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Childhood CBCL Bipolar Profile and Adolescent/Young Adult Personality Disorders: A 9-year Follow-up

Jeffrey M. Halperin[1,2,3], **Julia J. Rucklidge**[4], **Robyn L. Powers**[2], **Carlin J. Miller**[5], and **Jeffrey H. Newcorn**[3]

[1]Department of Psychology, Queens College of the City University of New York, NY
[2]Neuropsychology Doctoral Program, The Graduate Center of the City University of New York, New York, NY [3]Division of Child and Adolescent Psychiatry, Mount Sinai Medical Center, New York, NY [4]Department of Psychology, University of Canterbury, Christchurch, New Zealand
[5]Department of Psychology, University of Windsor, Windsor, Ontario, Canada

Abstract

Background—To assess the late adolescent psychiatric outcomes associated with a positive Child Behavior Checklist – Juvenile Bipolar Disorder Phenotype (CBCL-JBD) in children diagnosed with ADHD and followed over a 9-year period.

Methods—Parents of 152 children diagnosed as ADHD (ages 7–11 years) completed the CBCL. Ninety of these parents completed it again 9 years later as part of a comprehensive evaluation of Axis I and II diagnoses as assessed using semi-structured interviews. As previously proposed, the CBCL-JBD phenotype was defined as T-scores of 70 or greater on the Attention Problems, Aggression, *and* Anxiety/Depression subscales.

Results—The CBCL-JBD phenotype was found in 31% of those followed but only 4.9% of the sample continued to meet the phenotype criteria at follow up. Only two of the sample developed Bipolar Disorder by late adolescence and only one of those had the CBCL-JBD profile in childhood. The proxy did not predict any Axis I disorders. However, the CBCL-JBD proxy was highly predictive of later personality disorders.

Corresponding author: Jeffrey M. Halperin, Ph.D., Department of Psychology, Queens College, CUNY, 65-30 Kissena Blvd., Flushing, NY 11367, Telephone: (718) 997-3254, Fax: (718) 997-3218, jeffirey.halperin@qc.cuny.edu.

Conflicts of Interest

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Contributors

Dr. Halperin provided overall guidance in designing the study and data acquisition, wrote the grants that supported the research, oversaw the data analysis, and wrote substantial portions of the manuscript. Dr. Rucklidge wrote substantial portions of the manuscript and contributed to the conceptual basis of the study. Ms. Powers assisted with data analysis and participated in substantial portions of the data collection. Dr. Miller conducted and/or supervised most of clinical evaluations and organized the personality disorder component of the data set. Dr. Newcorn provided critical input to all aspects of this research including preparation of the grants funding the research, design of the study, and preparation of the manuscript. All authors contributed to and have approved the final manuscript.

Limitations—Only a subgroup of the original childhood sample was followed. Given this sample was confined to children with ADHD, it is not known whether the prediction of personality disorders from CBCL scores would generalize to a wider community or clinical population

Conclusions—A positive CBCL-JBD phenotype profile in childhood does not predict Axis I Disorders in late adolescence; however, it may be prognostic of the emergence of personality disorders.

Keywords

CBCL-JBD; pediatric bipolar disorder; ADHD; predictive utility; personality disorders

Introduction

The diagnosis of pediatric or juvenile bipolar disorder (JBD) is among the most controversial and contentiously debated diagnoses in modern child psychiatry (Carlson et al., 2003). Disagreement exists in how to define it, at what age to identify it, and whether it is equivalent or related to the more traditional diagnosis of bipolar disorder in adulthood. Variability in how to diagnose JBD has added to the controversy in that some researchers have proposed that the diagnosis in children should include the presence of affective storms, irritability, and rapid, ultrarapid, and ultradian cycling as opposed to episodic states (Hechtman and Greenfield, 1997; NIMH, 2001). Liebenluft and colleagues (2003) suggest using a phenotypic system for classifying juvenile mania consisting of a narrow phenotype (somewhat narrower than the DSM criteria), two intermediate phenotypes, and a broad phenotype (including those with greater variability in symptoms) in order to encourage consistency and ease for comparisons across studies. Others have suggested using the term JBD-NOS to identify children with variable manic-like presentations (NIMH, 2001). Along with the expanded criteria for JBD, over the last decade, psychometric instruments have been proposed to assist with differential diagnoses (see Youngstrom and Duax, 2005; Youngstrom and Youngstrom, 2005 for excellent reviews). In particular, an algorithm from the Child Behavior Checklist (CBCL; Achenbach, 1991) has been proposed as a useful diagnostic tool. Biederman and colleagues (1995) found excellent convergence between the CBCL subscales of Delinquent Behavior, Aggressive Behavior, Somatic Complaints, Anxious/Depressed, and Thought Problems and the diagnosis of mania in children. Since this publication, there has been a growing interest in the diagnostic utility and discriminant validity of the CBCL in identifying what has been coined the “CBCL-JBD phenotype”. The CBCL-JBD phenotype is defined by a profile of T-scores above 70 on the Anxious/Depressed, Aggression, and Attention Problems subscales. This profile has been found to be common in individuals diagnosed as JBD based on semi-structured interviews (Mick et al., 2003) and Biederman et al, based on 7 year longitudinal data, reported that the phenotype predicts JBD and its associated impairments (Biederman et al., 2009). Moreover, it has been suggested that this profile can distinguish youth with the CBCL –JBD phenotype from those with ADHD only, suggesting discriminant validity of the measure. Faraone et al (2005), using a sample of 243 ADHD probands, 229 non-ADHD controls, and their 410 siblings, used ROC analyses to determine that the CBCL-JBD phenotype was a highly efficient way to identify subjects with a current diagnosis of JBD. Hudziak et al (2005) estimated that in their sample of Dutch twins, the prevalence of this phenotype occurs in approximately 1% of children of all ages sampled (7, 10 & 12 years). When a Latent class analysis (LCA) approach was used, 4% of girls and 5% of boys were found to be consistent with this phenotype (Althoff et al., 2006). This research group has also shown stability of the phenotype across time (Boomsma et al., 2006), with genetic factors accounting for the majority of the variance in stability (Boomsma et al., 2006). Although the size of the Dutch sample is impressive, there are no data as to whether the children who have the CBCL-JBD

phenotype actually meet criteria for JBD, or subsequently develop bipolar disorder or other psychiatric disorders later in life.

The utility of the CBCL in the clinical identification of mania and discrimination of children with JBD and ADHD has also been questioned. For example, Youngstrom et al (2005) found that in their low-income community sample of 3086 archival cases, the CBCL was not a good indicator of JBD. Using logistic regression, they found that elevated scores on the Externalizing problems scale of the CBCL provided the best indicator of bipolar cases, with no other scale or combination of subscales accounting for additional significant variance. Although their CBCL profiles were similar to those of other research groups, there was also considerable variability in the profile within their JBD sample. Further, a high percentage of the profiles among those with other disorders (e.g., ADHD, ODD, CD without BD) also had the CBCL-JBD profile.

In a sample of 1346 twins, Volk and Todd (2007) found that among those who had the CBCL-JBD phenotype, none were diagnosed with JBD. Rather, the phenotype more closely matched those diagnosed with ADHD. Nevertheless, it was associated with more frequently endorsed suicidal behaviors. Likewise, Kahana et al (2003) found that youth with JBD showed elevations on several CBCL subscales (i.e., Delinquent, Aggressive, Withdrawn, & Anxious/Depressed) as compared with children who had ADHD, and concluded that the diagnostic utility of the CBCL for identifying JBD was limited. Similarly, Diler and colleagues (2008) reported that children with elevations on the Young Mania Rating Scale had elevations on *all* CBCL subscales, including the proposed CBCL-JBD subscales. However, only the Thought Problems subscale showed good predictability of mania symptoms. Consistent with these findings, Ayer et al (2009) have also suggested, based on a broad nationally representative sample of 2029 children, that the CBCL-JBD appears related to severe psychopathology but not to one specific syndrome.

Furthermore, even when the CBCL appears to discriminate between JBD and other samples, the pattern of elevations is not always consistent. For example, Geller et al (1998) and Biederman et al (1995) found significant differences between their JBD and ADHD groups on almost all of the CBCL subscales. Hazell et al (1999) showed that boys with both JBD and ADHD had higher mean ratings on some of the CBCL subscales as compared with boys with only ADHD; however, it was not on the subscales consistent with the CBCL-JBD phenotype (Withdrawn, Thought Problems, Delinquent Behaviors, and Aggressive Behaviors). Similarly, Dienes et al (2002), using the WASHU-K-SADS as the diagnostic tool, determined that their sample of children of bipolar parents differed from the ADHD group, not on the CBCL-JBD phenotype, but rather on the Aggressive Behaviors, Withdrawn, and Anxious/Depressed subscales. A Brazilian sample ascertained from a chart review of patients under 15 found that those CBCL subscales that showed differences between those diagnosed with JBD and those diagnosed with ADHD were the Anxious/Depressed, Delinquent Problems, and Aggressive Behavior subscales (Tramontina et al., 2003). Diler and colleagues (2007) found that their ADHD+JBD group was different from an ADHD group on the Anxious/Depressed, Social Problems, Thought Problems, and Aggression subscales. Thus, although elevations on the CBCL are common findings in JBD research, the specificity is questionable.

Based on these disparities in results, it seems unlikely that the CBCL-JBD phenotype is an accurate or specific indicator of JBD although it may depend on how JBD is defined. It appears that the more consistent the criteria are with narrower definitions of JBD phenotype, the less useful the CBCL-JBD phenotype is for diagnosis. Nevertheless, the CBCL-JBD phenotype does seem to identify a subgroup of children with severe psychopathology characterized by a mixture of externalizing and internalizing difficulties. Yet, it remains

unclear exactly who these children are, and perhaps more importantly, what is the clinical significance of such a profile. Among children with ADHD in the MTA Study, those with and without the CBCL-JBD phenotype did not differ in response to treatment with stimulant medication (Galanter et al. 2003). Yet, it is possible that the presence of the CBCL-JBD phenotype during childhood predicts some unique pattern of Axis I and/or Axis II morbidities in adulthood. Indeed, secondary data analyses from a longitudinal cohort conducted by Meyer et al (2009) reported that non-referred youth at risk for major depression who also displayed the CBCL-JBD phenotype, were at greater risk for ongoing comorbidity and impairment across a range of areas of functioning including cluster B personality disorders; however, the phenotype was not predictive of specific DSM disorders. Similarly, a 6-year follow-up study of children with ADHD found no differences in rates of Axis I or Axis II disorders between those with and without manic symptoms in childhood, although those with childhood manic symptoms were judged to have poorer global functioning at outcome (Hazell et al. 2003). No other studies have investigated the role that the phenotype might play in predicting Axis II psychopathology and it is quite possible that the specific nature of the sample in which the CBCL-JBD is being examined has an impact on findings.

We sought to expand the literature by investigating the long-term outcomes of children with ADHD who did and did not have CBCL-JBD phenotype by assessing both Axis I and Axis II disorders. This study identified individuals with the CBCL-JBD phenotype in a clinically-referred longitudinal sample of children with ADHD who were reevaluated 9 years later using the CBCL and semi-structured interviews which generated both Axis I and Axis II diagnoses at outcome. The primary aims were to determine the degree to which the CBCL-JBD phenotype 1) represents a temporally stable trait from childhood through late adolescence/early adulthood, 2) is predictive of later Axis I disorders, and 3) is predictive of the emergence of personality disorders during late adolescence/early adulthood.

Method

Participants

As part of their participation in a research protocol focusing on biological correlates of ADHD that was conducted between 1990 and 1997, 152 children (133 male; 87.5%), ranging in age from 7 – 11 years (mean = 9.03; SD = 1.31), were rated by their parents using the Child Behavior Checklist (CBCL; Achenbach, 1991) and their teachers using the IOWA Conners (Loney and Milich, 1982). In addition, parents were interviewed using the Diagnostic Interview Schedule for Children (DISC; Costello et al., 1985). Among these children, whose ascertainment is described in detail in publications based exclusively on childhood characteristics (Halperin et al., 2003; Halperin et al., 1994), all met diagnostic criteria for ADHD. In addition, 76 had comorbid oppositional-defiant disorder (ODD; 50%), 46 (30.3%) met criteria for comorbid conduct disorder (CD); 46 (30.3%) met criteria for an anxiety disorder, and 18 (11.8%) met criteria for a comorbid mood disorder, only one of whom had bipolar disorder as defined by DSM-IV. The sample was ethnically-diverse, consisting of 33 (21.7%) Caucasians, 39 (25.7%) African-Americans, 63 (41.4%) Hispanics, and 17 (11.2%) children of mixed or other ethnicity. Overall, the sample was urban and of lower to middle class socioeconomic status, with a substantial proportion of the families at or below the poverty line.

Among these original participants, 90 (59.2%) participated in a follow-up evaluation on average 9.27 years later (SD = 1.75 yrs.; range = 6.30 – 15.38 yrs.) when they had a mean (SD; range) age of 18.36 (1.80; 15.55 – 26.29) years. Those who were and were not assessed at follow-up did not differ significantly with regard to age at initial evaluation, sex (87.1% vs. 87.8% male), rates of childhood comorbid diagnoses, any CBCL subscale or the

combined CBCL-JBD score, or teacher-rated IOWA Conners score at initial assessment (all $p > .10$). Notably, however, those who participated in the follow-up were from a somewhat higher socioeconomic strata (Nakao and Treas, 1994) during childhood ($p = .04$). Childhood characteristics of those who were and were not followed are presented in Table 1.

All procedures were approved by the Institutional Review Boards of the participating institutions. Written informed consent was obtained from participants above 18 years-old and the parents of those under 18 years; assent was obtained from youth under 18 years-old.

Follow-up Assessment Measures

At follow-up, participants received a comprehensive psychiatric evaluation which included, in most cases, parent and self-reports on the 2001 version of the CBCL (n = 81) and Youth Self-report (YSR; n = 76), respectively (Achenbach & Rescoria, 2001). To determine current psychiatric diagnoses at follow-up, both parents and the youths were interviewed by either Ph.D.-level psychologists or by trained graduate students. The Kiddie-SADS-PL (Kaufman et al., 1996) was used to make Axis-I diagnostic determinations and the SCID-II (First et al., 1997) for the determination of Axis-II personality disorders as defined by DSM-IV. Diagnoses were formulated by combining parent and youth reports following a case presentation to the research team which included a licensed psychologist and a board certified child psychiatrist. The research team was blind to all childhood data at the time of diagnostic formulation.

Data Analysis

A “bipolar proxy” dimension was calculated for each participant by summing the Attention Problems, Aggression, and Anxiety/Depression T-scores from his/her parents’ rating on the CBCL at baseline. To determine the degree to which this proxy score represents a temporally stable trait, Spearman rho correlations, which do not assume a normal distribution of scores, were calculated to examine its relation to analogous proxy scores derived from the CBCL and YSR scores obtained at follow-up.

The predictive utility of bipolar proxy scores for psychopathology at follow-up was assessed dimensionally using separate logistic regression models in which the proxy score served as the predictor variable and the Axis I and Axis II diagnoses served as the dependent measures. Subsequently, a series of logistic regressions were performed, in which the predictor variable (bipolar proxy score) was dichotomized to establish groups of children who did and did not meet criteria for the “CBCL-JBD phenotype” (i.e., T-scores on the Attention Problems, Aggression, and Anxiety/Depression scales equal to or greater than 70). Adolescent/young adult diagnoses again served as the dependent measures. Due to the relatively low base rates of individual personality disorders at follow-up, all analyses of Axis II disorders focused on clusters, as defined in DSM-IV (i.e., A, B, and C), and the presence of “Any personality disorder.” The magnitude of the association between the bipolar proxy classification and diagnosis (i.e., effect size) was determined by transforming the absolute value of the standardized regression coefficient (β) for each factor into an odds ratio (OR).

Results

Stability of Proxy Scores and Relation to Childhood Comorbidity

Not surprisingly, bipolar proxy scores as ascertained via parent CBCL and youth YSR reports at follow-up were significantly lower than those derived from parent ratings in childhood (both $t > 7.50$, $p < .001$). This decrease in severity was not specific to the CBCL-JBD; almost all CBCL and YSR scores reported in adolescence were significantly lower

than those reported by parents in childhood. In childhood, 31% of the children in the sample met the criterion of T-scores of 70 or greater on the Attention Problems, Aggression, *and* Anxiety/Depression subscales as rated by parents on the CBCL. Notably, meeting this threshold was significantly associated with higher rates of ODD ($p = .002$), CD ($p = .002$), mood ($p < .001$) and anxiety ($p = .008$) disorders in childhood. At follow-up far fewer youth met criteria for a positive CBCL-JBD; 4.9% (4/81) based on parent report and 5.3% (4/76) based on self-report. Dimensional proxy scores derived from parent ($r_s = 0.325$, $p = .003$) but not youth ($r_s = 0.147$, $p > .10$), reported 9 years later, were significantly correlated with those ascertained in childhood. Among the seven individuals who met the categorical T-score cut-off at outcome (3 by parent-report; 3 by self-report; 1 by both), four (57%) were similarly classified in childhood ($\chi^2 = 2.45$, $p = .12$).

Predictive Utility

Axis I Diagnoses—Logistic regression analyses assessing the predictive validity of the bipolar proxy, as assessed during childhood, were conducted using the presence of persisting ADHD diagnosis, CD/ASPD, a mood disorder, an anxiety disorder and a substance use disorder (SUD) as the dependent measures. Irrespective of whether the bipolar proxy was characterized as a dimensional variable or a categorical variable, the childhood measure did not significantly predict the presence of any Axis I disorder (all $p > .20$). Further, there was no significant difference in the mean number of Axis 1 diagnoses at outcome in individuals who did (mean = 1.79, SD = 1.45) and did not (mean = 1.66, SD = 1.33) meet criteria for the bipolar proxy in childhood ($t(88) = 0.40$, $p > .10$). Analyses were not conducted specifically using mania or bipolar disorder at outcome because only two participants met criteria (one bipolar II; one bipolar NOS). One of these two participants met criteria for the CBCL-JBD phenotype in childhood. Table 2 shows the relations between the CBCL-JBD phenotype and Axis I disorders 9 years later.

Axis II Diagnoses—A clear pattern emerged with regard to Axis II disorders in adolescence/young adulthood. In particular, childhood bipolar proxy scores were significantly predictive of the presence of a Cluster C personality disorder irrespective of whether the predictor variable was dimensional or categorical. In addition, there was a trend ($p < .10$) such that higher bipolar proxy scores during childhood were associated with elevated rates of Cluster B diagnoses at follow-up. Table 3 shows the relations between the categorical (yes/no) CBCL-JBD phenotype and Axis II Personality Disorders 9 years later. Overall, the CBCL-JBD phenotype in childhood was associated with not only increased likelihood of having a personality disorder at outcome, but also with the number of personality disorders present at follow-up ($t = 3.11$, $p = .003$).

The predictive utility of the CBCL-JBD phenotype for identifying individual children with ADHD who are at greatest risk for developing later personality disorders was assessed further by examining indices of sensitivity, specificity, positive (PPP) and negative (NPP) predictive power. These later analyses, which are presented in Table 4, were limited to Cluster C and the presence of “Any Personality Disorder,” both of which demonstrated significant associations with the childhood CBCL-JBD phenotype (see Table 3). Sensitivity values (the proportion with the disorder that was correctly identified) are generally modest, although 2/3 of those who went on to develop Cluster C disorders did have the CBCL-JBD phenotype in childhood (sensitivity = .67). Perhaps more notable is that 2/3 of those with the CBCL-JBD phenotype in childhood went on to develop at least one personality disorder (PPP = .68) and 2/3 of those without the CBCL-JBD phenotype in childhood did not develop a personality disorder 9 years later (NPP = .65), although it should be noted that indices of PPP and NPP are closely linked to base rates of the disorders in the sample.

Discussion

Our aim was to study the predictive utility of the CBCL-JBD phenotype (defined as T scores>70 on the Attention, Aggression and Anxiety/depression subscales) in a sample of children identified in pre-adolescence with ADHD. These children were assessed again nearly 10 years later across Axis I and II diagnoses. In this sample, we found that while a third of the sample had the CBCL-JBD phenotype in pre-adolescence, only one of them met criteria for the DSM-IV definition of JBD as assessed at that same time point. Further, the number of youth meeting criteria for the CBCL-JBD phenotype dropped substantially at follow-up and only one of these individuals developed Bipolar Disorder by late adolescence, indicating that this phenotype shows poor predictive utility for Bipolar Disorder. It was also not predictive of any Axis I disorder, and this does not seem to be due to limited power as effects sizes, as measured by odds ratios, were all quite low (all Axis I OR < 2.0). These findings are similar to those reported by Hazell et al. (2003) who also found a reduction in CBCL scores at follow-up and a lack of predictive utility for later Axis I disorders. However, unlike Hazell et al. (2003), we found that the profile was more likely to be found within those who went on to develop one or more personality disorders, most particularly those with Cluster C, suggesting the CBCL-JBD phenotype may be identifying a trait that is relatively stable, even though it seems not to be Bipolar Disorder.

These findings are consistent with the results of other studies investigating the CBCL-JBD phenotype (Volk and Todd, 2007; Youngstrom et al., 2005) which indicate that the phenotype is not specific to Bipolar Disorder. The fact that nearly a third of our childhood sample met criteria for the CBCL-JBD phenotype, but almost none met DSM-IV criteria for JBD, suggests that the CBCL-JBD phenotype should be used very cautiously, if at all, for screening for mania in childhood (Biederman et al., 1995; Faraone et al., 2005). Further, given, in our sample, the high rate of having the CBCL-JBD phenotype during childhood, but the low rate of Bipolar Disorder at outcome, our findings are not consistent with 7 year longitudinal data indicating that the CBCL-JBD phenotype predicts subsequent Bipolar Disorder (Biederman et al., 2009). We found the diagnostic accuracy of the CBCL-JBD phenotype to be low for Axis I disorder outcomes and, like Meyer et al (2009) and Ayer et al (2009), we did not find that it maps onto specific DSM-IV classifications. Interestingly, Meyer et al found that the CBCL-JBD phenotype identified children at risk for long term emotional and ongoing psychiatric symptomatology in a 23 year follow-up study of children at risk for mood disorders. We found similar risk associated with the phenotype despite having investigated a very different sample. The high rate of personality disorders (45.6%) in this sample identified at follow-up is also consistent with the literature. For example, Rey and colleagues found that 36% of their sample of ADHD children had a Cluster B personality disorder in young adulthood, a rate similar to the 30% we found in our sample. Miller and colleagues (2007) found that ADHD was associated with an excess of both Cluster B (20.3%–47.4%) and C (15.8%–23.4%) personality disorder in their sample of adults with ADHD. Consistent with this finding, one retrospective study examining the question of whether patients with Borderline Personality Disorder have a retrospective history of ADHD found a positive association in 60% of cases (Fossati et al., 2002).

It is possible that the CBCL-JBD phenotype may be identifying a temperamental profile predictive of later personality disorder as well as on-going psychopathology. Despite the low rate of persistence of the categorically-defined phenotype, our results did show a positive correlation across the two assessment periods over 9 years apart, suggesting some stability of the phenotype over time. Moreover, two thirds of those in our sample who met criteria for the CBCL-JBD phenotype went on to meet criteria for at least one personality disorder by late adolescence/early adulthood. Consistent with Ayer et al (2009), the symptoms endorsed in childhood may simply be reflecting an ongoing and stable pattern of difficulty regulating

emotion, aggressive impulses, and other impulsive drives. However, despite the increased odd's ratios for those with a positive CBCL-JBD phenotype in those with personality disorders, the phenotype is at best a marker for those at increased risk.

Our results must be interpreted in the context of several limitations. First, and most importantly, a substantial subgroup of our original childhood sample (41%) was lost to follow-up. Although those who did and did not participate in the follow-up were not significantly different on any of the key childhood clinical measures, they did differ somewhat in socioeconomic status. As such, those who participated in the follow-up study may not have been fully representative of the childhood sample. Importantly, however, those who were and were not included in the follow-up study did not differ in the childhood CBCL-JBD profile score ($p > .40$). Thus, we believe that the results, which focus on the relations between the childhood CBCL-JBD profile and psychiatric outcomes that were derived from the followed subsample, are likely to be representative of what would have been obtained had the larger sample been successfully followed. Nevertheless, we cannot rule-out the possibility that the difference in childhood SES is in some way influencing our results or that our limited sample size resulted in some failures to detect real associations.

The original sample was assessed in the early 1990s, before the diagnosis of Bipolar Disorder NOS was fully conceptualized and more frequently used (NIMH, 2001). As such, we may have had children with this mood presentation, but that it was not identified. However, Bipolar Disorder was considered as a possible diagnosis during the follow-up assessment, and as such, we presumably would have captured it at this time point if it were present. Notably, our rates of Bipolar Disorder at follow-up are comparable to those reported in some longitudinal studies of ADHD (Barkley et al., 2008; Biederman et al., 2006a; Weiss et al., 1985), but not all (Biederman et al., 2006b; Tillman & Geller 2006), and the rates are not consistent with those reported as part of the National Comorbidity Survey (Kessler et al., 2006). It is possible that youth in our sample with vulnerability for Bipolar Disorder might simply not have expressed it, since this disorder can first appear in early adulthood or even later. Further follow up investigation would be required to eliminate this possibility.

In addition to the above limitations, the low base rate of JBD in our sample limits our ability to draw conclusions about how prominent the CBCL-JBD phenotype is within a sample that is either bipolar at initial assessment or at follow-up. It is also possible that, since the CBCL was completed during an assessment for ADHD, the responses of the parents may have focused primarily on levels of distress associated with parenting a child with ADHD symptoms, and minimized other problems.

Further, the fact that our participants were selected for the presence of ADHD implies that we cannot generalize these results to a wider community population, thereby limiting the external validity of the findings. It is possible that the high rate of personality disorders being identified in those with the CBCL-JBD phenotype is more specific to the subsample of youth with ADHD than it would be in the wider clinical population. However, the nature of our sample can also be seen as a strength as it enabled us to test the specificity of the proposed phenotype in a group of children not specifically recruited for JBD. It is highly likely that most children meeting criteria for the CBCL-JBD phenotype, which requires a CBCL Attention Problems score of greater than 70, will meet criteria for ADHD irrespective of the presence of JBD.

Finally, it is important to comment on the practice of diagnosing personality disorders in adolescents. As indicated in DSM-IV, personality disorders commonly emerge during adolescence, but by definition, the traits must be stable, of long duration and enduring. As such, while personality disorders can be diagnosed in children and adolescents (50% of our

sample was below the age of 18 years), traits identified in youth may not persist unchanged into adult life. Nevertheless, we applied strict DSM-IV criteria to ensure that in all cases the features were present for at least one year and not better accounted for by the presence of an Axis I disorder.

Despite these limitations, there are several clear conclusions that can be drawn from this study. First, meeting criteria for the CBCL-JBD phenotype is not uncommon among clinically-referred children with ADHD. Second, the CBCL-JBD phenotype in childhood does not predict the emergence of Bipolar Disorder or any axis I disorder by late adolescence in an ADHD sample. Third, despite points one and two above, the CBCL-JBD phenotype seems to be clinically meaningful, as it appears to portend risk for other psychiatric disorders, specifically personality disorders. As such, consistent with Ayer et al's (2009) conclusions, the CBCL-JBD phenotype may be a useful prognostic indicator that is merely in need of a name change.

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Table 1

Comparison of Baseline Status between Subjects Followed and Not Followed

	Followed N=90		Not Followed N=62	
	Mean	**SD**	**Mean**	**SD**
Age at initial assessment (in years)	9.1	1.3	8.9	1.3
Socioeconomic status in childhood*	36.3	17.8	30.1	13.5
Teacher IOWA Conners Inattention/Overactivity	11.3	3.2	10.7	3.0
Teacher IOWA Conners Oppositional/Defiant	8.1	4.7	8.1	4.5
Parent Ratings – CBCL T-scores				
Externalizing	69.4	11.4	67.4	10.2
Internalizing	65.2	11.9	64.2	9.8
Withdrawn	61.7	10.8	62.1	10.3
Somatic	60.8	9.5	59.1	8.9
Anxiety/Depression	66.8	12.2	64.7	10.3
Social Problems	69.0	10.9	67.5	10.8
Thought Problems	62.4	11.0	64.7	11.3
Attention Problems	72.3	10.0	73.4	8.2
Delinquency	67.8	9.6	65.8	8.4
Aggression	71.6	13.7	69.0	12.7
CBCL-JBD	210.7	32.1	207.1	25.5
Comorbid Diagnosis**	**%**	**#**	**%**	**#**
ODD (no CD)	51.1	46	48.4	30
CD	28.9	26	32.3	20
Anxiety disorder	27.8	25	33.9	21
Mood disorder	8.9	8	16.1	10

* p = .04. All other p-values > .10.

** Analyzed using Chi Square.

Table 2

Summary statistics from logistic regressions examining Axis I Psychiatric Outcomes in ADHD youth with and without the "CBCL-JBD phenotype"

	CBCL-JBD positive N = 28		**CBCL-JBD Negative N = 62**		***p***	**Wald**	**Odds Ratio**	**95% CI**
	%	**#**	**%**	**#**				
ADHD	42.9	12	44.3	27	.90	0.02	1.06	0.43–2.61
CD/ASPD	35.7	10	35.5	22	.98	0.00	1.01	0.40–2.57
Mood Disorder	25.0	7	14.5	9	.23	1.42	1.96	0.65–5.95
Anxiety Disorder	32.1	9	22.6	14	.34	0.92	1.62	0.60–4.39
Substance Abuse/Dependence†	42.9	12	50.8	31	.90	0.02	0.73	0.30–1.79

†Substance abuse data unavailable for one participant

Table 3

Summary statistics from logistic regressions examining Axis II Personality Disorder (PD) Outcomes in ADHD youth with and without the "CBCL-JBD phenotype"

	CBCL-JBD positive N = 28		CBCL-JBD Negative N = 62		*P*	Wald/ χ^2	Odds Ratio	95% CI
	%	#	%	#				
Any Cluster A	25.0	7	12.9	8	.161	1.97	2.25	0.73–6.98
Any Cluster B	42.9	12	24.2	15	.077	3.12	2.35	0.91–6.06
Any Cluster C	35.7	10	8.1	5	.003	9.13	6.33	1.91–20.97
Any PD	67.9	19	35.5	22	.005	7.73	3.84	1.49–9.91

Table 4

Predictive utility of the CBCL-JBD phenotype for diagnosing later personality disorders as measured by sensitivity, specificity, positive and negative predictive power

	Sensitivity	Specificity	Positive Predictive Power	Negative Predictive Power
Any Cluster C	.67	.76	.36	.92
Any Personality Disorder	.47	.82	.68	.65