ORIGINAL ARTICLE

Familial Influences on Conduct Disorder Reflect 2 Genetic Factors and 1 Shared Environmental Factor

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Context: Prior studies suggest that antisocial behavior in childhood and adolescence reflects multiple symptomatic dimensions. However, to our knowledge, no prior study has evaluated the underlying nature of the etiologic influences contributing to conduct disorder (CD) symptoms as defined in the *DSM*.

Objective: To determine the structure of genetic and environmental risk factors for CD.

Design: Population-based twin registry.

Setting: Virginia.

Participants: Two thousand seven hundred sixty-nine members of male-male twin pairs from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders.

Main Outcome Measure: Retrospective self-reported symptoms of CD.

Results: The best-fitting multivariate twin model included 2 genetic factors, 1 shared environmental common factor, and 1 nonshared environmental common factor, along with criterion-specific genetic and nonshared environmental effects. The CD criteria with the strongest loadings on the 2 genetic factors were, respectively, those reflecting rule breaking (eg, playing hooky) and overt aggressive acts (eg, hurting people). The shared environmental common factor had salient loadings on a distinct set of criteria reflecting covert delinquent acts (eg, stealing and hurting animals). Loadings on the single nonshared environmental common factor were more uniform and less selective. Scores on the 3 familial CD factors were differentially associated with a range of personality, psychopathology, and demographic factors.

Conclusions: From a genetic perspective, the *DSM* criteria for CD do not reflect a single dimension of liability. The familial risk to CD is composed of 2 discrete dimensions of genetic risk, reflecting rule breaking and overt aggression, and 1 dimension of shared environmental risk, reflecting covert delinquency. These 3 familial factors differ meaningfully in their association with a range of relevant validators.

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CONDUCT DISORDER (CD) AS defined in *DSM-IV* is a relatively common condition of childhood and adolescence, affecting 6% to 16% of males and 2% to 9% of females, and is associated with adverse long-term outcomes including adult antisocial behavior, educational underachievement, and substance problems.[1] Most twin studies evaluating the role of genetic and environmental factors in the etiology of *DSM*-defined CD[2-5] have established that, in contrast with other psychiatric disorders, in addition to important genetic influences, the shared environment substantially contributes to risk for CD. Other twin studies have examined various self- or parental-rated scales for conduct, antisocial, or externalizing problems in children and adolescence. A careful meta-analysis of twin and adoption studies on antisocial behavior estimated that genetic and shared environmental effects accounted for 46% and 20% of the variability, respectively, for childhood antisocial behaviors and 43% and 16% for such behaviors in adolescence.[6]

Traditional epidemiological studies implicitly assume that CD criteria reflect a single dimension of liability. However, several studies have suggested that CD symptoms, measured by self-rating scales or *DSM* criteria, are not unidimensional. Typologies have been proposed including aggression vs rule breaking[7-9]; covert, overt, and authority conflict[10]; and aggression, delinquency, and rule breaking.[11] Although some prior twin studies have examined the etiology of CD subdimen-

sions,[9,12] these studies have explored genetic and environmental influences on phenotypic factors, not individual CD symptoms.

The goal of the current report is to elucidate, in a population-based sample of male twins, the structure of genetic and environmental risk factors for individual CD criteria. Additional analyses were conducted in an attempt to validate the observed genetic and shared environmental factors. First, estimated factor scores were used to predict a range of relevant personality, psychopathology, and demographic factors. Second, we examined whether these genetic and environmental factors were evident in an exploratory phenotypic factor analysis.

Prior analyses in this sample, which examined CD as a dichotomous disorder[13] and as a symptom count,[2] revealed evidence for both genetic and shared environmental effects. This analysis extends prior work by addressing 2 questions. First, does more than 1 genetic factor underlie the CD criteria, and if so, would these factors map onto prior CD typologies? Second, would the shared environmental liability be distributed across CD symptoms or more strongly concentrated in select criteria? Given the replicable finding of aggressive and rule-breaking factors in prior work,[7,8,11] and evidence pointing to genetic influences on these dimensions,[9,12] our major hypothesis was that distinct genetic factors would be identified that would resemble these phenotypic factors. More tentatively, we postulated that additional factors corresponding to nonaggressive delinquency and/or covert antisocial behavior might emerge.[10,11] Regarding shared environmental influence, we predicted that this would be most important for criteria reflecting rule-breaking or covert CD symptoms. This prediction was based on findings of a meta-analysis of twin and adoption studies by Burt[12] that revealed a significant contribution of shared environmental influence to rule-breaking (nonaggressive or covert) forms of antisocial behavior but not aggressive delinquent acts.

METHODS

Participants were male-male twin pairs from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders.[14] These pairs came from a sample (birth years 1940-1974) ascertained from registry records containing all twin births. Zygosity was determined using standard twin questions validated against DNA genotyping.[15] The first interview was completed largely by telephone in 1993 to 1996 with a 72% response rate. The second wave of interviews, conducted in 1994 to 1998, was largely done face to face, yielding a response rate of 83%. Self-report questionnaires (SRQs) were obtained from 94% of individuals who completed the second interview (ie, 643 complete monozygotic and 430 complete dizygotic pairs plus 623 single twins). The mean (SD) age of male-male pairs at the time of SRQ completion was 37.2 (9.2) years.

Screening items for CD were included in the first interview but our analyses herein focus on the more complete assessment in the second-wave SRQ. In this questionnaire, twins reported how often they had engaged in 11 specific antisocial behaviors in childhood and adolescence. As detailed previously,[16] the *DSM-III-R*[17] CD criterion regarding forced sex was omitted in the SRQ because of its very low expected prevalence and its potential offensiveness. Two other criteria with similar content (has broken into someone else's house, building, or car and has stolen without confrontation) were combined into a single item. For 9 of the 11 SRQ items, 4 responses were possible (0, 1-2, 3-5, and ≥6 times) and assessed for 2 different developmental periods: before age 15 years and ages 15 to 17 years. The current analyses used the higher-frequency category reported for these 2 periods. The remaining 2 items (physical fights and telling lies), which occurred with higher frequency, were assessed only for the period prior to age 15 years, using 4 response options (never, rarely, sometimes, and often). The response rate for 1 SRQ item (robbed or mugged someone) was so low in the current sample that it was omitted from the analysis. Thus, the reported results focus on 10 CD criteria, for which short descriptions are seen in the **Figure**. We evaluated the test-retest reliability of these items (n=298; mean interassessment interval, 4.8 weeks), and for most of them, they were in the acceptable to good range. By weighted κ,[18] the range was +0.42 (starting physical fights) to +0.76 (playing hooky) with a mean of +0.62. By polychoric correlation, the range was +0.65 (starting fights) to +0.93 (running away) with a mean of +0.81.

Following the analysis directed at clarifying the etiologic structure of the CD criteria, we sought to validate the configuration of common etiological factors emerging from this analysis. These analyses focused on 3 domains of validating variables: personality, psychopathology, and demographics. For personality, we examined neuroticism, extraversion,[19] and novelty seeking.[20] For psychopathology, we examined adult antisocial personality disorder symptoms, generalized anxiety disorder (GAD), and cocaine and alcohol dependence diagnosed according to *DSM-III-R*[17] or *DSM-IV*[1] criteria using Structured Clinical Interview for *DSM-III-R* interview protocols.[21] The 2 demographic variables examined were years of education and age at interview. All validating variables were derived from the main interview except for adult antisocial personality symptoms, which were assessed via the SRQ.

A series of multivariate twin models positing different combinations of additive genetic (A), shared (or common) environmental (C), and nonshared (or unique) environmental (E) components were fit to the individual criterion-level twin data on the 1073 complete twin pairs and the 623 single twins. The various independent pathway models were fit to 2-group (monozygotic/dizygotic, male/male same-sex twin pair) raw data using full-information maximum likelihood estimation as implemented in Mx.[22] Each observed ordinal CD criterion was modeled as a set of estimated ordered thresholds on a normally distributed, continuous latent liability/response variable. Parameter estimation was carried out by integrating across these latent variable continua. Different threshold estimates were allowed for monozygotic and dizygotic male twin pairs. We began by fitting a "111_111" baseline model in which twin resemblance among the 10 CD criteria was posited to be adequately accounted for by single common additive genetic, shared environmental, and nonshared environmental components along with criterion-specific additive genetic, shared environmental, and nonshared environmental effects. Models omitting the common and criterion-specific C components, and then including additional A and E common components, were tested next. Criterion-specific nonshared environmental effects were not set to zero in any of the models tested because this entails the unrealistic assumption that individual responses to items were reported without error. Final orthogonal factor loadings were obtained using a varimax rotation.

The goal of model fitting was to achieve an optimal balance of explanatory power and analytic parsimony. We operationalized this goal by using the Bayesian information criterion (BIC), which performs particularly well with more complex models of the kind evaluated herein.[23] Lower values of BIC indicate relatively better fit.

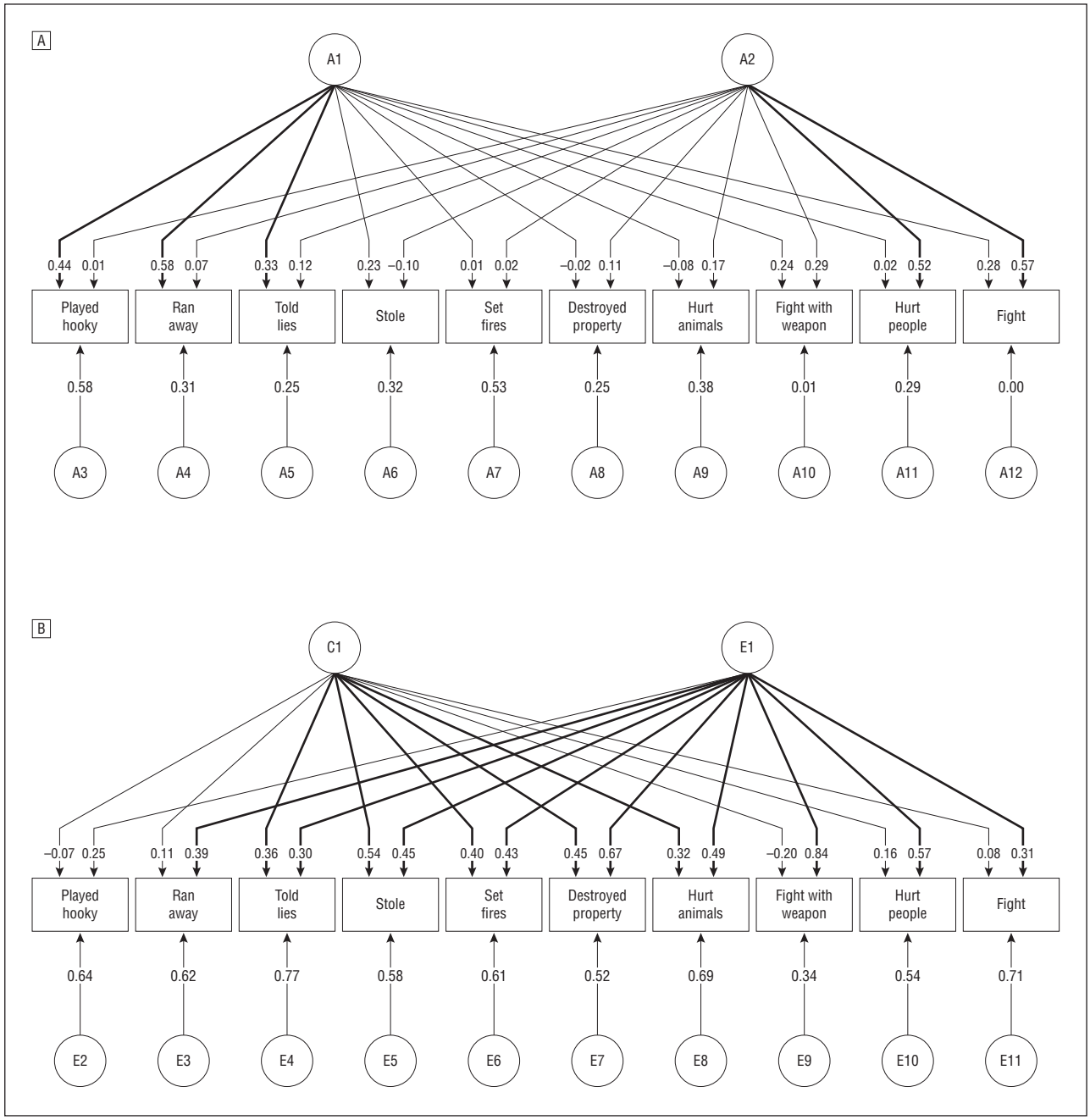

Figure. Genetic and shared and nonshared environmental parameter estimates from the best-fitting model (model 8) for the *DSM-III-R* A criteria for conduct disorder. A, Genetic parameter estimates. Two of the 13 *DSM-III-R* criteria (robbed or mugged someone and forced sex) were omitted from analyses because of low observed or expected prevalence, and 2 others (broken into house, building, or car and stolen without confrontation) were combined into a single item. For common factors, paths +0.30 or more are bolded. To calculate the proportion of variance in liability of a criterion that is estimated to be accounted for by the genetic or environmental factors, the path coefficient needs to be squared. B, Shared and nonshared environmental parameter estimates. For common factors, paths +0.30 or more are bolded.

Based on the combination of common factors for the best-fitting model, maximum likelihood genetic factor scores were estimated by computing the conditional likelihood of the twin pairs' item responses, weighted by the joint likelihood of the factor score estimates. This factor scoring model was iteratively fitted, separately for each twin zygosity group, to each twin pair's raw data to obtain estimates of scores on the factors of the best-fitting model for each individual.

To examine the validity of the scores for the 2 genetic factors and 1 shared environmental factor, 2 types of analyses were performed. First, separate regressions for these 3 factor scores were conducted to evaluate differences in the magnitude of the association with each outcome variable. Second, we examined whether we could constrain regression coefficients for all 3 factor score variables to be equal in predicting each outcome variable. The robust weighted least-squares mean- and variance-adjusted estimator in Mplus version 6.0[24] was used to obtain estimates for these models. In this approach, probit regression coefficients are estimated for each of the genetic factor score variables. Since the estimated genetic factor scores are calibrated on a uniform standard scale, the effect size units are more readily interpretable when comparing coefficients across the different factor score variables. Age was treated as a nonnormal continuous variable and fit using the MLR estimator in Mplus.

Table 1. Summary of Model-Fitting Results for *DSM-IV* Conduct Disorder Criteria in Males

Model No.	$A_cC_cE_c\_A_sC_sE_s$ [a]	–2 Log Likelihood	*df*	BIC	Δ BIC
1 [b]	111_111	31 717.0	27 419	–86 101.8	
2	111_101	31 729.4	27 429	–86 132.8	–31.0
3	111_011	31 730.6	27 429	–86 132.2	–30.4
4	111_001	31 839.0	27 439	–86 115.2	–13.4
5	011_101	31 860.3	27 439	–86 104.5	–2.7
6	101_101	31 861.2	27 439	–86 104.1	–2.3
7	110_101	32 174.7	27 439	–85 947.4	+154.4
8 [c]	211_101	31 649.7	27 420	–86 139.2	–37.4
9	121_101	31 659.9	27 420	–86 134.1	–32.3
10	112_101	31 678.4	27 420	–86 125.8	–23.0
11	311_101	31 613.1	27 412	–86 127.7	–25.9
12	221_101	31 620.6	27 411	–86 120.3	–18.5
13	212_101	31 658.5	27 411	–86 101.3	+0.5

Abbreviation: Bayesian information criterion.

[a] Respectively: number of additive genetic (A), shared environmental (C), and nonshared environmental (E) common factors where the subscript *C* stands for common and presence (1) vs absence (0) of criterion-specific additive genetic (A), shared environmental (C), and nonshared environmental (E) common factors where the subscript *S* stands for specific.

[b] Selected baseline comparison model: –2 log likelihood = 31 717.0; *df* = 27 419; BIC = –86 101.8.

[c] Best-fitting model according to BIC.

Exploratory phenotypic factor analysis was performed on the individual phenotypic data in Mplus with varimax rotation. Overall model fit was evaluated using the Tucker-Lewis Index (TLI),[25] the Comparative Fit Index (CFI),[26] and the root mean square error of approximation (RMSEA).[27] For the TLI and CFI, values between 0.90 and 0.95 are considered acceptable and 0.95 or more, as good. For the RMSEA, good models have values of 0.05 or less.

RESULTS

MODEL FITTING

Table 1 depicts the multivariate twin models we evaluated. The initial baseline model (1) included single genetic, shared environmental, and nonshared environmental common factors along with criterion-specific genetic, shared, and nonshared environmental influences. Next, we compared the fit of alternative models that omitted, respectively, the criterion-specific shared environmental factor (model 2), the criterion-specific genetic factor (model 3), and then both together (model 4). The BIC values for models 2 and 3 were both clearly superior to BIC for model 1, with fit being slightly better for model 2 than model 3. The BIC for model 4, which omitted both the criterion-specific shared environmental factor and the criterion-specific genetic factor, was in turn much worse. Therefore, working from model 2, we then dropped (in models 5, 6, and 7, respectively) the single genetic, shared environmental, and nonshared environmental common factors. The BIC values for these 3 models were all substantially poorer than for model 2. Since simplification of model 2 did not improve the fit, we then sought to improve fit by including additional common factors. Specifically, in models 8, 9, and 10, respectively, a second genetic, shared, or nonshared environmental common factor was added. Models 8 and 9 were both superior to model 2 in BIC values, with BIC for model 8 appreciably better than for model 2.

Finally, to evaluate whether the fit of model 8 could be improved further, variants of this model that included a third genetic common factor (model 11) or a second shared or nonshared environmental common factor (models 12 and 13, respectively) were fitted. The BIC was worse for all 3 of these models than that for model 8, indicating the "best" fit for this model relative to others.

The Figure shows the parameter estimates for the best-fitting model 8. Focusing on factor loadings of 0.30 or more, 6 features of this model are noteworthy. First, 1 of the genetic factors is marked by prominent selective loadings for 2 CD criteria, running away from home and playing hooky, with a somewhat lesser loading for telling lies. We interpreted this genetic factor as reflecting rule breaking.[7,8,11] Second, the other genetic factor is marked by substantial loadings for 2 other CD criteria, starting fights and hurting people, with a lesser loading for using a weapon in a fight. The indicators of this factor and their relative loadings mirror findings of Tackett et al.[9] We interpreted this factor as reflecting overt aggression. Third, the single shared environmental common factor shows appreciable loadings for 5 CD criteria, 3 of which (destroying property, setting fires, and hurting animals) do not load on either genetic factor and 2 others (telling lies and stealing) that load secondarily on the rule-breaking genetic factor. We interpreted this factor as reflecting covert delinquency. Fourth, the single nonshared environmental common factor exhibits a less distinctive patterning of loadings for most CD criteria, with the exception of high loadings (>+0.50) for using weapons, destroying property, and hurting people. Fifth, prominent criterion-specific genetic factors were evident for 5 of the CD criteria: hooky, setting fires, hurting animals, stealing, and running away. Finally, prominent nonshared criterion-specific environmental influences (which also include errors of measurement) were evident for all CD criteria.

Table 2 provides a complementary perspective on the results of this best-fit model. This Table depicts es-

Table 2. Estimated Total Genetic and Shared and Nonshared Environmental Contributions to the Liability Toward *DSM-III-R* Conduct Disorder Criteria[a]

	Genetic Influences				Shared Environment	Nonshared Environmental Influences		
Conduct Disorder Criteria	Total a^2	Factor 1: Rule Breaking, %	Factor 2: Overt Aggression, %	Specific, %	Total c^2 Covert Delinquency, Single-Factor %	Total e^2	Factor 1, %	Specific, %
Played hooky	**0.53**	**37**	0	63	0.00	**0.47**	13	87
Run away	**0.44**	**77**	1	22	0.01	**0.55**	29	71
Told lies	0.19	**58**	9	33	0.13	**0.68**	13	87
Stole	0.17	**32**	6	62	0.29	**0.54**	38	62
Set fires	0.28	0	0	100	0.16	**0.56**	33	67
Destroyed property	0.08	1	16	83	0.20	**0.72**	**62**	38
Hurt animals	0.18	4	16	80	0.10	**0.72**	**33**	67
Fight with weapon	0.14	**41**	**59**	0	0.04	**0.82**	**86**	14
Hurt people	**0.35**	0	**77**	23	0.03	**0.62**	**52**	48
Fight	**0.40**	20	**80**	0	0.01	**0.60**	16	84

[a] In the columns for Total variance and Factor % under the "Genetic Influences" and "Nonshared Environmental Influences" headings, values +0.30 or more are bolded. No bolding is given for the criterion-specific values. Only a single (Total variance) column is included under "Shared Environment" because the best-fitting structural model contained only a single common factor, with no criterion-specific shared environmental (C) effects.

timated percentages of overall variance in liability to each of the CD criteria attributable to the aforementioned genetic, shared environmental, and nonshared environmental influences and decomposes variance associated with each into portions reflecting common factors and criterion-specific influences. Focusing first on variance in individual symptoms attributable to genetic vs environmental influences: (1) In general, nonshared environmental influences account for the greatest proportion of variance in CD symptoms (>45% in all cases), followed by genetic influences (>15% in all but 2 cases), with shared environment accounting for the least (>5% in only 5 cases). (2) The relative contribution of genes to overall variance is highest for the 2 criteria that most strongly define the rule-breaking factor (running away and hooky) and the 2 that most strongly define the overt aggression factor (fights and hurting people). (3) The 5 criteria for which the contribution of shared environment is highest (10%-29%) are those that define the covert delinquency factor.

Regarding relative contributions of common and criterion-specific influences to genetic and unique environmental components of symptom variance, the following notable patterns are evident in Table 2: (1) Genetic common factors (rule breaking and overt aggression) contribute substantially more than criterion-specific genetic influences to 3 of the 4 most heritable symptoms (running away, fights, and hurting people) and to 2 other modestly heritable symptoms (telling lies and using a weapon). (2) Criterion-specific genetic influences contribute prominently to 2 symptoms (hooky and stealing) that evidence subsidiary contributions from one or the other genetic common factor. (3) Criterion-specific genetic influences account for most or all of the modest heritable variance in 3 of the 5 symptoms (setting fires, destroying property, and hurting animals) that define the shared environmental (ie, covert delinquency) common factor. (4) The nonshared environmental factor accounts for more than 50% of the nonshared environmental influence in 3 of the CD criteria (destroying property, using a weapon, and hurting people), whereas criterion-specific nonshared environmental effects (which include measurement error) contribute more substantially to the other 7 criteria.

VALIDATION OF THE FAMILIAL FACTORS

Factor scores for the 3 familial factors—2 genetic and 1 common environmental—were computed for each twin. As depicted in **Table 3**, we then examined the association between these factor scores and the domains of personality, psychiatric disorders, and demographics. In the domain of personality, both the rule-breaking (A1) and overt aggression (A2) factors were significantly and positively related to neuroticism, whereas the covert delinquency factor (C1) was not. Rule breaking was positively related to extraversion whereas covert delinquency showed a negative association, with overt aggression unrelated to extraversion. All 3 familial factors were significantly and positively associated with novelty seeking, although prediction was stronger for the rule-breaking factor than for overt aggression or covert delinquency.

With regard to psychiatric disorders, the 3 familial factors did not differ significantly in their association with a lifetime diagnosis of GAD, although when examined individually, regression coefficients were significant for rule breaking and overt aggression but not covert delinquency. However, the 3 factors differed significantly in their association with antisocial personality disorder symptoms, with the effect size especially strong for overt aggression, slightly weaker for rule breaking, and much weaker (albeit significant) for covert delinquency. The 3 familial factors did not differ significantly in their association with cocaine and alcohol dependence, although the regression coefficient appeared weakest in each case for covert aggression. Interestingly, whereas cocaine dependence was associated most strongly with overt aggression, alcohol dependence was associated most strongly with rule breaking.

Table 3. Predictive Relations for 2 Genetic Factors (A1 and A2) and 1 Shared Environmental (C1) Common Factor With External Outcome Variables[a]

Model Predictors (Factor Scores)	Criterion Variable	Estimated Effect Size (β Coefficient)	Robust χ^2_2 Test for Constrained Model	*P* Value
A1→	Neuroticism	0.235	=	<.001
A2→		0.215	=	<.001
C1→		–0.036	=	.37
A1 = A2 = C1		R^2 = 0.02	20.2	<.001
A1→	Extraversion	0.140	=	.001
A2→		–0.052	=	.27
C1→		–0.076	=	.04
A1 = A2 = C1		R^2 = 0.01	9.4	.001
A1→	Novelty seeking	0.300	=	<.001
A2→		0.129	=	.007
C1→		0.091	=	.02
A1 = A2 = C1		R^2 = 0.06	7.1	.03
A1→	*DSM-5* LT GAD	0.224	=	.002
A2→		0.166	=	.03
C1→		0.067	=	.30
A1 = A2 = C1		R^2 = 0.05	1.9	.40
A1→	Number of ASPD criteria	0.424	=	<.001
A2→		0.517	=	<.001
C1→		0.259	=	<.001
A1 = A2 = C1		R^2 = 0.16	26.2	<.001
A1→	Cocaine dependence	0.278	=	.003
A2→		0.343	=	<.001
C1→		0.175	=	.03
A1 = A2 = C1		R^2 = 0.06	2.7	.25
A1→	Alcohol dependence	0.325	=	<.001
A2→		0.270	=	<.001
C1→		0.174	=	.001
A1 = A2 = C1		R^2 = 0.07		.26
A1→	Educational level	–0.619	=	<.001
A2→		0.087	=	.08
C1→		0.403	=	<.001
A1 = A2 = C1		R^2 = 0.11	114.4	<.001
A1→	Age	0.088	=	.85
A2→		–2.515	=	<.001
C1→		–1.165	=	.005
A1 = A2 = C1		R^2 = 0.02	17.2	<.001

Abbreviations: ASPD, antisocial personality disorder; GAD, generalized anxiety disorder; LT, lifetime.

[a]The A1 factor is termed *rule breaking*; the A2 factor, *overt aggression*; and C1 is termed *covert delinquency*. All ordinal coefficient invariance tests were performed in Mplus[24] using the robust weighted least-squares mean- and variance-adjusted estimator χ^2 difference test.

We then tested for differential association of the 3 familial factors with education and age at diagnostic interview. For education, robust negative and positive coefficients, respectively, were evident for the genetic rule-breaking factor and the shared environmental (covert delinquency) factor. By contrast, the genetic overt aggression factor was not associated with educational level. A quite different pattern was found for age. Rule breaking did not emerge as a significant predictor, but both overt aggression and covert delinquency exhibited a relatively strong inverse association with age.

Finally, using exploratory phenotypic factor analysis, we fitted 1- and 3-factor solutions to the CD criteria. The 3-factor model fit better on all indices (CFI = 0.98, TLI = 0.94, RMSEA = 0.04 vs CFI = 0.89, TLI = 0.86, RMSEA = 0.07). As seen in **Table 4**, the 3 phenotypic factors closely resembled, respectively, the first rule-breaking genetic common factor, the covert delinquency shared environmental common factor, and the second overt aggression genetic common factor.

COMMENT

The goal of this report was to delineate, for the first time to our knowledge, the contributions of genetic and environmental etiologic influences to the individual *DSM-III-R* criteria for CD and, in the process, advance conceptualization of distinctive dimensions underlying CD symptoms. Our best-fitting model indicated 3 familial common factors (2 genetic and 1 shared environmental) and 1 nonshared environmental common factor contributing to CD symptoms, along with criterion-specific genetic and nonshared environmental factors. Each of the 3 familial factors exhibited readily interpretable loadings for individual CD criteria. Consistent with prediction, the 2 genetic factors coincided with dimensions identified most consistently in prior work. The first evidenced strongest loadings for select CD criteria reflecting rule-breaking or authority-challenging behaviors. The second showed substantial loadings for 3 other criteria that

Table 4. Factor Loadings From a 3-Factor Exploratory Phenotypic Factor Analysis of *DSM-III-R* Conduct Disorder Criteria[a]

Conduct Disorder Criteria	Factor 1	Factor 2	Factor 3
Played hooky	**0.50**	0.04	0.13
Run away	**0.70**	0.23	0.21
Told lies	**0.35**	**0.38**	0.18
Stole	0.31	**0.71**	0.01
Set fires	0.09	**0.57**	0.17
Destroyed property	0.14	**0.69**	0.36
Hurt animals	0.01	**0.52**	0.35
Fight with weapon	0.41	0.29	**0.65**
Hurt people	0.12	0.31	**0.75**
Fight	0.30	0.11	**0.57**

[a]Varimax rotation. The strongest loading for each individual criterion is bolded with the exception of told lies where the top 2 loadings, which were quite similar, are both bolded. Factor 1 closely resembles the first rule-breaking genetic common factor. Factor 2 closely resembles the single shared environmental common factor, labeled covert delinquency. Factor 3 closely resembles the second overt aggression genetic common factor.

reflected public, interpersonal aggressive acts. We proposed the label *overt aggression* for this factor. The shared environmental common factor evidenced strong loadings for 5 CD criteria, 4 reflecting less readily observable antisocial behaviors directed at nonhuman targets and the fifth (telling lies) entailing concealment by definition. We proposed the term *covert delinquency* for this factor. By contrast, the nonshared environmental common factor was less readily interpretable, having substantial loadings on all but 1 of the assessed CD criteria.

The cleanest indicators of the genetic rule-breaking and overt aggression factors were, respectively, criteria reflecting truancy and running away and hurting people and initiation of fights. These criteria loaded selectively on 1 genetic common factor or the other, exhibited prominent individual heritabilities ($a^2 \geq 0.35$) traceable in large part to their affiliated genetic factor, and had negligible contributions of shared environment ($c^2 \leq 0.03$). These findings diverged somewhat from those of Barker et al,[28] who reported evidence of a common genetic factor underlying teacher-rated behaviors classified as "aggressive" and "deceptive." Aside from differences in mode of assessment (ie, teacher ratings vs self-report) and model specification (ie, correlated vs uncorrelated factors), the 2 studies differed in the number and types of behavioral indicators used. In particular, 3 of the 4 *DSM*-based indicators in the current study that "pulled" most strongly for separate genetic factors (ie, truancy, running away, and hurting people) were not represented in the Barker et al study, which relied on non-*DSM* indicators.

A third indicator of the genetic rule-breaking factor, telling lies, exhibited weaker heritability and comparable convergence with the shared environmental common factor. Notably, this result converges with the findings of Barker et al,[28] who reported a shared environmental contribution to deceptive but not aggressive behaviors. A third indicator of the genetic overt aggression factor, used weapon, also exhibited weak heritability and only slightly weaker convergence with the genetic rule-breaking factor. These 2 criteria may be points of intersection between the rule-breaking and covert delinquency factors and the overt aggression and rule-breaking factors, respectively. The cleanest indicators of the shared environmental covert delinquency factor were criteria reflecting fire setting, property destruction, and hurting animals because they loaded prominently on the shared environmental factor and weakly on either genetic factor. A fourth indicator of the nonshared covert delinquency factor, stealing, modestly cross-loaded on the genetic rule-breaking factor. This criterion represents a further point of intersection (with telling lies) between the genetic rule-breaking factor and the shared environmental covert delinquency factor.

We also evaluated the association of these 3 familial factors with relevant measures of personality, psychopathology, and demography. For many outcome variables examined, the magnitudes of the associations differed meaningfully for the 3 factor scores. The finding of robust relations for the rule-breaking factor with traits of novelty seeking and neuroticism suggests that this component of CD relates most closely to the disinhibitory-externalizing dimension of psychopathology identified in the child and adult clinical literatures.[13,29,30] Also consistent with this interpretation are the robust positive relations of this factor with anxious-dysthymic (internalizing) tendencies in the form of GAD and alcohol dependence and its moderate negative relationship with educational attainment. By contrast, the more selective association of the overt aggression factor with the broad trait of neuroticism (encompassing hostility and mistrust, as well as anxiousness) and its lesser relationship with the narrower GAD variable (reflecting general anxiousness and dysphoria), coupled with its strong association with adult antisocial personality disorder symptoms, appears consistent with a more callous-antagonistic disposition, perhaps indicative of the "meanness" facet of psychopathy.[12,31,32] Finally, a 3-factor exploratory phenotypic factor analysis exhibited a better fit to our data than a 1-factor model. The factor loading pattern for this model closely followed the pattern of loadings found for the 3 common factors detected in our multivariate twin analysis.

LIMITATIONS

The current findings help to reconcile overlapping but somewhat contrasting conceptualizations of CD symptom dimensions reported in the literature. The first genetic common factor of our model captures a narrower version of the rule-breaking factor identified in prior studies[7-9,12] that appears similar to the "authority conflict" dimension described by Loeber and Hay.[10] Our second genetic common factor reflects antisocial tendencies characterized in differing studies as "aggressive"[7,9] or "overt."[8,10] The shared environmental common factor of our model combines aspects of delinquent and covert dimensions identified in prior studies.[10,11] Beyond the level of phenotypic description, our findings tie these thematic subdimensions of CD to distinctive etiologic determinants. Prior research points to an especially strong role for genetic influences and only a weak role for shared environmental influences in callous-aggressive forms of

CD.[9,12,31,33] Consistent with this, criteria that emerged as indicators of the overt aggression factor in the current study showed clear contributions of genetic influences but minor or negligible contributions of shared environment. By contrast, prior research points to lesser genetic and more pronounced shared environmental contributions to nonaggressive forms of CD.[9,12] The current study clarifies this picture by indicating a more prominent role for genetic influences in rule-breaking symptoms of CD and a more prominent role for shared environmental influences in covert delinquent tendencies. The implication is that studies that combine the 2 into a single nonaggressive symptom category will tend to find weaker genetic-etiologic effects than for aggressive symptoms and stronger shared environmental effects.[12]

Further work is needed to clarify what aspects of covert delinquent behavior are especially susceptible to shared environmental influences. Perhaps some such behaviors (eg, destroying property, setting fires, and stealing) reflect antisocial activities in which young male twins are particularly likely to collude with co-twins, regardless of zygosity. The selective positive association of this factor with the trait of novelty seeking, coupled with its positive relations with antisocial personality disorder symptoms, alcohol/drug dependence, and (somewhat counterintuitively) higher educational attainment, could reflect an "open"/experience-seeking disposition conducive to experimentation. Alternatively, it could be that certain adverse environments contribute generally to behaviors of this type by fostering isolation and hostile feelings. The observed negative association of the covert delinquency factor with extraversion appears consistent with this latter possibility, although its null associations with neuroticism and GAD do not.

Our findings can be usefully compared with results from recently completed parallel analyses with *DSM-IV* criteria for antisocial personality disorder.[34] Using the same methods and sample, we found evidence for 2 genetic factors underlying the antisocial personality disorder criteria, labeled aggressive-disregard and disinhibition, but no shared environmental common factor. The aggressive-disregard factor—characterized by prominent loadings on criteria assessing fighting and reckless disregard—resembles our CD overt aggression genetic factor. The disinhibition factor—with stronger loadings on criteria reflecting failure to plan and being deceitful and irresponsible—bears some similarity to our rule-breaking genetic factor.

The results of the current study provide an illustration of the capacity for genetically informed designs to dissect complex phenotypes in ways difficult to accomplish with other designs. Neither epidemiological nor family designs could distinguish our covert delinquency from the rule-breaking and overt aggression factors. This was only possible because of the ability of the twin design to discriminate environmental from genetic sources of familial resemblance.

The current findings should be interpreted in the context of 6 potential methodological limitations. First, we examined only twins from male-male pairs born in Virginia. (Endorsement rates of several CD criteria in female-female pairs were too low for meaningful analysis.) Our results may not extrapolate to females or other ethnic groups. Second, our analyses used CD criteria from *DSM-III-R*[17] not *DSM-IV*.[1] Two criteria were omitted because of rarity, and 2 others were collapsed. However, 9 of the 10 *DSM-III-R* items assessed were similar to *DSM-IV* CD criteria, with all criterion subsets represented: aggression to people/animals (3 items), property destruction (2 items), deceitfulness/theft (2 items), and serious rule violations (2 items). Third, CD criteria in the current study were assessed by self-report questionnaire, not structured interview. However, for socially undesirable traits like CD behaviors, research suggests that more accurate responses are obtained by more anonymous means of assessment.[35] Furthermore, several prior studies support the validity of self-report assessment of CD.[36-38] Fourth, CD symptoms were reported retrospectively. We cannot, therefore, rule out a substantial effect of memory bias. It is, however, difficult to construct a plausible scenario in which monozygotic twins are more highly correlated than dizygotic twins in their biases for recall of some items (those loading on the 2 genetic factors) while equally correlated in their biased recall for other sets of items (those loading onto the common environmental factor). Furthermore, Nock et al recently reviewed the literature on retrospective reports of CD symptoms and concluded that, despite evidence for underreporting of less deviant behaviors, ". . .adults recall childhood experiences with sufficient accuracy to provide useful information in retrospective studies."[39(p708)] Fifth, our modeling assumed that genetic and environmental factors acted additively on risk for CD symptoms and were uncorrelated. Finally, few subjects in our sample endorsed large numbers of CD criteria. Thus, most of the information used in our analyses comes from individuals with modest numbers of symptoms.

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