from the model, while the oppositional behavioral

dimension of ODD remained as a significant predictor (IRR = 1.16, se = .08, p = .03). Table 4 provides the statistics for this model.

Discussion

Among boys in this sample, the prevalence of BPD was proportionally consistent with that observed in other studies (Johnson et al., 2003), despite the fact that the sample consisted of a clinical sample of boys notable for a high level of disruptive behavioral problems. The present analyses suggest that among childhood psychiatric disorders, childhood and adolescent ADHD and ODD, rather than CD, depression, or anxiety were indicative of increased symptoms of BPD in young adulthood. Based on the models and the size of the increase in rates of BPD for each increase in the symptoms of ADHD or ODD, the potential difference in outcome for a child with many symptoms of either disorder compared to one without is large.

Contrary to our hypothesis, testing of the dimensions of ODD found that it was the behavioral dimension, rather than the affective dimension of ODD that predicted later BPD symptoms. While marijuana use was significantly predictive among substance use and psychopathology, its effect was not significant after accounting for the relationship between APD, other personality disorders and BPD.

Because the ODD affective dimension, consisting of the items angry, spiteful and irritable, has been associated with depression and appears to reflect affective dysregulation, we anticipated that it would be predictive of BPD symptoms. However, in the present results, it was the behavioral dimension of arguing, defying and losing temper that predicted BPD. This was consistent with the overall nature of the other predictors in the model, in that measures of affect (depression and affective ODD) were not predictive of BPD, whereas certain measures of behavior (ADHD and oppositional ODD behavior, but not CD) were. It was also consistent with the findings of Stringaris et al. (2009), who found that a construct of irritability consisting of anger and temper tantrums was not predictive of BPD. These findings may also provide support to the speculation, raised by Cole, Llera and Pemberton (2009), that the affective dysregulation of BPD may differ from that within ODD because of the instable, rapid and unexpected changes in emotion that are typically associated with BPD.

The hypothesis that ADHD would also predict BPD was born out. Conceptual linkages between the disorders hinge on the high degree of impulsivity that exists in both disorders. Like ODD, ADHD is a disorder of childhood that has historically been given less than adequate examination as a disorder persisting into adulthood. However, efforts over the past decade have been undertaken for understanding adult ADHD in a way that is not evident for ODD. This includes efforts to consider how best to identify and describe adult ADHD (e.g. McGough & Barkley, 2004; Faraone, Biederman & Spencer, 2010). It makes sense that, in conjunction with a basic tendency to argue and act in defiance, a failure to think through to likely outcomes and to act impulsively rather than deliberately may lead to difficulties finding appropriate interpersonal negotiation strategies that work or being able to give thought as to why past strategies have not been successful.

The predictive links between ODD oppositional behavior and BPD appear to be rather specific, in that they remained even after controlling for contemporaneous measures of APD and other personality disorders. The same was not true of ADHD nor of marijuana use, both of which were not significant after including other personality disorders. Thus it may be the case that ADHD and marijuana use portend some general risk for later personality disorders,

whereas ODD, and particularly the oppositional behaviors within ODD, suggest some specific risk for later BPD.

The link between the behavioral dimension of ODD and adult BPD may perhaps involve instability in close relationships. Parental perceptions of children arguing, defying and losing tempers need not only relate to rule breaking behavior. These features of ODD are also not necessarily limited to authority figures alone. Certainly being irritable and angry provide a noxious context for interacting with others, but arguing, defying and losing one's temper, rather than finding prosocial and appropriate strategies for negotiating with frustrations and impasses in an interpersonal exchange are also off-putting to others. Children who are high in ODD behavioral problems may not learn more appropriate interpersonal skills and may bring these poor strategies into their interpersonal interactions with others in adulthood. Future studies will be needed to examine whether the behavioral or affective dimensions of ODD show similar distinctions in the prediction of BPD for young women.

Given the relatively scant literature on childhood psychopathology and the prediction of adult BPD, it is difficult to place the present findings in an existing context. The present paper is the only paper we are aware of that prospectively examines childhood psychopathology as predictors of BPD in a manner in which distinctions can be made among the disorders. These findings are nevertheless consistent with previous research suggesting that the disruptive behavior disorders are predictive of Cluster B disorders (Kasen et al., 1999), and with evidence implicating attention and relationship functioning in childhood and adolescence as predictors of later BPD (Carlson et al., 2009). ADHD has previously been described as a correlate of BPD (Miller, Nigg & Faraone, 2007), but evidence for its predictive role has been inconsistent, based in large measure on data from methodologically limited studies (Davids & Gastpar, 2005). Similarly, CD has been discussed as a potential precursor to BPD, but again, the supportive evidence has been inconsistent (Paris, 2000). For instance, brain maturation distinguished between those with BPD versus those with CD (Ceballos et al., 2006). For both disorders, the available evidence has not included adequate data from prospective studies from childhood to BPD measured in adulthood. Based on the present results, it would appear that ADHD does indicate increased risk, but that it is ODD rather than CD that predicts later BPD, and perhaps a particular dimension of oppositional behavioral symptoms that carries the weight of the predictive strength for ODD. Since neither the negative affective dimension of ODD nor our measure of depression were predictive of later BPD, we did not find evidence of emotionality predicting later BPD. However, it is important to note that our measures of these processes were limited to those typically indicative of specific childhood psychiatric disorders, rather than being designed to assess alternate or more nuanced aspects of affective dysfunction.

Of additional importance is the evidence from the present results of outcomes of ODD as distinct from CD. The conception of ODD as benign apart from the degree to which it predicts CD may not fully describe the risks associated with the disorder. As Rowe and colleagues (2010) noted, in light of mounting evidence that ODD and CD represent distinct forms of psychopathology, it may be time to discard the conceptualization of ODD as hierarchically subsumed under CD. Whether these findings indicate an instance of heterotypic comorbidity (e.g. Angold et al., 1999), wherein a continuous process manifests in different forms over time, or whether BPD as expressed in childhood might overlap with ODD and ADHD cannot be answered in the present data set. Nevertheless, youth with ODD and ADHD appear to be at increased risk to show BPD symptoms in adulthood.

Limitations

The present study is limited by the nature of the data, which is a modestly sized clinical sample of boys who were disproportionately over-representative of disruptive behavioral

problems in childhood. The fact that this sample was derived from clinic referral raises concerns of a bias to see increased rates of other concurrent disorders. While it is useful to look at the present results as suggestive of hypothetical models of development, it is particularly important that they be replicated in samples that are representative of the community. The nature and strengths of the relationships between these disorders may be different among youth in a community sample. It is also important for prospective studies to examine the prediction from childhood psychopathology to adult BPD among girls as well. Finally, in the present data, we did not have a measure of abuse, but rather employed an indicator of the use of harsh physical discipline instead. These two constructs are clearly quite different, and it will be important to examine the degree to which abuse more appropriately measured might impact the prediction from measures of child psychopathology to BPD in adulthood. Nevertheless, the absence of prospective studies of predictors of BPD and of outcomes of ODD limit the ability to inform clinical prognosis for youth appearing in clinics at present.

References

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 3. Washington, D. C: American Psychiatric Association; 1987. revised

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4. Washington, D. C: American Psychiatric Association; 1994.

Angold A, Costello EJ, Erkanli A. Comorbidity. Journal of Child Psychology and Psychiatry and Allied Disciplines. 1999; 40:57–87.

Becker DF, McGlashan TH, Grilo CM. Exploratory factor analysis of borderline personality disorder criteria in hospitalized adolescents. Comprehensive Psychiatry. 2006; 47(2):99–105. [PubMed: 16490567]

Boylan K, Vaillancourt T, Boyle M, Szatmari P. Comorbidity of internalizing disorders in children with oppositional defiant disorder. Eur Child Adolesc Pscyhiatry. 2007; 16(8):484–494.

Burke, JD. The relationship between Conduct Disorder and Oppositional Defiant Disorder and their continuity with antisocial behaviors: Evidence from longitudinal clinical studies. In: Shaffer, D.; Leibenluft, E.; Rohde, LA., editors. Externalizing disorders of childhood: Refining the research agenda for DSM-V. Arlington, VA: American Psychiatric Association; (in press)

Burke JD, Hipwell AE, Loeber R. Dimensions of Oppositional Defiant Disorder as predictors of depression and Conduct Disorder in preadolescent girls. Journal of the American Academy of Child and Adolescent Psychiatry. 2010; 49(5):484–492. [PubMed: 20431468]

Burke JD, Loeber R. Oppositional defiant disorder and the explanation of the comorbidity between behavioral disorders and depression. Clinical Psychology: Science and Practice. 2010; 17:319–326.

Burke JD, Loeber R, Birmaher B. Oppositional Defiant Disorder and Conduct Disorder: A Review of the Past 10 Years, Part II. J Am Acad Child Adolesc Psychiatry. 2002; 41(11):1275–1293. [PubMed: 12410070]

Burke JD, Loeber R, Lahey BB, Rathouz PJ. Developmental transitions among affective and behavioral disorders in adolescent boys. Journal of Child Psychology and Psychiatry. 2005; 46(11):1200–1210. [PubMed: 16238667]

Burke JD, Waldman I, Lahey BB. Predictive validity of childhood oppositional defiant disorder and conduct disorder: Implications for DSM-V. Journal of Abnormal Psychology. 2010; 119:739–751. [PubMed: 20853919]

Carlson EA, Egeland B, Sroufe LA. A prospective investigation of the development of borderline personality symptoms. Dev Psychopathol. 2009; 21(4):1311–1334. [PubMed: 19825270]

Ceballos NA, Houston RJ, Hesselbrock VM, Bauer LO. Brain Maturation in Conduct Disorder versus Borderline Personality Disorder. Neuropsychobiology. 2006; 53(2):94–100. [PubMed: 16557039]

Cole PM, Llera SJ, Pemberton CK. Emotional instability, poor emotional awareness, and the development of borderline personality. Dev Psychopathol. 2009; 21(4):1293–1310. [PubMed: 19825269]

Coolidge FL, Segal DL, Stewart SE, Ellett JAC. Neuropsychological Dysfunction in Children with Borderline Personality Disorder Features: A Preliminary Investigation. Journal of Research in Personality. 2000; 34(4):554–561.

Copeland WE, Shanahan L, Costello EJ, Angold A. Childhood and adolescent psychiatric disorders as predictors of young adult disorders. Archives of General Psychiatry. 2009; 66(7):764–772. [PubMed: 19581568]

Costello, AJ.; Edelbrock, C.; Dulcan, MK.; Kalas, R.; Klaric, S. Diagnostic Interview Schedule for Children (DISC). Pittsburgh, PA: Western Psychiatric Institute and Clinic; 1987.

Cotter RB, Burke JD, Loeber R, Mutchka J. Predictors of contact difficulty and refusal in a longitudinal study. Criminal Behaviour and Mental Health. 2005; 15(2):126–137. [PubMed: 16470508]

Crick, NR.; Woods, K.; Murray-Close, D.; Han, G. Personality disorders in childhood and adolescence. Hoboken, NJ: John Wiley & Sons Inc; US; 2007. The development of borderline personality disorder: Current progress and future directions; p. 341-384.

Davids E, Gastpar M. Attention deficit hyperactivity disorder and borderline personality disorder. Progress in Neuro-Psychopharmacology and Biological Psychiatry. 2005; 29:865–877. [PubMed: 15951086]

Elliott, DS.; Huizinga, D.; Ageton, SS. Explaining Delinquency and Drug Use. Beverly Hills, CA: Sage Publications; 1985.

Faraone SV, Biederman J, Spencer T. Diagnostic Efficiency of Symptoms for Identifying Adult Attention-Deficit/Hyperactivity Disorder. Journal of ADHD and Related Disorders. 2010; 1:38–48.

Fertuck EA, Makhija N, Stanley BS. The nature of suicidality in borderline personality disorder. Primary Psychiatry. 2007; 14(12):40–47.

First, MB.; Gibbon, M.; Spitzer, RL.; Williams, JBW.; Benjamin, LS. User's guide for the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II). Washington, D.C: American Psychiatric Press; 1997.

First MB, Spitzer RL, Gibbon M, Williams JBW, Davies M, Borus J, Howes MJ, Kane J, Pope HG, Rounsaville B. The Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II), Part II: Multi-site Test-retest Reliability Study. Journal of Personality Disorders. 1995; 9(2):92–104.

Grant BF, Chou SP, Goldstein RB, Huang B, Stinson FS, Saha TD, et al. Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. J Clin Psychiatry. 2008; 69(4):533–545. [PubMed: 18426259]

Gratz KL, Tull MT, Reynolds EK, Bagge CL, Latzman RD, Daughters SB, et al. Extending extant models of the pathogenesis of borderline personality disorder to childhood borderline personality symptoms: the roles of affective dysfunction, disinhibition, and self- and emotion-regulation deficits. Dev Psychopathol. 2009; 21(4):1263–1291. [PubMed: 19825268]

Greene RW, Doyle AE. Toward a transactional conceptualization of oppositional defiant disorder: Implications for treatment and assessment. Clinical Child and Family Psychology Review. 2000; 2:129–148.c. [PubMed: 11227071]

Hardin, JW.; Hilbe, JM. Generalized Linear Models and Extensions. 2. College Station, TX: Stata Press; 2007.

Johnson DM, Shea MT, Yen S, Battle CL, Zlotnick C, Sanislow CA, Grilo CM, Skodol AE, Bender DS, McGlashan TH, Gunderson JG, Zanarini MC. Gender differences in borderline personality disorder: Findings from the Collaborative Longitudinal Personality Disorders study. Comprehensive Psychiatry. 2003; 44:284–292. [PubMed: 12923706]

Kasen S, Cohen P, Skodol AE, Johnson JG, Brook JS. Influence of child and adolescent psychiatric disorders on young adult personality disorder. Am J Psychiatry. 1999; 156(10):1529–1535. [PubMed: 10518162]

Linehan, MM. Cognitive-behavioral treatment of borderline personality disorder. New York: Guilford; 1993.

Loeber R, Green SM, Lahey BB, Stouthamer-Loeber M. Optimal informants on childhood disruptive behaviors. Development and Psychopathology. 1989; 1:317–337.

Loeber R, Green SM, Lahey BB, Frick PJ, McBurnett K. Findings on disruptive behavior disorders from the first decade of the Developmental Trends Study. Clin Child Fam Psychol Rev. 2000; 3(1):37–60. [PubMed: 11228766]

Maffei C, Fossati A, Agostoni I, Barraco A, Bagnato M, Deborah D, Namia C, Novella L, Petrachi M. Interrater reliability and internal consistency of the structured clinical interview for DSM-IV axis II personality disorders (SCID-II), version 2.0. Journal of Personality Disorders. 1997; 11(3):279–284. [PubMed: 9348491]

McGough JJ, Barkley RA. Diagnostic controversies in adult attention deficit hyperactivity disorder. American Journal of Psychiatry. 2004; 161:1948–1956. [PubMed: 15514392]

Menard, S. Applied Logistic Regression Analysis: Sage University Series on Quantitative Applications in the Social Sciences. Thousand Oaks, CA: Sage; 1995.

Miller TW, Nigg JT, Faraone SV. Axis I and II comorbidity in adults with ADHD. Journal of Abnormal Psychology. 2007; 116:519–528. [PubMed: 17696708]

Moura MA, Burns GL. Oppositional defiant disorder toward adults and oppositional defiant disorder toward other children: Evidence for two separate constructs with mothers' and fathers' ratings of Brazilian children. Journal of Child Psychology and Psychiatry. 2010; 51:23–30. [PubMed: 19941633]

Murphy KR, Barkley RA, Bush T. Young adults with attention deficit hyperactivity disorder: subtype differences in comorbidity, educational, and clinical history. Journal of Nervous and Mental Disorders. 2002; 190(3):147–157.

Newhill CE, Eack SM, Mulvey EP. Violent behavior in borderline personality. J Pers Disord. 2009; 23(6):541–554. [PubMed: 20001173]

Nock MK, Kazdin AE, Hiripi E, Kessler RC. Lifetime prevelance, correlates, and persistence of oppositional defiant disorder: Results form the National Comorbidity Survey Replication. Journal of Child Psychology and Psychiatry. 2007; 48(7):703–713. [PubMed: 17593151]

Offord DR, Boyle MH, Szatmari P, Rae-Grant NI, Links PS, Cadman DT, et al. Ontario Child Health Study. II. Six-month prevalence of disorder and rates of service utilization. Arch Gen Psychiatry. 1987; 44(9):832–836. [PubMed: 3498458]

Paris J. Childhood precursors of borderline personality disorder. Psychiatric Clinics of North America. 2000; 23:77–88. [PubMed: 10729932]

Philipsen A. Differential diagnosis and comorbidity of attention-deficit/hyperactivity disorder (ADHD) and borderline personality disorder (BPD) in adults. European Archives of Psychiatry and Clinical Neuroscience. 2006; 256(Suppl1):42–46.

Robins LN, Helzer JE. The Diagnostic Interview Schedule: Its development, evolution, and use. Social Psychiatry and Psychiatric Epidemiology. 1988; 23:6–16. [PubMed: 3130671]

Rowe R, Costello EJ, Angold A, Copeland WE. Developmental pathways in oppositional defiant disorder and conduct disorder. Journal of Abnormal Psychology. 2010; 119:726–738. [PubMed: 21090876]

Rowe R, Maughan B, Pickles A, Costello EJ, Angold A. The relationship between DSM-IV oppositional defiant disorder and conduct disorder: findings from the Great Smoky Mountains Study. J Child Psychol Psychiatry. 2002; 43(3):365–373. [PubMed: 11944878]

Shiner RL. The development of personality disorders: perspectives from normal personality development in childhood and adolescence. Dev Psychopathol. 2009; 21(3):715–734. [PubMed: 19583881]

Skodol AE, Gunderson JG, McGlashan TH, Dyck IR, Stout RL, Bender DS, Grilo CM, Shea T, Zanarini MC, Morey LC, Sanislow CA, Oldham JM. Functional impairment in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder. Am J Psychiatry. 2002; 159(2):276–283. [PubMed: 11823271]

Skodol AE, Pagano ME, Bender DS, Shea MT, Gunderson JG, Yen S, Stout RL, Morey LC, Sanislow CA, Grilo CM, Zanarini MC, McGlashan TH. Stability of functional impairment in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder over two years. Psychological Medicine. 2005; 35(3):443–451. [PubMed: 15841879]

Stringaris A, Goodman R. Three dimensions of oppositionality in youth. Journal of Child Psychology and Psychiatry and Allied Disciplines. 2009; 50:216–233.

Stringaris A, Cohen P, Pine DS, Leibenluft E. Adult outcomes of youth irritability: a 20-year prospective community-based study. Am J Psychiatry. 2009; 166(9):1048–1054. [PubMed: 19570932]

Taylor TK, Burns GL, Rusby J, Foster EM. Oppositional defiant disorder toward adults and oppositional defiant disorder toward peers: Initial evidence for two separate constructs. Psychological Assessment. 2006; 18:439–443. [PubMed: 17154765]

Zanarini MC, Frankenburg FR, Dubo ED, Sickel AE, Trikha A, Levin A, Reynolds V. Axis II comorbidity of borderline personality disorder. Compr Psychiatry. 1998; 39(5):296–302. [PubMed: 9777282]

Zanarini MC, Frankenburg FR, Reich DB, Marino MF, Haynes MC, Gunderson JG. Violence in the lives of adult borderline patients. J Nerv Ment Dis. 1999; 187(2):65–71. [PubMed: 10067945]

Table 1

Descriptive statistics of study constructs at baseline.

Variable	Mean	s.d.	Min	Max
Oppositional defiant disorder	5.01	2.26	0	8
Conduct disorder	1.29	1.40	0	6
Attention deficit hyperactivity disorder	8.15	3.47	0	14
Depression	1.44	1.86	0	8
Anxiety	3.19	2.84	0	12
Alcohol *	2.94	9.64	0	102
Tobacco *	23.16	85.44	0	365
Marijuana *	0.58	4.52	0	51
Hard drugs *	0.51	5.38	0	70
Maternal age	24.76	5.11	15	38
Siblings	1.19	0.91	0	5
SES	35.61	13.76	11	66
BPD symptoms **	1.20	1.9	0	9
APD symptoms **	2.05	1.57	0	6
Other Cluster B symptoms **	2.54	3.70	0	18

Note:

* = first assessed in study year 3,

** = assessed at age 24, BPD = borderline personality disorder,

APD = antisocial personality disorder

Table 2

Correlations among psychopathology predictors

	ODD	CD	ADHD	Depression
CD	0.47			
ADHD	0.53	0.32		
Depression	0.28	0.24	0.19	
Anxiety	0.37	0.29	0.35	0.41

Note: These correlations are estimations of the zero-order relationship between each pair of variables accounting for the correlated observations within the panel data set. ODD = oppositional defiant disorder; CD = conduct disorder; ADHD = attention deficit-hyperactivity disorder.

Table 3

Child and adolescent psychopathology symptom counts predicting young adult BPD symptoms.

	IRR	se	p	95% Confidence Interval	
ODD	1.11	.06	.05	1.00	1.22
CD	0.96	.06	.47	0.85	1.07
Depression	0.96	.03	.14	0.90	1.01
Anxiety	1.03	.03	.40	0.97	1.09
ADHD	1.08	.03	.003	1.3	1.13

Table 4

Final Model of Predictors of BPD symptoms.

	IRR	Se	P	95% Confidence Interval	
ODD Oppositional Behavior	1.16	.08	.03	1.02	1.33
APD	1.28	.08	<.001	1.13	1.44
Other Personality Disorders	1.18	.03	<.001	1.13	1.24
ADHD	1.04	.03	.09	0.99	1.09
Marijuana	1.00	.001	.71	.99	1.00
Age	1.03	.025	.160	.99	1.08