Published in final edited form as:

Psychol Assess. 2010 December ; 22(4): 816–826. doi:10.1037/a0020975.

The Structure of Childhood Disruptive Behaviors

Michelle M. Martel,

Department of Psychology, University of New Orleans

Monica Gremillion,

Department of Psychology, University of New Orleans

Bethan Roberts,

Department of Psychology, University of New Orleans

Alexander von Eye, and

Psychology Department, Michigan State University

Joel T. Nigg

Department of Psychiatry, Oregon Health and Sciences University

Abstract

Attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) frequently co-occur. Comorbidity of these 2 childhood disruptive behavior domains has not been satisfactorily explained at either a structural or etiological level. The current study evaluated a bifactor model, which allows for a “g” factor in addition to distinct component factors, in relation to other models to improve understanding of the structural relationship between ADHD and ODD. Participants were 548 children (321 boys, 227 girls) between the ages of 6 years and 18 years who participated in a comprehensive diagnostic assessment incorporating parent and teacher ratings of symptoms. Of these 548 children, 153 children were diagnosed with ADHD (without ODD), 114 children were diagnosed with ADHD + ODD, 26 children were diagnosed with ODD (without ADHD), and 239 children were classified as non-ADHD/ODD comparison children (including subthreshold cases). ADHD symptoms were assessed via parent report on a diagnostic interview and via parent and teacher report on the ADHD Rating Scale. ODD symptoms were assessed via teacher report. A bifactor model of disruptive behavior, comprising a “g” factor and the specific factors of ADHD and ODD, exhibited best fit, compared to 1-factor, 2-factor, 3-factor, and 2nd-order factor models of disruptive behaviors. It is concluded that a bifactor model of childhood disruptive behaviors is superior to existing models and may help explain common patterns of comorbidity between ADHD and ODD.

Keywords

disruptive behavior; ADHD; ODD; bifactor model

Attention-deficit/hyperactivity disorder (ADHD) and oppositional-defiant disorder (ODD) are common childhood disruptive behavior (DB) disorders that co-occur in nearly 50% of diagnosed cases (Angold, Costello, & Erkanli, 1999; Jensen, Martin, & Cantwell, 1997; Nock, Kazdin, Hiripi, & Kessler, 2007). As defined by the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev., *DSM-IV-TR*; American Psychiatric Association [APA], 2000), ADHD is characterized by behavioral symptoms of inattention and hyperactivity-impulsivity. ODD is characterized by negativistic interactions with others, including behavioral symptoms of opposition and defiance (APA, 2000). Whereas the initial partial distinction between conduct problems/aggression and hyperactivity/inattention was established by seminal reviews over 2 decades ago (Hinshaw, 1987), the relations between ODD and ADHD remain poorly described, and the reason for their extremely high co-occurrence is still debated (Connor & Doerfler, 2008; Jensen et al., 1997). Clarifying the relation between ADHD and ODD is important for improving specificity of diagnostic assessment and treatment protocols and has particular relevance for improving developmental outcomes, since children with ADHD + ODD are often more impaired than children with either disorder alone (Biederman et al., 2008; Connor & Doerfler, 2008; Gadow & Nolan, 2002).

Whereas the current report focuses on ADHD and ODD, the structure of childhood DBs received much attention before, during, and shortly following the publication of the *DSM-IV-TR*, with relatively less evaluation recently despite the pending revision of the *DSM*. Factor analyses, which were in many cases confirmatory, verified a two-, three-, or four-factor structure of childhood DB (Burns, Boe, Walsh, Sommers-Flanagan, & Teegarden, 2001; Burns, Walsh, Owen, & Snell, 1997; Burns, Walsh, Patterson, et al., 1997; Pelham, Evans, Gnagy, & Greenslade, 1992; Pelham, Gnagy, Greenslade, & Milich, 1992; Pillow, Pelham, Hoza, Molina, & Stultz, 1998). For studies that did not examine conduct disorder, two or three factors were found: ADHD and ODD or ADHD inattention, ADHD hyperactivity-impulsivity, and ODD (Burns et al., 2001; Pelham, Gnagy, et al., 1992).

Most of these studies utilized confirmatory factor analyses to validate a hypothesized three- or four-factor solution in line with the *DSM-IV-TR*. However, such models are fairly limited in that they merely address the number of relative factors and their association to one another, rather than allowing for the presence of a more sophisticated hierarchical or co-occurring structure. Such a hierarchical structure is increasingly seen as the most biologically plausible, however (e.g., Krueger et al., 2002). To address this, a few studies compared alternative models of the structure of DBs, including more sophisticated factor models such as second-order factor or bifactor models (Burns et al., 2001; Dumenci, McConaughy, & Achenbach, 2004; Lahey et al., 2008; Pillow et al., 1998). However, none provided a competitive test of simple and complex factorial models of the structure of DBs in childhood. Such evaluation of all structural models is essential in order to establish the best structural model for childhood DB.

Either a second-order factor model or a bifactor model¹ would provide meaningful alternatives to simple factor models by enabling simultaneous estimation of general and specific factors. However, second-order and bifactor models are still distinct from each other in their implications for underlying structure, etiology, and clinical practice. A second-order

factor model allows ADHD and ODD symptom domains to be modeled separately, with both symptom domains being entirely encompassed by a higher order DB factor. This idea would support the idea of an overarching DB diagnostic domain, perhaps with overlapping etiological inputs.

A bifactor model of DB, with a “g” factor and two specific factors of ADHD and ODD, is conceptually distinct. It allows for individual DB symptoms to simultaneously load onto an overall, or general (“g”), DB factor along with completely or partially distinct (specific [s]) factors of ADHD and ODD. Support for this model would suggest substantial interindividual heterogeneity among children with DB, potentially helping to explain why some children exhibit a comorbid clinical picture of ADHD + ODD, whereas others exhibit salient features of only one disorder or the other (reminiscent of insights suggested by Hinshaw, 1987). This model would suggest the importance of both an overall DB category and individual ADHD and ODD diagnostic categories. That is, ADHD and ODD would appear to share some facets that could be captured by an overall category such as DB, but they would also appear to have specific components that are unique to their individual diagnostic category. In line with this idea, a bifactor model suggests that there are distinct, as well as overlapping, etiological influences that converge on the same syndrome, similar to supported bifactor models of the adult externalizing spectrum (Krueger, Markon, Patrick, Benning, & Kramer, 2007) and helping to explain contradictory findings of general and specific influences on DB symptom domains.

There has been some prior support for both second-order and bifactor models of childhood DB. For example, Lahey and colleagues (2008) recently evaluated the structure of childhood DB disorder utilizing a series of confirmatory factor analyses. A simple four-factor model based on the *DSM-IV-TR* provided the best fit to the data, with four distinct, yet correlated, factors: ADHD inattention, ADHD hyperactivity-impulsivity, ODD, and conduct disorder. A second-order factor model (in which the four factors loaded onto an overall externalizing factor) also fit fairly well, although not as well as the simple four-factor model. A bifactor model was not tested, however. Krueger and colleagues (2007) examined both second-order and bifactor models of the adult externalizing spectrum, including antisocial behavior, substance use, and impulsive and aggressive personality traits, finding support for a bifactor model in adults. Finally, there has also been some empirical support for bifactor models of ADHD, based on statistically based assessment approaches and *DSM-IV-TR* symptom counts (e.g., Dumenci, McConaughy, & Achenbach, 2004; Martel, von Eye, & Nigg, 2010; Toplak et al., 2009). However, these studies did not assess DBs such as ODD. Thus, no study to date has provided a comprehensive and competitive test of both simple and complex factorial models of common childhood DB.

Comparing results from these more sophisticated structural models can further understanding of the structure of DB, and this kind of testing is critical for the development of new *DSM* conceptualizations of the structure of common childhood DB disorders. To this

¹Related to multitrait-multimethod models (Campbell & Fiske, 1959), the bifactor model, also called hierarchical model, was introduced to methodologists decades ago (Holzinger & Swineford, 1937). However, it was not introduced to the psychopathology field until more recently (Gibbons & Hedeker, 1992).

end, the current study has a sample of children with ADHD who exhibit substantial comorbidity with ODD in order to test one-factor, two-factor, and three-factor models of common DB (i.e., simple factor models), in addition to second-order factor and bifactor models (i.e., more complex factor models). Based on previous work on second-order factor models of DB and the extensive heterogeneity of clinical presentation of individuals with DB, it is hypothesized that the bifactor model may provide the best approximation to the structure of common DB, or ADHD and ODD.

Method

Participants

Overview—Participants were 548 children (321 boys, 227 girls) between the ages of 6 years and 18 years recruited for a study of ADHD. Children were initially included in one of two groups: those diagnosed with ADHD ($n = 302$) and non-ADHD comparison youth (controls, $n = 220$). Of the 220 non-ADHD comparison children, 21 children were classified as situational (i.e., many symptoms endorsed by one rater and fewer symptoms endorsed by a second rater) or subthreshold (i.e., fewer than required number of symptoms) ADHD or ODD and were included to provide more complete coverage of the dimensional trait space of ADHD and ODD (Levy et al., 1997; Sherman, Iacono, & McGue, 1997). Using a *DSM-IV-TR* perspective, the ADHD group included 110 ADHD-predominantly inattentive type (ADHD-PI; i.e., met criteria for six or more inattentive symptoms, plus impairment, onset, and duration, and never in the past met criteria for combined type) and 192 ADHD-combined type (ADHD-C; i.e., met criteria for six or more inattentive symptoms and six or more hyperactive-impulsive symptoms, plus impairment, onset, and duration). The current sample included no children with the hyperactive-impulsive ADHD subtype, similar to other clinical samples of children with ADHD (e.g., Shaw et al., 2007). As shown in Table 1, 140 children met *DSM-IV-TR* criteria for oppositional-defiant disorder; 114 of those children had comorbid ADHD. Nineteen children met criteria for conduct disorder, 41 for major depressive disorder (lifetime), 13 for dysthymia (lifetime), and 45 for generalized anxiety disorder (lifetime). Children came from 468 families; 388 families had one child in the study, and 80 families had two children in the study.

Recruitment and identification—A broad community-based recruitment strategy was used, with mass mailings to parents in local school districts, public advertisements, and fliers at local clinics, in an effort to mimic the recruitment strategy of the MTA study (Arnold et al., 1997). Families initially recruited then passed through a standard multigate screening process to establish diagnostic groupings. At Stage 1, all families were screened by phone to rule out youth prescribed long-acting psychotropic medication (e.g., antidepressants), neurological impairments, seizure history, head injury with loss of consciousness, other major medical conditions, or a prior diagnosis of mental retardation or autistic disorder, as reported by the parent. All families screened into the study at this point completed written and verbal informed consent procedures, and all study procedures conformed to human subjects guidelines of the National Institute of Mental Health and of the Michigan State University research review board, as well as with APA ethical standards.

At Stage 2, parents and teachers of remaining eligible youth completed the ADHD Rating Scale (ADHD-RS; DuPaul, Power, Anastopolous, & Reid, 1998). In addition, parents completed a structured clinical interview to ascertain duration, impairment, and symptom presence. Parents and teachers were instructed to rate children based on child behavior off medication. Children completed IQ and achievement testing. Families were screened out here only if they did not meet study eligibility requirements (i.e., if they failed to attend the diagnostic visit or if teacher ratings could not be obtained).

The choice of diagnostic interview depended on the year of data collection. For participants who participated between 1997 and 2001 ($N = 218$), the Diagnostic Interview Schedule for Children (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) was completed with the parent by telephone or during on-campus visits. A trained interviewer (i.e., a graduate student or advanced undergraduate with at least 10 hr of training) administered the DISC-IV. Fidelity to interview procedure was checked by having the interview recorded with 5% reviewed by a certified trainer. For children who were administered the DISC-IV and met duration, onset, and impairment criteria for *DSM-IV-TR*, an “or” algorithm was used to establish the diagnostic group and to create the symptom count. Teacher-reported symptoms on the ADHD-RS (i.e., items rated as a 2 or 3 on the 0 to 3 scale) could be added to the parent-endorsed symptom total, up to a maximum of three additional symptoms, to obtain the total number of symptoms (Lahey et al., 1994). Children failing to meet cutoffs for all parent and teacher ADHD rating scales at the 80th percentile and having four or fewer symptoms of ADHD with the “or” algorithm were considered controls.

For participants who participated from 2002 to 2008, youth and their primary caregiver completed the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS-E; Puig-Antich & Ryan, 1986). The data from the interviews and parent and teacher rating scales were then presented to a clinical diagnostic team consisting of a board certified child psychiatrist and licensed clinical child psychologist. They were allowed to use the same “or” algorithm in their diagnostic decision making. Their agreement rates were acceptable for ADHD diagnosis, subtypes, and current ODD and conduct disorder (all $\kappa s \geq .89$).

Pooling the data across families that received the KSADS and the DISC was justified based on our analysis of agreement between the two methods in 430 youth for whom a parent completed both a KSADS and a DISC-IV. The two interviews agreed adequately for total number of symptoms (inattention, intraclass correlation = .88; hyperactivity, intraclass correlation = .86), presence of six or more symptoms of ADHD ($\kappa = .79$), presence of impairment ($\kappa = .64$), and presence of ADHD (defined as six or more symptoms + cross situational impairment in each interview for purposes of computing agreement; $\kappa = .79$). As an additional check on potential interview effects on model results, type of interview was used as a manifest-level predictor of model latent variables with no significant changes in model results.

Measures: Symptom Counts

Maternal report on ADHD symptoms was available via report on diagnostic interview and on the ADHD Rating Scale (ADHD-RS). Teacher report on ADHD was available via report on the ADHD-RS, and teacher report on ODD symptoms was available via report on the

Swanson, Nolan, and Pelham Rating Scale-Fourth Edition (SNAP-IV; Swanson, 1992). Main study analyses utilized parent + teacher report symptom counts for ADHD, using parent report on diagnostic interview and teacher report on the ADHD-RS and teacher-reported symptom counts for ODD. Secondary analyses focused on informant differences in symptoms utilized parent and teacher report on the ADHD Rating Scale.

Data Analysis

A series of confirmatory factor analyses were estimated with the Mplus software package (Muthén & Muthén, 2007). Missingness was minimal in the current study, affecting less than 3% of the sample, and was addressed using pairwise present analysis. The presence of siblings and the resulting nonindependence of data points were addressed with the clustering feature of Mplus. This clustering feature takes into account the nonindependence of the data when computing test statistics and significance tests. Weighted least squares means and variance adjusted (WLSMV) estimation was used.

One-factor, two-factor, three-factor, second-order factor, and bifactor models were estimated sequentially. Goodness of fit was evaluated with chi-square (χ^2) fit statistics, root-mean-square error of approximation (RMSEA), and comparative fit index (CFI). Smaller chi-square and RMSEA values and larger CFI values indicate better fit. Generally speaking, nonsignificant chi-square, RMSEA equal to or below .05, and CFI above .9 indicate reasonable fit (Kline, 2005).

Results

Simple Models

One-factor DB model—First, a one-factor DB model was estimated in which all DB symptoms were hypothesized to load onto a single factor, termed DB. This model assumed that the variance in DB symptoms were best captured by a single underlying factor, assumed to be the diagnostic entity DB. Shown in Figure 1, this model exhibited relatively poor fit, as indicated by a large, significant, chi-square value and an RMSEA value over .1, $\chi^2(45, N = 548) = 726.31, p < .01$; CFI = .95; RMSEA = .17. All paths were significant.

Two-factor ADHD and ODD model—Second, a two-factor ADHD and ODD model was estimated in which all ADHD symptoms were hypothesized to load onto a single factor, ADHD, and all ODD symptoms were hypothesized to load onto a second factor, ODD. Shown in Figure 2, this model exhibited relatively poor fit, as indicated by a large, significant, chi-square value and an RMSEA value of .09, $\chi^2(51, N = 548) = 283.83, p < .01$; CFI = .98; RMSEA = .09. All paths were significant.

Three-factor ADHD inattention, ADHD hyperactivity–impulsivity, and ODD model—Third, a three-factor model was estimated in which inattention, hyperactivity–impulsivity, and oppositional-defiance symptoms were hypothesized to load onto three separate factors. In this model, it was assumed that the three correlated DB symptom domains best captured the variance of individual DB symptoms and that these three symptom domains were not better captured by an overarching diagnostic concept like DB.

This model also exhibited relatively poor fit as indicated by a large, significant chi-square statistic and an RMSEA value over .05, $\chi^2(53, N = 548) = 320.07, p < .01$; CFI = .98; RMSEA = .09. As shown in Figure 3, all paths were significant. In addition, the correlation between ADHD inattention and ADHD hyperactivity was very high ($r = .99, p < .01$), suggesting that ADHD inattention and ADHD hyperactivity–impulsivity were not distinct enough to load onto separate factors and might be most appropriately combined into a single ADHD factor.

Complex Models

Second-order DB factor model—Next, a second-order factor model was estimated in which ADHD and ODD symptoms were hypothesized to load onto two separate factors. These two factors were in turn hypothesized to load onto a higher order factor, termed DB. Thus, this model assumed that ADHD and ODD symptoms loaded onto two separate factors that fell within one overarching category, DB. This overarching DB category is hypothesized to capture the shared variance between the two symptom domains. This model also exhibited relatively poor fit, as indicated by a high, significant chi-square statistic and an RMSEA value of .1, $\chi^2(53, N = 548) = 301.81, p < .01$; CFI = .98; RMSEA = .09). As shown in Figure 4, all paths were significant.

Bifactor DB model—In this model, all DB symptoms were hypothesized to load onto a single factor, termed DB. At the same time, ADHD and ODD symptoms were hypothesized to load onto two factors, termed ADHD and ODD. Whereas the bifactor model assumed that DB symptoms share some common variance (captured by the single factor DB, as in the second-order factor model), it differed from the second-order factor model in that the two individual symptom factors were assumed to capture variance that is unique from the overarching diagnostic category. Compared with other models, this model exhibited the best fit to the data. Although the chi-square statistic remained significant, it was much lower, and the RMSEA value was .05, $\chi^2(77, N = 548) = 176.04, p < .01$; CFI = .99; RMSEA = .05, indicating close fit to the data. As shown in Figure 5, all paths were significant.

Comparison of Models

Although a formal chi-square difference test cannot be conducted on nonnested models such as these, a comparison of model fit statistics can be seen in Table 2. As discussed above, the bifactor model exhibited the best fit to the data, suggesting that DB can be best conceptualized as an overarching diagnostic category with two somewhat separable, but correlated, symptom domains: ADHD and ODD.

Validity Checks

Symptom rater: Parent versus teacher—As a check, one-factor, two-factor, three-factor, second-order factor, and bifactor models were conducted separately for mother and teacher symptom ratings. These results showed the same pattern of results as those depicted for combined mother and teacher ratings earlier. Bifactor models of DB symptoms also exhibited the best fit when relying on mother or teacher ratings separately, as shown in Table 3.

Two-group models: ADHD diagnostic status, age, and sex—Because the loadings of individual symptoms might be expected to vary based on age, sex, or diagnostic status (and all were correlated with one of the factors when included as covariates), possible group differences in symptoms and model fit were examined. Three different fully constrained two-group models (diagnosis, age, sex) were estimated. In these models, all parameters except residual variances were constrained to be equal across the groups. A fully constrained two-group (ADHD diagnostic status) bifactor model was estimated, and this model exhibited satisfactory fit to the data, $\chi^2(33, N = 548) = 57.02, p < .01$; CFI = .96; RMSEA = .05. Although age was a significant covariate for all factors ($p < .01$) when examined as a continuous variable, a fully constrained two-group (age) model exhibited satisfactory fit to the data, $\chi^2(41, N = 548) = 68.95, p < .01$; CFI = .995; RMSEA = .05.

However, the fully constrained two-group (sex) model only exhibited moderate fit to the data, $\chi^2(73, N = 548) = 181.28, p < .01$; CFI = .99; RMSEA = .08. There were several significant modification indices for girls. There was a significant negative correlation between the ODD symptom (“spiteful or vindictive”; *DSM-IV-TR*) and the ADHD symptoms (“difficulty sustaining attention”; “does not seem to listen”; “loses things”; “forgetful”; *DSM-IV-TR*; $p < .05$). There was also a significant positive correlation between the ODD symptom (“spiteful or vindictive”; *DSM-IV-TR*) and the ADHD symptom (“interrupts or intrudes”; *DSM-IV-TR*; $p < .01$). For girls, there was also a significant inverse correlation between the ODD symptom (“argues with adults”; *DSM-IV-TR*) and the ODD symptom (“blames others”; *DSM-IV-TR*; $p < .01$). Once these sex-specific relations were added to the model, the model exhibited reasonable fit to the data, $\chi^2(72, N = 548) = 163.31, p < .01$; CFI = .99; RMSEA = .07.

In all, although there was some variation by age, sex, and diagnostic status in the factor weightings as indicated by the significant covariates, the two-group model analyses suggested that these group differences are relatively minor.

Discussion

In order to elucidate the structure of common childhood DB, a series of confirmatory factor analyses was fit to parent- and teacher-rated ADHD and ODD symptoms. One-factor, two-factor, three-factor, second-order factor, and bifactor models of DB were compared. A bifactor model provided the best fit to the data, suggesting that DB is comprised of an integrated DB category, while simultaneously including ADHD and ODD symptom domains that capture unique individual variance. This supported DB bifactor model further exhibited some important sex differences, although these did not overturn the main conclusion. The elucidation of the structure of childhood DB has important implications for improving understanding of the structure of DB, improving clinical assessment of DB, and aiding in refinement of the behavioral phenotype of DB for research examining etiology.

Previous work on the factor structure of DB (without conduct disorder) has tended to support a two- or three-factor structure of DB, even when second-order factor structures were examined (Burns et al., 2001; Burns, Walsh, Owen, & Snell, 1997; Burns, Walsh, Patterson, et al., 1997; Lahey et al., 2008; Pelham, Evans, et al., 1992; Pelham, Gnagy, et al.,

1992; Pillow et al., 1998). These findings are in line with the findings of the current study, with the exception that the current study added examination of a bifactor model of DB. Support for a bifactor DB model in the current study suggests that a general DB factor incorporating ADHD and ODD coexists alongside specific factor of ADHD and ODD, in line with seminal work suggesting only partial independence of attention deficit/hyperactivity and externalizing behavior problems/aggression (Hinshaw, 1987). Thus, the structure of childhood DB appears to be somewhat more complex than first supposed, in that there appears to be both general and specific components of DB.

This type of bifactor DB model has also been supported more broadly in work done on adults with externalizing disorders. Krueger and colleagues (2002) have advocated a bifactor, or hierarchical, model of the adult externalizing spectrum, and this model has encompassed antisocial behavior, substance use, and maladaptive personality characteristics. They suggest that comorbidity of adult externalizing disorders can be explained by the general externalizing risk factor. However, they indicated that specific externalizing disorders also exhibit somewhat specific risk factors that explain individual differences in liability to single and comorbid disorders (Krueger et al., 2002; Krueger et al., 2007).

Support for a bifactor conceptualization of DB sheds some light on the high levels of DB comorbidity in children with ADHD or ODD and also explains interindividual heterogeneity in patterns of DB comorbidity. Based on the bifactor model, an individual may exhibit a general risk for DB that would manifest in ADHD + ODD and/or an additive risk for ADHD and ODD (Waschbusch, 2002). However, an individual might also exhibit more specific risk for one of the individual DB domains, manifesting in individual diagnoses of ADHD or ODD. Thus, the general DB factor in the bifactor model may help to explain the high levels of comorbidity between ADHD and ODD, whereas the coexistence of general and specific factors of DB may explain interindividual heterogeneity in the patterns of comorbidity of the two disorders.

Sex differences were notable in the current childhood DB bifactor model. For girls, the “spiteful/vindictive” symptom exhibited significant correlations with several ADHD symptoms. These sex differences might, in part, reflect the widely studied sex difference in covert and overt aggression (Crick & Grotpeter, 1995). Since girls typically exhibit higher levels of covert or relational aggression, these forms of aggression may be differentially related to increased risk for specific ADHD symptoms for girls (vs. boys; Crick & Grotpeter, 1995). Examination of these kinds of sex differences deserves further attention in future work.

This bifactor DB conceptualization also has important implications for the way DB is conceptualized in the DSM. Although findings are somewhat in line with the idea that there is a broad overarching category of DB and specific DB diagnostic categories of ADHD and ODD, the bifactor model suggests that it is incorrect to describe ADHD and ODD as merely lower level expressions of DB. Instead, individual symptoms of ADHD and ODD appear to exhibit some communalities captured by a general DB factor, while also exhibiting specific components that are best thought of as separate and unique from a general DB factor. Thus, assessment of childhood DB must comprehensively assess each symptom domain

individually rather than relying on composite measures of DB, since general risk for ADHD + ODD may have different implications than specific risk for ADHD or ODD alone.

The current study makes an important contribution toward refining the DB phenotype at the symptom level. Such refinement may aid in elucidation of complex, multifactorial genetic etiology by making the phenotypic behavioral markers of DB that much more accurate. One possible implication of the current study finding is that individuals high in general DB may be characterized by a somewhat different etiology than individuals exhibiting high ADHD or ODD alone.

The current study's support of a bifactor model of DB is in line with previous behavioral genetic work on genetic and environmental influences on DB symptoms. Specifically, prior work suggests substantial genetic and environmental overlap between the DB symptom domains. ADHD and ODD appear to share genetic and environmental influences and are subject to specific genetic and environmental influences (Burt, Krueger, McGue, & Iacono, 2001; Coolidge, Thede, & Young, 2000; Martin, Levy, Pieka, & Hay, 2006; Nadder, Rutter, Silberg, Maes, & Eaves, 2002; Tuvblad, Zheng, Raine, & Baker, 2009; Waldman, Rhee, Levy, & Hay, 2001). Further, molecular genetic work suggests that ADHD co-segregates with other DB (Jain et al., 2007). The bifactor model provides an integrated structural model of DB that helps to understand these findings.

The supported bifactor model in the current study is limited by the fact that conduct disorder symptoms were not included. Conduct disorder was not prioritized in this study because its determinants are so certain to vary across the age range studied. Moreover, conduct disorder was relatively uncommon in this sample, possibly due to the relatively young age of the children included or due to the failure of severely disturbed families and children to volunteer for the study; however, extending this modeling approach to include conduct disorder will be an important next step for future work. In addition, the current set of models were conducted based on higher level disorder constructs (i.e., ADHD and ODD) rather than on an exhaustive examination of lower order disorder components (e.g., inattention and hyperactivity-impulsivity within ADHD). It should be noted that inclusion of inattention and hyperactivity-impulsivity components did not seem justified based on these components extremely high correlation in the three-factor model. The bifactor model of DB should be further examined and replicated in clinic-referred and general population samples to assess generalizability. The current study was cross-sectional and could not provide direct evidence of developmental changes in symptom expression. Thus, although no significant age differences in the bifactor model were found in the current study, this is an area that could be examined further with a longitudinal research design. The sex differences found in the bifactor model also merit attention in further work, as do potential interactions between sex and age.

One important future direction for this work would be to validate this model with clinical correlates that are considered to be general to DB or specific to ADHD or ODD. For example, children with ADHD and ODD are characterized by higher levels of negative emotionality (Martel & Nigg, 2006). However, poor executive function is specific to ADHD, and parent-child conflict is specific to ODD. Thus, future work might attempt to

validate the general DB and specific ADHD and ODD factors with negative emotionality, executive function, and parent–child conflict measures, respectively. In addition, studies of etiology, including genetic studies, might examine associations with these general and specific DB symptom domains. Finally, the current model could be examined in relation to internalizing symptoms, particularly anxiety/mood problems, which frequently co-occur with both ADHD and ODD. Doing so would aid in assessing the model's suitability for other forms of psychopathology.

In summary, the current study suggests that a bifactor model best describes DB in children with some noted sex differences. This bifactor model suggests that DB has both a shared general component and simultaneously two somewhat distinct symptoms domains of ADHD and ODD. This model has important implications for *DSM* conceptualization, clinical assessment, and etiological study of childhood DB.

Acknowledgments

This research was supported by National Institutes of Health, National Institute of Mental Health Grants R01-MH63146, MH59105, and MH70542 to Joel T. Nigg. We are indebted to the families and staff who made this study possible.

References

- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th., text rev.. Washington, DC: American Psychiatric Association; 2000.
- Angold A, Costello EJ, Erkanli A. Comorbidity. *Journal of Child Psychology and Psychiatry*. 1999; 40:57–87.10.1111/1469-7610.00424 [PubMed: 10102726]
- Arnold LE, Abikoff HB, Cantwell DP, Conners CK, Elliott G, Greenhill LL, et al. Wells KC. National Institute of Mental Health collaborative multimodal treatment study of children with ADHD (the MTA). Design challenges and choices. *Archives of General Psychiatry*. 1997; 54:865–870. [PubMed: 9294378]
- Biederman J, Petty CR, Dolan C, Hughes S, Mick E, Monuteaux MC, Faraone SV. The long-term longitudinal course of oppositional defiant disorder and conduct disorder in ADHD boys: Findings from a controlled 10-year prospective longitudinal follow-up study. *Psychology Medicine*. 2008; 38:1027–1036.10.1017/S0033291707002668
- Burns GL, Boe B, Walsh JA, Sommers-Flanagan R, Teegarden LA. A confirmatory factor analysis on the *DSM-IV* ADHD and ODD symptoms: What is the best model for the organization of these symptoms? *Journal of Abnormal Child Psychology*. 2001; 29:339–349.10.1023/A:1010314030025 [PubMed: 11523839]
- Burns GL, Walsh JA, Owen SM, Snell J. Internal validity of attention deficit hyperactivity disorder, oppositional defiant disorder, and overt conduct disorder symptoms in young children: Implications from teacher ratings for a dimensional approach to symptom validity. *Journal of Clinical Child Psychology*. 1997; 26:266–275.10.1207/s15374424jccp2603_5 [PubMed: 9292384]
- Burns GL, Walsh JA, Patterson DR, Holte CS, Sommers-Flanagan R, Parker CM. Internal validity of the disruptive behavior disorder symptoms: Implications from parent ratings for a dimensional approach to symptom validity. *Journal of Abnormal Child Psychology*. 1997; 25:307–319.10.1023/A:1025764403506 [PubMed: 9304447]
- Burt SA, Krueger RF, McGue M, Iacono WG. Sources of covariation among attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder: The importance of the shared environment. *Journal of Abnormal Psychology*. 2001; 110:516–525.10.1037/0021-843X.110.4.516 [PubMed: 11727941]
- Campbell DT, Fiske DW. Convergent and discriminant validation by the multitrait-multimethod matrix. *Psychological Bulletin*. 1959; 56:81–105.10.1037/h0046016 [PubMed: 13634291]

- Connor DF, Doerfler LA. ADHD with comorbid oppositional defiant disorder or conduct disorder: Discrete or nondistinct disruptive behavior disorders? *Journal of Attention Disorders*. 2008; 12:126–134.10.1177/1087054707308486 [PubMed: 17934178]
- Coolidge FL, Thede LL, Young SE. Heritability and the comorbidity of attention deficit hyperactivity disorder with behavior disorders and executive function deficits: A preliminary investigation. *Developmental Neuropsychology*. 2000; 17:273–287.10.1207/S15326942DN1703_1 [PubMed: 11056845]
- Crick NR, Grotpeter JK. Relational aggression, gender, and social-psychological adjustment. *Child Development*. 1995; 66:710–722.10.2307/1131945 [PubMed: 7789197]
- Dumenci L, McConaughy SH, Achenbach TM. A hierarchical three-factor model of inattention-hyperactivity-impulsivity derived from the attention problems syndrome of the teacher's report form. *School Psychology Review*. 2004; 33:287–301.
- DuPaul, GJ.; Power, TJ.; Anastopolous, AD.; Reid, R.; DuPaul, GJ.; Power, TJ.; et al. Reid, R. ADHD rating scale—IV: Checklists, norms, & clinical interpretation. New York, NY: Guilford Press; 1998.
- Gadow KD, Nolan EE. Differences between preschool children with ODD, ADHD, and ODD+ADHD symptoms. *Journal of Child Psychology and Psychiatry*. 2002; 43:191–201.10.1111/1469-7610.00012 [PubMed: 11902598]
- Gibbons RD, Hedeker DR. Full-information item bi-factor analysis. *Psychometrika*. 1992; 57:423–436.10.1007/BF02295430
- Hinshaw SP. On the distinction between attentional deficits/hyperactivity and conduct problems/aggression in child psychopathology. *Psychological Bulletin*. 1987; 101:443–463.10.1037/0033-2909.101.3.443 [PubMed: 3602250]
- Holzinger KJ, Swineford F. The bi-factor method. *Psychometrika*. 1937; 2:41–54.10.1007/BF02287965
- Jain M, Palacio G, Castellanos FX, Palacio JD, Pineda D, Restrepo MI, et al. Muenke M. Attention-deficit/hyperactivity disorder and comorbid disruptive behavior disorders: Evidence of pleiotropy and new susceptibility loci. *Biological Psychiatry*. 2007; 61:1329–1339.10.1016/j.biopsych.2006.06.026 [PubMed: 16950213]
- Jensen PS, Martin D, Cantwell DP. Comorbidity in ADHD: Implications for research, practice, and *DSM–V*. *Journal of the American Academy of Child and Adolescent Psychiatry*. 1997; 36:1065–1079.10.1097/00004583-199708000-00014 [PubMed: 9256586]
- Kline, RB. Principles and practice of structural equation modeling. 2nd. New York, NY: Guilford Press; 2005.
- Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*. 2002; 111:411–424.10.1037/0021-843X.111.3.411 [PubMed: 12150417]
- Krueger RF, Markon KE, Patrick CJ, Benning SD, Kramer MD. Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. *Journal of Abnormal Psychology*. 2007; 116:645–666.10.1037/0021-843X.116.4.645 [PubMed: 18020714]
- Lahey BB, Applegate B, McBurnett K, Biederman J, Greenhill L, Hynd GW, et al. Richters J. *DSM–IV* field trials for attention deficit hyperactivity disorder in children and adolescents. *American Journal of Psychiatry*. 1994; 151:1673–1685. [PubMed: 7943460]
- Lahey BB, Rathouz PJ, Van Hulle C, Urbano RC, Krueger RF, Applegate B, et al. Waldman ID. Testing structural models of the *DSM–IV* symptoms of common forms of child and adolescent psychopathology. *Journal of Abnormal Child Psychology*. 2008; 36:187–206.10.1007/s10802-007-9169-5 [PubMed: 17912624]
- Levy F, Hay DA, McStephen M, Wood CH, Waldman I. Attention-deficit/hyperactivity disorder: A category or a continuum? Genetic analysis of a large-scale twin study. *American Academy of Child & Adolescent Psychiatry*. 1997; 36:737–744.10.1097/00004583-199706000-00009

- Martel MM, Nigg JT. Child ADHD and personality/temperament traits of reactive and effortful control, resiliency, and emotionality. *Journal of Child Psychology and Psychiatry*. 2006; 47:1175–1183.10.1111/j.1469-7610.2006.01629.x [PubMed: 17076757]
- Martel MM, von Eye A, Nigg JT. Revisiting the latent structure of ADHD: Is there a “g” factor? *Journal of Child Psychology & Psychiatry*. 2010; 51:905–914. [PubMed: 20331490]
- Martin NC, Levy F, Pieka J, Hay DA. A genetic study of attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder and reading disability: Aetiological overlaps and implications. *International Journal of Disability, Development, and Education*. 2006; 53:21–34.10.1080/10349120500509992
- Muthe'n, LK.; Muthe'n, BO. Mplus user's guide. 4th. Los Angeles, CA: Author; 2007.
- Nadder TS, Rutter M, Silberg JL, Maes HH, Eaves LJ. Genetic effects on the variation and covariation of attention deficit-hyperactivity disorder (ADHD) and oppositional-defiant disorder/conduct disorder (ODD/CD) symptomatologies across informant and occasion of measurement. *Psychological Medicine*. 2002; 32:39–53.10.1017/S0033291701004792 [PubMed: 11883729]
- Nock MK, Kazdin AE, Hiripi E, Kessler RC. Lifetime prevalence, correlates, and persistence of oppositional defiant disorder: Results from the National Comorbidity Survey Replication. *Journal of Child Psychology and Psychiatry*. 2007; 48:703–713.10.1111/j.1469-7610.2007.01733.x [PubMed: 17593151]
- Pelham WE, Evans SW, Gnagy EM, Greenslade KE. Teacher ratings of *DSM-III-R* symptoms for the disruptive behavior disorders: Prevalence, factor analyses, and conditional probabilities in a special education sample. *School Psychology Review*. 1992; 21:285–299.
- Pelham WE, Gnagy EM, Greenslade KE, Milich R. Teacher ratings of *DSM-III-R* symptoms for the disruptive behavior disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1992; 31:210–218.10.1097/00004583-199203000-00005 [PubMed: 1564021]
- Pillow DR, Pelham WE, Hoza B, Molina BSG, Stultz CH. Confirmatory factor analyses examining attention deficit hyperactivity disorder symptoms and other childhood disruptive behaviors. *Journal of Abnormal Child Psychology*. 1998; 26:293–309.10.1023/A:1022658618368 [PubMed: 9700521]
- Puig-Antich, J.; Ryan, N. Kiddie schedule for affective disorders and schizophrenia. Pittsburgh, PA: Western Psychiatric Institute; 1986.
- Shaffer D, Fisher P, Lucas C, Dulcan MK, Schwab-Stone M. NIMH Diagnostic Interview Schedule for Children, Version IV (NIMH DISC-IV): Description, differences from previous versions and reliability of some common diagnoses. *Journal of American Academy of Child and Adolescent Psychiatry*. 2000; 39:28–38.10.1097/00004583-200001000-00014
- Shaw P, Gornick M, Lerch J, Addington A, Seal J, Greenstein D, et al. Polymorphisms of the dopamine D4 receptor, clinical outcome, and cortical structure in Attention-Deficit/Hyperactivity Disorder. *Archives of General Psychiatry*. 2007; 64:921–931.10.1001/archpsyc.64.8.921 [PubMed: 17679637]
- Sherman DK, Iancono WG, McGue MK. Attention-deficit hyperactivity disorder dimensions: A twin study of inattention and impulsivity-hyperactivity. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1997; 36:745–753.10.1097/00004583-199706000-00010 [PubMed: 9183128]
- Swanson, JM. School-based assessments and interventions for ADD students. Irvine, CA: KC; 1992.
- Toplak ME, Pitch A, Flora DB, Iwenofu L, Ghelani K, Jain U, Tannock R. The unity and diversity of inattention and hyperactivity-impulsivity in ADHD: Evidence for a general factor with separable dimensions. *Journal of Abnormal Child Psychology*. 2009; 37:1137–1150.10.1007/s10802-009-9336-y [PubMed: 19562477]
- Tuvblad C, Zheng M, Raine A, Baker LA. A common genetic factor explains the covariation among ADHD, ODD, and CD symptoms in 9–10 year old boys and girls. *Journal of Abnormal Child Psychology*. 2009; 37:153–167.10.1007/s10802-008-9278-9 [PubMed: 19015975]
- Waldman, ID.; Rhee, SH.; Levy, F.; Hay, DA. Causes of the overlap among symptoms of attention deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder. In: Levy, F.; Hay, DA., editors. *Attention, genes and ADHD*. East Sussex, England: Brunner-Routledge; 2001. p. 115-138.

Waschbusch DA. A meta-analytic examination of comorbid hyperactive-impulsive-attention problems and conduct problems. *Psychological Bulletin*. 2002; 128:118–150.10.1037/0033-2909.128.1.118 [PubMed: 11843545]

Figure 1.
One-factor disruptive behavior (DB) model.

Figure 2.
Two-factor attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) model.

Figure 3. Three-factor attention-deficit/hyperactivity disorder (ADHD) inattention, ADHD hyperactivity-impulsivity, and oppositional defiant disorder (ODD) model. Inatt = inattention; Hyper = hyperactivity.

Figure 4. Second-order disruptive behavior (DB) factor model. ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder.

Figure 5.

Bifactor disruptive behavior (DB) model. ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder.

Table 1
Descriptive Statistics on Sample

Statistic	ADHD	ODD	ADHD + ODD	Control
Boys <i>n</i> (%)	101 (66)	11 (42.3)	79 (69.3)	119 (49.80)*
Ethnic minority <i>n</i> (%)	41 (26.80)	6 (23.10)	28 (24.56)	64 (26.78)
Age (years)	11.28 (2.99)	10.98 (2.74)	11.19 (2.74)	12.18 (3.20)
IQ	103.10 (13.64)	108.38 (17.50)	103.29 (13.33)	109.55 (15.1)*
Family income (\$)	73,074.38 (87,143.03)	68,000.00 (33,995.10)	50,919.57 (31,207.38)	70,295.46 (48,298.57)
Inattentive Sx (P + T)	5.95 (2.35)	0.46 (0.95)	6.28 (2.37)	1.53 (2.45)*
Hyperactive Sx (P + T)	5.93 (2.37)	0.58 (1.36)	6.88 (2.38)	1.82 (2.74)*
Inattentive Sx (P)	17.28 (5.04)	3.08 (3.99)	18.91 (5.01)	6.21 (6.58)*
Hyperactive Sx (P)	10.20 (7.21)	2.63 (2.63)	14.29 (6.96)	4.02 (5.27)*
Inattentive Sx (T)	15.05 (7.06)	2.12 (2.60)	15.17 (6.66)	4.59 (5.71)*
Hyperactive Sx (T)	9.30 (8.01)	1.08 (1.38)	10.93 (7.54)	2.98 (5.06)*
ODD Sx (T)	2.28 (3.08)	0.63 (1.21)	4.39 (4.73)	0.89 (2.15)*

Note. Means are provided, with standard deviations in parentheses, for age, IQ, family income, and symptoms. ADHD *n* = 188; ODD *n* = 26; ADHD + ODD *n* = 114; Control *n* = 220. Sx = symptoms; (P + T) = Parent + Teacher rated symptoms; (P) = Parent-rated symptoms; (T) = Teacher-rated symptoms; ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder.

* $p < .01$, via analysis of variance or chi-square.

Table 2
Confirmatory Factor Analysis Fit Statistics for Parent + Teacher Symptom Ratings

Model	χ^2	<i>df</i>	CFI	RMSEA
One-factor model	726.31 *	45	.95	.17
Two-factor model	283.83 *	51	.98	.09
Three-factor model	320.07 *	53	.98	.09
Second-order factor model	301.81 *	53	.98	.09
Bifactor model	176.04 *	77	.99	.05

Note. For chi-squares, $N = 548$. RMSEA = Root-mean-square error of approximation; CFI = comparative fit index.

*
 $p < .01$.

Table 3
Confirmatory Factor Analysis Fit Statistics for Mother and Teacher Symptom Ratings

Model	χ^2	df	CFI	RMSEA
Mother ratings				
One-factor model	965.77*	36	.92	.22
Two-factor model	584.19*	56	.94	.13
Three-factor model	202.54*	65	.98	.07
Second-order factor model	462.60*	55	.94	.14
Bifactor model	167.09*	67	.99	.06
Teacher ratings				
One-factor model	1050.97*	35	.88	.23
Two-factor model	654.08*	43	.95	.16
Three-factor model	230.47*	59	.98	.09
Second-order factor model	410.35*	39	.96	.16
Bifactor model	136.85*	56	.99	.06

Note. For chi-squares, $N = 548$. RMSEA = Root-mean-square error of approximation; CFI = comparative fit index.

* $p < .01$.