Published in final edited form as:
J Abnorm Psychol. 2005 August ; 114(3): 483–487.

Symptom-Based Subfactors of *DSM*-Defined Conduct Disorder: Evidence for Etiologic Distinctions

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Abstract

Past research investigating the structure of conduct disorder (CD) symptoms has provided evidence of a phenotypic distinction between aggressive and rule-breaking subfactors of CD. However, evidence of etiologic distinctions between these subfactors has not been reported to date. In the current study, the authors investigated this issue in a sample of 1,151 male twins who were 17 years of age. The results indicate that aggressive and rule-breaking CD subfactors have both common and distinct etiologic influences, with shared environmental influences playing a significant role in rule-breaking behaviors. The authors discuss implications of these findings for the assessment and treatment of CD.

Keywords

conduct disorder; subfactors; antisocial behavior; aggression

Childhood and adolescent antisocial behavior (ASB) represent a serious societal problem, with almost one fifth of overall crimes being committed solely by individuals under the age of 18 years (Federal Bureau of Investigation, 2003). Nevertheless, the etiology of these behaviors appears complex, as studies differ in their conclusions about genetic and environmental contributions to childhood and adolescent ASB. One potential reason for this lack of agreement among studies may be inadequate attention among researchers to potential heterogeneity within the domain of disruptive behaviors (Rutter, 2003). Currently, major classification systems for childhood and adolescent psychopathology take different approaches to parsing the domain of ASB. The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) classifies severe ASBs within the unitary diagnostic category of conduct disorder (CD). Another widely used classification system, the Child Behavior Checklist (CBCL; Achenbach, 1991) divides disruptive behaviors into Aggressive and Delinquent Behaviors.

Building on previous factor analytic investigations, Tackett, Krueger, Sawyer, and Graetz (2003) recently found that *DSM–IV* symptoms of CD divide into two correlated subfactors corresponding with the Aggressive and Delinquent Behavior syndromes of the CBCL. The goal of the current study was to extend Tackett et al.'s (2003) findings in a genetically informative sample. If the division of CD into two correlated subfactors is important in understanding childhood ASB, then the two subfactors should correspond to partially distinct etiologies. Examining these behaviors in a twin sample allows for an investigation into both shared and distinctive etiological contributions to these two proposed CD subfactors. The

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context for our study is provided by the existing behavioral genetic literature on the CBCL Aggressive and Delinquent Behavior syndromes and *DSM*-defined CD.

Behavioral Genetic Studies of the CBCL

Unlike the conceptualization of CD in the *DSM* as a unitary entity, Achenbach's CBCL (Achenbach, 1991) separates *aggressive* behaviors from *delinquent* (or *rule-breaking*, in the most recent version of the CBCL; Achenbach & Rescorla, 2001) behaviors. The scales in the CBCL were derived from factor analysis and appear to be correlated subcomponents of a broader externalizing behavior syndrome.

Several twin and adoption studies have investigated genetic and environmental influences on aggression and delinquency as measured by the CBCL. In general, these studies have found a substantial genetic component (around 65%) but no significant shared environmental component (i.e., influences that are common to two siblings growing up in the same household and make them similar, such as neighborhood effects) for Aggressive Behavior, whereas Delinquent Behavior has shown a moderate genetic component (around 35%) and a moderate shared environmental component (also around 35%; e.g., Edelbrock, Rende, Plomin, & Thompson, 1995; Eley, Lichtenstein, & Moffitt, 2003; Van Den Oord, Boomsma, & Verhulst, 1994). The remaining variance in both syndromes is attributable to nonshared environmental influences (i.e., influences that affect two siblings independently and make them different). These findings have been generally replicable, although some studies have found occasional variation across reporter, age group, or gender. Taken together, these studies provide evidence for distinct etiologies of Aggressive Behavior and Delinquent Behavior as defined by the CBCL.

Behavioral Genetic Studies of CD

Estimated magnitudes of genetic and environmental effects on *DSM* CD vary from study to study (Rhee & Waldman, 2002; Simonoff, 2001). Results have been reported from studies that have used different informants and various editions of the *DSM* (e.g., Eaves et al., 1997; Kendler, Prescott, Myers, & Neale, 2003; Lyons et al., 1995). These studies have typically found a moderate genetic component, averaging 36% (although estimates from these studies were wide ranging, from 6% to 71%); a smaller, sometimes nonsignificant shared environmental component, averaging 12% (estimates ranged from 0% to 37%); and a substantial nonshared environmental component, averaging 53% (estimates ranged from 31% to 86%).

The Current Study

As noted in recent reviews (Rhee & Waldman, 2002; Simonoff, 2001), the overall estimates from behavioral genetic studies of *DSM*-defined CD have been significantly heterogeneous. One factor that may be contributing to this heterogeneity in results is that *DSM*-defined CD may not be an etiologically unitary entity. As reviewed above, behavioral genetic results for the CBCL Aggressive and Delinquent Behavior syndromes have been largely consistent and have supported distinct etiologies for these two syndromes, whereas results for *DSM*-defined CD have been more heterogeneous. Thus, one potential explanation for the heterogeneity in behavioral genetic findings for *DSM* CD may be the phenotypic heterogeneity among the *DSM* CD symptoms.

Research on potential subfactors of CD is not a new topic of interest. Previous factor analytic investigations of *DSM* symptoms of CD have provided evidence for subfactors of CD representing aggressive and nonaggressive behaviors (Frick et al., 1991, 1993; Loeber & Schmaling, 1985; Quay, 1986), and these potential subfactors of CD have been linked to the

CBCL syndromes of similar content (Tackett et al., 2003). Specifically, the aggressive CD subfactor was uniquely related to the CBCL Aggressive Behavior syndrome, whereas the nonaggressive CD subfactor was uniquely related to the CBCL Delinquent Behavior syndrome. These factor analytic studies have provided evidence for phenotypic heterogeneity within the *DSM* conceptualization of CD and have linked potential CD subfactors with the Aggressive and Delinquent Behavior syndromes of the CBCL. However, despite evidence that subfactors of CD exist at a phenotypic level, this distinction has not been incorporated into the *DSM*. Evidence of this distinction at the etiologic level would add to the validity of these subtypes and further the case for including this distinction in future editions of the *DSM*.

Although some work has suggested that subfactors of CD have distinct genetic and environmental influences (Simonoff, Pickles, Meyer, Silberg, & Maes, 1998), to our knowledge, the possibility of distinct genetic and environmental influences on CD derived from in-person interviews with an empirically derived set of CD subfactors has not been addressed directly. The current study was designed to address this issue. Specifically, we were interested in the structure and etiological influences on subfactors of *DSM*-defined CD. We hypothesized that *DSM*-defined CD symptoms would subdivide into meaningful subfactors of a broader dimension, similar to the conceptualization of childhood ASB in the CBCL. Given this hypothesis, we expected CD subfactors to show both common influences (those related to the broader dimension linking them) and distinct influences (those that are subfactor specific). To test this hypothesis, we first sought to replicate results from previous factor analytic studies of CD symptoms that provided evidence for subfactors within CD (e.g., Frick et al., 1993; Tackett et al., 2003). Then, we investigated genetic and environmental influences that both linked and differentiated the resulting subfactors.

Method

Participants

Participants were male twins recruited for the Minnesota Twin Family Study (MTFS), a longitudinal study of twins and their families. The participants in the current study included male participants from two cohorts, followed longitudinally at 3-year intervals. The sample was epidemiological, with participants representative of the Minnesota population at time of birth (see Holdcraft & Iacono, 2004; Iacono, Carlson, Taylor, Elkins, & McGue, 1999). Similar to the population of Minnesota at that time, approximately 98% of the twins were Caucasian. More complete information on the MTFS can be found in Iacono and McGue (2002).

For the purposes of this study, we combined male participants from two cohorts at the overlapping assessment point of age 17 years. In the first cohort, boys were initially interviewed in person at an average age of 11 years, with follow-up interview assessments at 3 and 6 years later. At the second follow-up assessment, 574 male participants had complete data for Diagnostic and Statistical Manual of Mental Disorders (3rd ed., rev.; *DSM–III–R*; American Psychiatric Association, 1987) conduct disorder, with an average age of 17.94 at time of assessment (age range = 17–20 years). In the second cohort, male participants were initially assessed at an average age of 17.49 years (range = 17–19 years). A total of 577 male participants from this cohort had complete data for *DSM–III–R* CD. Overall, the resulting sample included 1,151 male participants, balanced between cohorts (49.9% from 11-year-old intake cohort, 50.1% from 17-year-old intake cohort), with an average age of 17.72 years.

For the behavioral genetic analyses, the sample was reduced to exclude individuals without cotwin data and who were triplets ($n = 39$). This resulted in a sample of 369 monozygotic (MZ) twin pairs and 187 dizygotic (DZ) twin pairs. Overall, this made up 97% of the sample used for the factor analyses, with a final n of 1,112 for the behavioral genetic analyses. In addition, female participants could not be included in these analyses because they did not exhibit

sufficient variance in the CD symptoms. Specifically, only 3 of the 12 available CD symptoms (stolen without confrontation, often lies, and initiates physical fights) showed 5% endorsement in the female sample at any level of endorsement (i.e., including subthreshold endorsement), whereas 10 of the 12 available CD symptoms exceeded this rate in the male sample. *Subthreshold-level symptoms* were defined as behavior that was deemed as noteworthy in severity or frequency but was not severe or frequent enough to warrant full symptom endorsement.

Zygosity

The MTFS uses three estimates in combination to determine twin zygosity. Specifically, zygosity measures include a standard zygosity questionnaire completed by the parents, an evaluation by MTFS staff of physical similarity, and an algorithm involving fingerprint ridge count and ponderal and cephalic indices. A validation study ($N = 50$) showed 100% accuracy in zygosity determination when all three estimates agreed. When these three estimates disagree, a blood sample is drawn for a serological analysis.

Measures

We assessed *DSM–III–R* CD symptomatology via in-person interviews using the Diagnostic Interview for Children and Adolescents—Revised (Reich & Welner, 1988). Of the 13 *DSM–III–R* symptoms, 12 were assessed; the symptom regarding forced sexual behavior was not asked. In addition, two symptoms were deleted from the analyses because of extremely low prevalence (stolen with confrontation, 99.0% rated as absent; and runaway overnight, 97.7% rated as absent). Thus, 10 of the 13 CD symptoms were included in these analyses. Symptoms could be rated as present, present at a subthreshold level, or absent. Ratings of symptoms were made by a consensus team of advanced clinical psychology graduate students (see Iacono et al., 1999, for more information on the consensus approach to diagnosis). Both cohorts (age 11 years and age 17 years) reported on lifetime behaviors at intake and reported on behavior over the last 3 years at subsequent follow-up assessments. Thus, variables were created that reflected lifetime behavior at age 17 years for both cohorts.

Behavioral Genetic Analyses

To make use of our genetically informative sample, we used twin-study methodology (for more explanation on twin-study methodology, see Plomin, DeFries, McClearn, & McGuffin, 2000). By analyzing the data within MZ (who share all of their genes) and DZ (who share on average 50% of their segregating genes) twin pairs, the phenotypic variance is decomposed into that attributed to genetic influences, shared environmental influences, and nonshared environmental influences (which also includes measurement error). We used the Cholesky decomposition method to estimate genetic and environmental associations among the potential CD subfactors (see Neale & Cardon, 1992, for more information about the Cholesky model).

Results

Factor Analyses

First, we wanted to examine the structure of the 10 *DSM–III–R* CD symptoms. We conducted factor analyses using Mplus (Muthén & Muthén, 1998). As an initial step, we conducted exploratory factor analyses, which replicated the methodology used in Tackett et al.'s (2003) study. We conducted initial exploratory analyses because previous studies have not agreed on some symptoms as being primarily aggressive or nonaggressive. The symptom "cruel to animals" did not load substantially on either factor (i.e., did not show a loading of greater than . 30). Thus, this item was dropped from our analyses. The first subfactor was made up of rule-breaking behaviors, whereas the second subfactor was made up of aggressive behaviors, which

replicated the factor structure found in our previous study (Tackett et al., 2003). We use the labels “aggressive” and “rule-breaking” to refer to these sub-factors throughout the article. (It is also important to note that these are the same labels used in the CBCL for the two ASB syndromes; Achenbach & Rescorla, 2001.)

Following the exploratory analyses, we conducted confirmatory factor analyses to estimate model fit statistics to formally compare fit differences between various models that represented different factor structures. We accounted for the correlated nature of twin data by clustering individuals within families. In the two-factor confirmatory model, we allowed the CD symptoms to load on the same factors they did in the exploratory analysis (see Table 1).[1] We also fit a one-factor confirmatory model. Comparing the one-factor and two-factor models, multiple fit indices indicated that the two-factor model (Akaike information criterion [AIC; Akaike, 1987] = −15.33, root-mean-square error of approximation [RMSEA] = .02, comparative fit index = .99, Tucker–Lewis Index = .98) was a better account of the covariation among the CD symptoms than the one-factor model (AIC = 24.49, RMSEA = .04, comparative fit index = .94, Tucker–Lewis Index = .92). It is important to note that the two factors showed a notable correlation (.73). This correlation indicates that the two factors are not entirely distinct but, rather, represent distinguishable subfactors of a broader CD dimension. The fact that they are distinguishable is indicated both by the improved fit of the two-factor model over the one-factor model, as well as the fact that the .73 correlation in the two-factor model was significantly different from 1.0 (95% confidence interval = .68–.78).

Behavioral Genetic Analyses

Our next step was to examine the genetic and environmental influences on the two *DSM–III–R* CD subfactors. To provide measures of the aggressive and rule-breaking subfactors, we summed the ratings for each of the symptoms that loaded on a given factor. In other words, the aggressive subfactor score for a given individual was equal to the sum of their scores on the three symptoms loading on that subfactor, whereas the rule-breaking subfactor score equaled the sum of the six symptoms loading on that subfactor.

We then estimated polychoric correlations, which assume an underlying normal distribution for the behaviors, among the aggressive and rule-breaking subfactors for MZ and DZ twins using Mplus. These polychoric correlations were input into Mx (Neale, Boker, Xie, & Maes, 2002) to estimate the bivariate Cholesky model. The Cholesky decomposition model provided estimates for the proportion of variance for each subfactor that was attributable to genetic, shared environmental, and nonshared environmental influences, as well as the proportion of these influences that acted on both subfactors. This model produced a good fit (AIC = −7.13, RMSEA = .00), $\chi^2(5) = 2.87$. Parameter estimates from this model are presented in Table 2.

As Table 2 shows, the aggression subfactor was significantly influenced by only genetic and nonshared environmental influences, not by shared environmental influences. Similarly, the covariance between the aggression and rule-breaking subfactors was significantly influenced by only genetic and nonshared environmental influences. Alternatively, the rule-breaking subfactor was significantly influenced by all three influences: additive genetic, shared

[1]We also wanted to check for any significant ($p < .05$) differences between the two cohorts (11 years of age at intake and 17 years of age at intake) to examine whether the different numbers of assessments were influencing the results. Although there were some significant differences in the average endorsement between cohorts, the structure of the symptoms appeared to be the same. Independent samples *t* tests revealed that often lies, $t(1149) = 8.10$; fire-setting, $t(1149) = 2.04$; used a weapon, $t(1149) = 4.20$; initiates physical fights, $t(1149) = 2.81$; and cruelty to people, $t(1149) = 3.16$; were all endorsed at significantly higher rates in the 11-year-old cohort. However, running the factor analytic models separately within each cohort produced the same results presented for the combined sample. Specifically, the exploratory and confirmatory models showed the same overall structure (items loaded on the same factors) and relative fit (preferring a two-factor model) in each cohort separately.

environmental, and nonshared environmental. In addition, the genetic and shared environmental influences on rule-breaking were equal (at least to 2 decimal points).

Discussion

In the current study, we sought to investigate etiologic differences in subfactors of CD. Specifically, we replicated our previous work by using factor analysis to uncover two subfactors of CD, related to aggressive and rule-breaking behaviors. We then used a twin design to investigate both common and unique genetic and environmental influences on these CD subfactors. Our results indicate that although some influences on aggressive and rule-breaking subfactors were in common, each subfactor also had unique influences. Specifically, the influence of shared environmental factors was different for aggressive and rule-breaking behaviors, such that the rule-breaking subfactor showed significant shared environmental influences, whereas the aggression subfactor did not. Although this pattern of results has been well documented in research that has used the CBCL, these results extend a body of literature documenting phenotypic subfactors of *DSM*-defined CD symptoms and provide support for distinguishing between these subfactors on the basis of etiological distinctions in future editions of the *DSM*.

These findings also support the hypothesis that the aggressive and rule-breaking CD subfactors are correlated subfactors of an underlying dimension of ASB, suggesting a hierarchical conceptualization of childhood ASB, such as that used in the CBCL. Our previous work established empirical relationships between the CBCL Aggressive Behavior and Rule-Breaking (or Delinquent) Behavior syndromes with *DSM–IV* CD subfactors of similar content (Tackett et al., 2003). To expand on the continuity between these two classification systems, we present genetic and environmental influences on *DSM*-defined CD subfactors in this article that are largely commensurate with behavioral genetic studies of the CBCL. It is important to establish clear links between the *DSM* and the CBCL to facilitate interpretability across research that uses these different systems.

It is important to note some limitations of our study. First, because only *DSM–III–R* symptoms were available at all time points, we were unable to include the two CD symptoms added to *DSM–IV* (bullies, threatens, or intimidates; often stays out at night without parental permission). Future investigations into the etiology of subfactors of CD should also include these additional *DSM–IV* CD symptoms (assuming they reach adequate prevalence rates). However, replication between systems would be expected, as all of the symptoms used in these analyses were carried over from *DSM–III–R* to *DSM–IV*. In addition, clinical samples with higher prevalence rates could include the *DSM–III–R* symptoms that were excluded from these analyses.

Second, the analyses presented in this study included data from boys only. Although girls were assessed in the MTFS, their prevalence rates for CD symptoms were too low to be included in the analyses. Although investigations of childhood ASB often exclude girls because of low prevalence, extensions of this work may use samples in which greater female endorsement is more likely (e.g., clinical or adjudicated samples). Another way to increase female endorsement may be to incorporate measures that assess a larger variety of ASBs (such as the CBCL), which may provide a more comprehensive assessment of the dimension of ASB.

Although this study has limitations, the findings presented here have key implications for the classification of childhood and adolescent behavior. These results are consistent with previous findings (e.g., Frick et al., 1993; Loeber & Schmaling, 1985; Tackett et al., 2003) in demonstrating that symptoms found in the *DSM* construct of CD may be best conceptualized as two correlated subfactors of an underlying dimension. However, this study extends these

results by finding both distinct and shared etiological influences on these empirically derived subfactors of CD. Thus, the current study supports existing evidence for distinguishing CD subfactors at the phenotypic level, as well as providing new evidence for this distinction at the etiologic level.

However, despite these findings, the *DSM* currently defines subtypes of CD only on the basis of age of onset, not on types of behaviors. The current study underscores the advantages of differentiating these behavioral subtypes by highlighting the different causal processes that appear to be contributing to aggressive and rule-breaking behaviors. These results suggest that studying CD as a unitary construct may be masking important etiological processes that fit in with a larger literature examining subtypes of childhood ASB. For example, research on differential age of onset has suggested that subtyping based on age of onset has important implications for course and prognosis on a variety of dimensions (e.g., Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). In addition, age of onset has been linked with the subtypes proposed here, such that individuals with an early age of onset tend to show more aggressive behaviors (Lahey et al., 1998). Thus, this potential connection between subtypes based on age and subtypes based on behavior might be an important area for future research in terms of obtaining a clearer understanding of course and prognosis for various individuals.

It is important for future research to differentiate between these behavioral subfactors when studying childhood ASB to identify both specific and common correlates to aggressive and rule-breaking behaviors. Future studies investigating specific environmental factors could differentiate between aggressive and rule-breaking behaviors, as environmental factors may increase risk for rule-breaking behaviors more so than aggressive behaviors.

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Table 1

DSM–III–R CD Symptom Prevalence Rates and Factor Loadings in a Two-Factor Confirmatory Solution

Symptom	Factor[a]	
	Aggressive subfactor	Rule-breaking subfactor
Often intiates physical fights (22.3%)	.94	
Physically cruel to people (8.1%)	.61	
Used a weapon in a fight (9.4%)	.46	
Stolen without confrontation (20.6%)		.96
Destroyed property (20.9%)		.88
Deliberate fire setting (26.3%)		.79
Often lies (31.4%)		.70
Broken into a home or car (6.6%)		.53
Often truant from school (7.8%)		.50

Note. Prevalence rates are presented in parentheses next to each symptom. These rates reflect endorsement (at the subthreshold level or higher) of the behavior for the overall sample. CD = conduct disorder.

[a] Factor intercorrelation = .73.

Table 2

Estimates of the Proportion of Variance for Each CD Subfactor and the Covariance Between the Subfactors Attributable to Genetic, Shared Environmental, and Nonshared Environmental Influences

Variable	Additive genetic	Shared Environment	Nonshared environment
Aggression subfactor	**.35**	.14	**.51**
Covariance	**.51**	.28	**.21**
Rule-breaking subfactor	**.28**	**.28**	**.44**

Note. Significant parameter estimates are in bold. CD = conduct disorder.