ORIGINAL ARTICLE

Distinct Contributions of Conduct and Oppositional Defiant Symptoms to Adult Antisocial Behavior

Evidence From an Adoption Study

Douglas R. Langbehn, MD, PhD; Remi J. Cadoret, MD; William R. Yates, MD; Edward P. Troughton, BA; Mark A. Stewart, MD†

Background: We conducted an exploratory multivariate analysis of juvenile behavior symptoms in an adoption data set. One goal was to see if a few *DSM*-interpretable symptom dimensions economically captured information within the data. A second goal was to study the relationships between any such dimensions, biological and environmental background, and eventual adult antisocial behavior.

Methods: The data originated from a retrospective adoption study. Probands with a biological background for parental antisocial personality or alcoholism were heavily oversampled. Symptoms were ascertained by proband and adoptive parent interview. We performed, by gender, orthogonal rotated principal component analyses of juvenile behavior disturbance symptoms (females, n = 87; males, n = 88). We used structural equation modeling to examine the relationships hypothesized above.

Results: For both genders, an oppositional defiant disorder (ODD) component and at least 1 conduct component emerged. Regardless of the conduct component scores, the ODD components were significant predictors of adult antisocial behavior. For males, the ODD component was predicted by an antisocial biological background, but not by scores on the Adverse Adoptive Environment Scale. The conduct components were predicted by adoptive environment alone. For females, biological background or biological-environmental interactions predicted each of the components.

Conclusions: There has been little previous distinction between conduct disorder and ODD in studies of genetic and environmental influences on juvenile behavior. The study suggests that adolescent ODD symptoms may be a distinct antecedent of adult antisocial personality. In males, adolescent ODD symptoms may represent early expression of genetic sociopathic personality traits.

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From the Departments of Psychiatry, VA Hospital (Dr Langbehn), University of Iowa (Drs Langbehn, Cadoret, and Stewart and Mr Troughton), Iowa City; and University of Oklahoma College of Medicine, Tulsa (Dr Yates).

†Deceased.

ASSOCIATIONS have been found repeatedly between both childhood disruptive behavioral disorders and adult sociopathy in parents and similar disorders in their offspring.[1-7] Furthermore, separate genetic and environmental influences—and perhaps a synergistic interaction—have been demonstrated many times for these same disorders. This assertion is supported by an extensive literature consisting of both adoption[8-19] and twin studies[20-22] Other reports show that juvenile behavioral disturbances are strong predictors of adult antisocial behavior.[2,5,23-27]

Within this literature, there has been little attempt to distinguish between specific disruptive behavior patterns, at least in terms of *DSM* concepts such as conduct disorder and oppositional defiant disorder (ODD).[28] This is despite the fact that distinct symptom dimensions approximating these concepts have been supported in many factor analytic studies of childhood or adolescent behavior data.[7,28-33] Instead, all such behaviors have typically been lumped into one general category[34,35] or separated into alternative taxonomies such as delinquency vs aggressivity[18] or covert vs overt conduct.[36] One research group that has considered the role of specific *DSM* diagnoses has postulated that a certain severe, early form of ODD is often the first manifestation of poor-prognosis conduct disorder.[37] Another study suggests that either conduct disorder or ODD is a familial risk factor for antisocial behavior, at least in the presence of attention-deficit problems.[4] Beyond this, the role of ODD does not seem to be well examined.

This article is an exploratory reanalysis of adoption data previously presented by Cadoret et al.[17-19] In the original analysis,[18] high scores on the Adverse Adoptive Environment Scale and a biological background that included a sociopathic parent were both significant risk factors

SUBJECTS AND METHODS

STUDY DESIGN AND RECRUITMENT

The design and methodology of the original adoption study are described in detail in 2 articles by Cadoret et al.[17,18] Key elements are reviewed here.

Subjects were recruited from 4 Iowa adoption agencies. Two groups of potential subjects were identified. The first was defined by evidence of antisocial personality and/or alcohol or substance use problems in at least 1 biological parent. The second group had no evidence of either the above or any other psychiatric diagnosis. The groups were matched 1:1 on the basis of adoption agency, gender, age, and age of biological mother at the time of birth. (For the present analysis, biological parents with a history of alcohol/substance abuse but no sociopathy were pooled with the original control group.)

All adoptees were separated from their biological families within a few days of birth and eventually adopted by nonrelatives. The adoptees were between 18 and 47 years old at the time of the study.

Adoptive parents of potential participants were initially contacted by their respective adoption agencies. If agreeable, they were contacted by the investigators. Written informed consent was obtained from all adoptees and adoptive parents. We were careful to preserve the confidentiality of all adoptive family and biological parent information throughout the initial contact procedure and subsequent study. The investigators were blinded as to the identity of the biological parents. All methods were approved by the Human Subjects Internal Review Board of the University of Iowa College of Medicine, Iowa City.

The refusal rate was 54%. Refusal was not correlated with biological parent status. The final data included 197 adoptees, 95 males and 102 females.

BIOLOGICAL PARENT DIAGNOSIS

Initially, 11 700 biological parent names were compared with the records of all Iowa public mental health hospitals and correctional facilities. Apparent matches were confirmed by comparison of ancillary family and demographic information. Institutional records (coded to preserve the parents' anonymity) were reviewed independently by 3 psychiatrists (R.J.C., W.R.Y., and M.A.S.). Diagnoses were assigned using *DSM-III-R*[38] criteria. The pairwise κ statistics[39] for interrater agreement were deemed satisfactory, ranging from 0.67 to 0.79 for antisocial personality disorder. Disagreements were settled by conference between the raters. If no agreement was reached, the subject was excluded. Biological parent status of the adoptees analyzed in this article were as follows: antisocial only, n = 18; antisocial and alcohol/substance abuse, n = 28; alcohol/substance abuse only, n = 30; neither diagnosis, n = 99.

ADOPTEE, ADOPTIVE PARENT, AND ENVIRONMENTAL ASSESSMENT

The adoptees and their parents were interviewed separately. Nearly all adoptees and parents were interviewed within 6 months of each other. Interviewer assignments were based on geographic convenience. Thus, the same person sometimes conducted both interviews. Interviewers were blinded as to biological parent status.

Adoptees were administered the *Diagnostic Interview Schedule, Version III, Revised* (*DIS*)[40] and a structured interview assessing the adoptive environment. A similar interview was administered to adoptive parents. This interview contained sections on emotional and psychiatric problems within the adoptive family and adverse environmental conditions during the adoptee's childhood. The parents also received the computer-administered *DIS* screening interview. Adoptee and parent *DSM-III-R* diagnoses were based on the *DIS*. Parents also answered a series of questions regarding adoptee "aggressivity" based on the scale developed by Loney et al[41] (5 new items were added). Ages 6 to 12 and 13 to 18 years were covered separately. Ratings for the Adverse Adoptive Environment Scale were based on both the adoptee and parent interviews. Items were considered positive if attested to on either interview.

The Adverse Adoptive Environment Scale has been described previously by Cadoret et al.[17] It consists of the total of the following parental conditions met while the adoptee was in the home: divorce or separation, presence of other

for adult antisocial symptoms in the adoptees. There was also evidence that a synergistic interaction between these effects was a significant risk factor for symptoms of juvenile (age <15 years) conduct disorder and childhood and adolescent "aggressivity." The relationship between juvenile and adult behavior was not examined.

We have now used a descriptive multivariate statistical method to examine the gender-specific relationships between the individual symptoms that comprised the juvenile conduct and adolescent aggressivity scales in the original article. We used the results of this analysis in models of the ultimate effects of biological parent sociopathy and adverse adoptive environment on adult antisocial behavior. Our purpose was to examine whether uncorrelated biological and environmental influences predict adult antisocial behavior via pathways involving juvenile symptom dimensions that, in adoptees, are also uncorrelated. We also considered the relationship between these symptom dimensions and the *DSM* concepts of conduct disorder and ODD.

The adoption paradigm, which allows a separation of the genetic from the environmental influence of having antisocial parents, distinguishes this study from other multivariate analyses of the conduct/oppositional defiant disorder distinction.

RESULTS

PRELIMINARY ANALYSIS

Univariate descriptive statistics for variables relevant to our models are presented in **Table 1**. Note the higher symptom rates in males. **Table 2** describes the individual items from the original conduct and adolescent aggressivity scales. We indicate the status of these symptoms (in our judgment) relative to *DSM* definitions of conduct disorder and

marital problems, alcohol/substance abuse or dependence, major depression, anxiety disorders, other psychiatric conditions, and legal problems. Using the current data, this scale has been shown to be a predictor of several adoptee psychiatric outcomes, either as a main effect or in interaction with biological parent variables.[17-19,42] A nearly identical scale's predictive value for male adolescent antisocial behavior was shown in an earlier adoption study involving different data.[9,13]

Conduct disorder and adult antisocial symptoms in adoptees were determined primarily by the *DIS*. For several symptoms, relevant parent interview information was also available. In these cases, a symptom was considered present if reported by either source. Adult antisocial symptoms had to occur after age 18 years (rather than 15 years). The adolescent aggressivity scale items were derived solely from the parental reports covering ages 13 to 18 years. No relevant information from the adoptee interviews was available.

STATISTICAL METHODS

Our analysis focuses on conduct symptoms and items from the adolescent aggressivity scale. We refer to these collectively as the "behavior data."

We excluded 9 males and 15 females from the analyses because of incomplete data. They did not differ significantly from the remaining subjects in terms of mean Adverse Adoptive Environment Scale scores, adult antisocial symptoms, or antisocial biological parent rate.

We used *t* tests for simple comparisons of count data. Rate comparisons were based on the Pearson χ^2 test or the Fisher exact test if less than 25% of the cells had expected null hypothesis counts of 5 or less.[39] We analyzed the males and females separately. We discuss the justification for this in the "Results" section.

We used principal components analysis with varimax (orthogonal) rotation[43] to explore the most important underlying dimensions of the juvenile behavior data. This is a statistical method that identifies a lower-dimensional "space" in which a large portion of the overall data variability occurs. The principal components form axes that define dimensions of this space. The correlations between these dimensions and the original symptoms can be estimated, and an individual's symptom pattern can be translated into scores on these dimensions.

The analysis was conducted on the correlation (φ coefficient) matrices. The decision of how many components to retain is ultimately subjective. We based our decisions primarily on breaks in the eigenvalue patterns (a "scree plot" analysis). However, interpretability of the results also played a role.

We used path analysis[44] for the second part of our evaluation. This is a method for combining the results of several individual linear regressions into one overall model. A variable may serve both as an outcome for one portion of the analysis and, at the same time, as a predictor for another portion. Variables that serve only as predictors are referred to as "exogenous" variables. All other variables are "endogenous."

These models explored the evidence for various possible paths leading to adoptee adult antisocial symptoms. The paths began with the following exogenous variables: antisocial biological parent, Adverse Adoptive Environment Scale score, and the multiplicative interaction between them. The adoptees' estimated scores on each of the principal component dimensions served as intermediate variables in the paths, potentially predicted by the exogenous variables and, in turn, potentially predictive of adult antisocial symptoms. Because component scores were uncorrelated by design, there was no need to consider possible paths between them.

We initially examined saturated models in which all exogenous variables were fit as predictors of each behavior dimension. At the same time, we tested the exogenous variables plus all juvenile behavior dimensions as potential predictors of adult antisocial symptoms. These models were pruned in a stepwise fashion by successively removing predictors with $P > .05$.

No "latent" variables were inferred as the path analyses were fit. Therefore, the mathematical requirements for valid inference are the same as those for the individual regressions that compose the overall analysis.[44] We checked the adequacy of these assumptions in each case. Residual distributions were examined for marked nonnormality or nonrandomness. We also checked the influence of possible outliers on model fit. No data transformations or deletions were deemed necessary.

ODD. Several items were rarely positive. These items, indicated in Table 2, were excluded from principal component analyses because they would have made the results quite unstable. Again, there is marked variation in the rates of most behaviors between males and females. Because of this, the further analyses were separated by gender.

MALES

Table 3 contains the principal components results for males. The analysis using the first 3 components was most easily interpreted and was also supported by a small break in the eigenvalue pattern.

The first component correlates primarily with ODD symptoms (aggressivity scale stealing is an exception). The second component, uncorrelated with the first, is most strongly related to conduct symptoms. A third uncorrelated component is difficult to interpret, but seems to contrast physical aggression vs early experience with substance abuse and sex. Combined, these 3 components account for 49% of the estimated common variance in the male data.

The path analysis reported in **Table 4** and illustrated in **Figure 1** incorporates the regression relationships involving these principal components, antisocial biological parentage, adverse adoptive environment, and adult antisocial symptoms. Antisocial biological background, but not adverse environment, is a significant predictor of the ODD component. The converse is true for the conduct component. Neither variable nor their interaction predicts the third component. This path model suggests that the biological influence of having an antisocial parent on eventual adult antisocial behavior may operate via an ODD-like component in adolescence. On the other hand, much of the effect of adverse environment may be mediated via the conduct component. There

Table 1. Descriptive Statistics by Gender

Variable	Males (N = 88)			Females (N = 87)			Test Statistic*	P
	Mean	Median	SD	Mean	Median	SD		
Age at interview, y	26.0	24	6.64	25.4	24	6.02	0.65	.52
Antisocial biological parent, %	27	. . .	. . .	25	. . .	. . .	0.09	.77
Adverse Adoptive Environment Scale score	1.63	1	1.45	1.43	1	1.47	0.85	.39
No. of conduct disorder symptoms prior to age 15 y	2.42	2	2.34	0.99	0	1.78	4.56	<.001
Conduct disorder rate, %	33	. . .	. . .	13	. . .	. . .	10.24	.001
No. of adolescent aggressivity behaviors	2.75	1	3.51	1.52	0	2.80	2.56	.01
No. of adult antisocial symptoms	3.66	3	2.36	1.49	1	1.85	6.74	<.001
Rate of *DSM-III* adult antisocial criteria fulfilled (≥4 symptoms), %	41	. . .	. . .	11	. . .	. . .	19.54	<.001

**Rates of antisocial biological parent, conduct disorder, and adult antisocial criteria were all tested using the Pearson χ^2 statistic, 1* df. *All other comparisons are based on* t *tests, 173* df.

Table 2. Frequency of Individual Behavioral Symptoms Among Males (N = 88) and Females (N = 87)*

Item	Original Scale	CD vs ODD	Males, %	Females, %	χ^2 Test	P
Expelled from school	C	CD	16	5	6.07	.01
School troubles	C	CD	32	9	13.70	.001
Poor grades	C	CD	23	8	7.23	.007
Sex at an early age	C	CD	18	12	1.55	.22
Early substance abuse	C	CD	18	14	0.63	.43
Stole (conduct questions)	C	CD	43	10	24.01	.001
Stole (aggressivity scale)	A	CD	19	5	8.98	.003
Vandalism (conduct questions)†	C	CD	15	2	8.69	.003
Vandalism (aggressivity scale)†	A	CD	11	2	5.63	.02
Started fights (conduct question)†	C	CD	15	3	6.75	.01
Frequently fought (aggressivity scale)‡	A	CD	6	2	F	.28
Was arrested‡	C	CD	2	1	F	1.00
Was a bully‡	A	CD	5	3	F	.44
Was cruel‡	A	CD	0	1	F	1.00
Set fires‡	A	CD	1	0	F	1.00
Was physically abusive‡	A	CD	0	0	F	1.00
Threatened adults‡	A	CD	5	1	F	.37
Frequent tantrums	A	ODD	16	8	2.56	.11
Defiant	A	ODD	35	18	6.31	.01
Lied frequently (conduct questions)	C	B?	49	25	10.42	.001
Lied frequently (aggressivity scale)	A	B?	32	14	8.06	.005
Quarreled frequently	A	ODD	9	7	0.29	.59
Rebellious	A	ODD	33	21	3.35	.07
Swore frequently	A	B?	17	15	0.14	.70
Teased frequently	A	ODD	10	7	0.62	.43
Wouldn't mind authority	A	ODD	34	16	7.53	.006
Sassy	A	ODD	36	24	3.10	.08
Verbally abusive	A	B?	6	7	0.11	.74

**CD indicates symptom more consistent with conduct disorder; ODD, symptom more consistent with oppositional defiant disorder; B?, symptom consistent with both conduct disorder and oppositional defiant disorder; C, originally part of conduct scale; A, originally part of aggression scale; and F, Fisher exact test. All χ^2 tests are Pearson χ^2 with 1* df.

†Excluded from female analysis due to rarity.

‡Excluded from male and female analysis due to rarity.

was no evidence that an interaction between the 2 components had any additional predictive value for adult antisocial symptoms. In male adoptees, these biological and environmental influences seem to lead to adult antisocial symptoms via separate pathways.

FEMALES

For women, the principal component analysis again supported a 3-component model (**Table 5**). Oppositional defiant disorder behaviors again load most heavily on the first component. However, in contrast to the men, the other items are spread out over all 3 components. Along with numerous ODD behaviors, stealing and early sexual experience are associated with the first component. Substance abuse, school difficulties, and habitual lying load on the second. The third component also contains items related to academic difficulties, stealing, and several items (quarrelling, teasing, and verbal abusiveness) that suggest verbal aggression. We label the first component the "ODD component," the second "conduct component 1," and the third "conduct component 2." Despite the la-

Table 3. Principal Component Analysis of the Correlation Matrix for Males: Correlations Between Behaviors and Rotated Components*

Item	Component 1 (ODD)	Component 2 (Conduct)	Component 3 (Early Experimenting vs Violence)
Expelled from school	32	**52**	–20
School troubles	35	**75**	11
Poor grades	33	12	–9
Sex at an early age	28	27	**53**
Early substance abuse	17	**54**	**42**
Stole (conduct questions)	20	**67**	–3
Stole (aggressivity scale)	**72**	30	–1
Vandalism (conduct questions)	–8	**74**	7
Vandalism (aggressivity scale)	35	–5	31
Started fights	9	**56**	**–50**
Frequent tantrums	**60**	17	–10
Defiant	**76**	16	31
Lied frequently (conduct questions)	**49**	25	–1
Lied frequently (aggressivity scale)	**79**	10	2
Quarreled frequently	**43**	10	**–68**
Rebellious	**82**	8	5
Swore frequently	**66**	12	2
Teased frequently	**45**	5	–33
Wouldn't mind authority	**82**	17	21
Sassy	**68**	11	–13
Verbally abusive	**40**	32	5
Common variance accounted for, %	27	14	8

**All correlations multiplied × 100. Correlations greater than 40 are in boldface.*

Table 4. Regression Model Parameters Associated With the Male Path Analysis (N = 88)*

Predictor Variable	B	95% CI	t_{86}	P
ODD Component, R^2 = 0.13				
Intercept	–0.22	–0.44 to 0.01	. . .	. . .
Biological parent ASPD†	0.81	0.37 to 1.26	3.63	<.001
Conduct Component, R^2 = 0.08				
Intercept	–0.32	–0.63 to 0.02	. . .	. . .
AAES‡	0.20	0.06 to 0.34	2.80	.006
Adult Antisocial Symptoms, R^2 = 0.34				
Intercept	3.25	2.61 to 3.88	. . .	. . .
ODD component§	1.00	0.58 to 1.42	4.77	<.001
Conduct component§	0.75	0.32 to 1.18	3.45	<.001
AAES‖	0.25	–0.05 to 0.55	1.68	.10

**Component variables are measured in SDs from the mean. B indicates estimated regression coefficient; CI, confidence interval; ODD, oppositional defiant disorder; ASPD, antisocial personality disorder; and AAES, Adverse Adoptive Environment Scale.*
†SD for present vs absent.
‡SD per point on 7-point scale.
§Symptoms per SD.
‖Symptoms per point on 7-point scale.

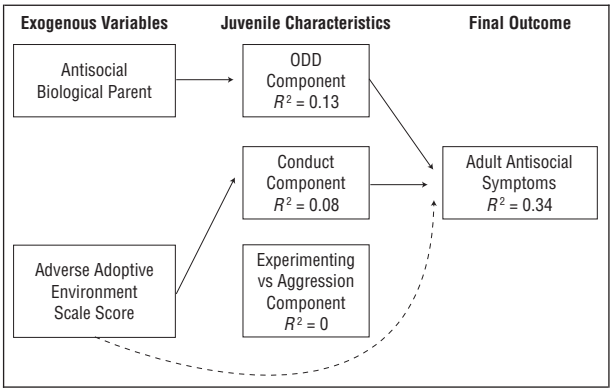

Figure 1. *Male correlation model. Solid line indicates* P<*.05; dashed line, .05*<P<*.15.* R^2 *values for individual regressions are listed under the outcome variable. See Table 4 for model parameters.*

bels, contrasts between these components seems less clear than in the males. The 3 components accounted for 59% of the common variance of these behaviors.

The individual regression models comprising the female path analysis are described in **Table 6** and illustrated in **Figure 2**. In contrast to the males, no simple separation of paths exists. Although having an antisocial biological parent is a modest risk factor for all behavioral components, even in the absence of adverse environment, the interaction of biological and environmental influences forms an additional risk factor for the ODD and conduct 2 components. (These interactions seems to account for the significant interactions in the combined-gender analysis reported in Cadoret et al.[18]) After controlling for the principal components, residual effects of the exogenous variables and their interaction on adult antisocial symptoms were negligible.

COMMENT

Consistent with several other studies, our male analysis supports the division of disruptive behaviors into distinct dimensions that approximate the *DSM* notions of ODD and conduct disorder. The conduct symptoms correlate with more than 1 component in both genders, and this may be taken as evidence for heterogeneity in that concept.

Table 5. Principal Component Analysis of the Correlation Matrix for Females: Correlations Between Behaviors and Rotated Components*

Item	Component 1 (ODD)	Component 2 (Conduct 1)	Component 3 (Conduct 2)
Expelled from school	–15	39	**41**
School troubles	–5	**45**	**60**
Poor grades	7	**82**	8
Sex at an early age	**68**	32	–19
Early substance abuse	33	**64**	–8
Stole (conduct questions)	**42**	**41**	34
Stole (aggressivity scale)	**52**	7	**65**
Frequent tantrums	**70**	35	–5
Defiant	**83**	8	17
Lied frequently (conduct questions)	36	**63**	13
Lied frequently (aggressivity scale)	**62**	**54**	17
Quarreled frequently	24	**59**	36
Rebellious	**70**	7	15
Swore frequently	**73**	14	33
Teased frequently	16	–4	**77**
Wouldn't mind authority	**64**	39	7
Sassy	**72**	4	0
Verbally abusive	**68**	18	**42**
Common variance accounted for, %	29	17	12

**All correlations multiplied ×100. Correlations greater than 40 are in boldface.*

Table 6. Regression Model Parameters Associated With the Female Path Analysis (n = 87)*

Predictor Variable	B	95% CI	t_{83}	P
ODD Component, R^2 = 0.23				
Intercept	–0.16	–0.48 to 0.15	. . .	. . .
Biological parent ASPD†	0.59	0.14 to 1.03	2.62	.01
AAES‡	0.04	–0.11 to 0.19	0.54	.59
ASPD × AAES§	0.57	0.27 to 0.88	3.71	.003
Conduct Component 1, R^2 = 0.05				
Intercept	–0.13	–0.37 to 0.11	. . .	. . .
Biological parent ASPD†	0.51	0.03 to 0.99	2.10	.04
Conduct Component 2, R^2 = 0.13				
Intercept	–0.03	–0.36 to 0.31	. . .	. . .
Biological parent ASPD†	0.55	0.08 to 1.03	2.33	.02
AAES‡	–0.05	–0.21 to 0.11	–0.67	.50
ASPD × AAES§	0.47	0.15 to 0.80	2.88	.004
Adult Antisocial Symptoms, R^2 = 0.45				
Intercept	1.49	1.20 to 1.79	. . .	. . .
ODD component‖	0.89	0.59 to 1.19	5.87	<.001
Conduct component 1‖	0.84	0.54 to 1.14	5.56	<.001
Conduct component 2‖	0.24	–0.06 to 0.54	1.59	.12

**Component variables are measured in SDs from the mean. B indicates estimated regression coefficient; CI, confidence interval; ODD, oppositional defiant disorder; ASPD, antisocial personality disorder; and AAES, Adverse Adoptive Environment Scale.*

†SD for present vs absent.

‡SD per point on 7-point scale.

§Additional SD per point on 7-point scale when antisocial biological parent is present.

‖Symptoms per SD.

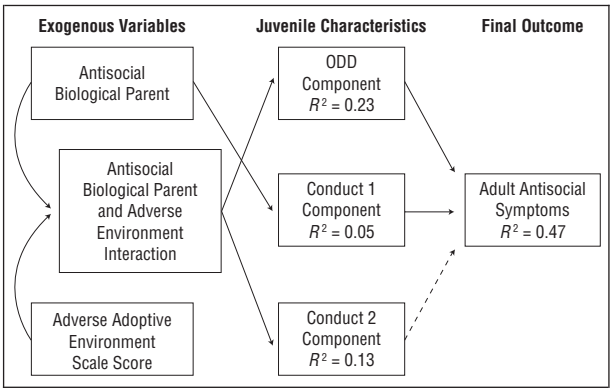

Figure 2. *Female correlation model. Solid line indicates P<.05; dashed line, .05<P<.15. R^2 values for individual regressions are listed under the outcome variable. See Table 6 for model parameters.*

It seems plausible that a genetically transmitted liability to sociopathy might manifest earlier in life as the personality-like symptoms of ODD rather than the behaviorally oriented criteria for conduct disorder. After all, it is difficult to conceive of a gene for stealing cars.[21,45] On the other hand, genetic influences on temperament leading to typical ODD features do not seem so far-fetched.

Our models may account for one of the results in the large study by Lyon et al[21] of male twins from the Vietnam-era veteran registry. They found a relatively strong genetic effect for adult antisocial behavior, but predominantly a shared environmental effect for conduct symptoms. The biological pathway through ODD in our male model could be the missing link to the genetic adult effect.

There is already evidence in the literature suggesting genetic transmission of aggressive tendencies[20,46] but not nonaggressive delinquent behaviors.[36] The fact that our ODD component was extracted primarily from items in Loney's aggressivity scale suggests that perhaps what we have done is replicated these findings and merely relabeled aggression as ODD. However, there was very little indisputably aggressive behavior documented in our subjects. Most of the aggressivity scale items that clearly suggest physical aggression (eg, bullying, physical abusiveness, threatening adults) were too rare to include. We therefore feel comfortable labeling the component as ODD. Because of the rarity of overt aggression, we cannot tell

whether the ODD component should be considered distinct from or on a continuum with other researchers' aggressivity constructs. We note however that a history of starting fights, the only physically aggressive symptom common enough to include in the male analysis, did not load on the ODD component.

STUDIES SUCH as that by Loeber et al,[37] which found that severe childhood conduct disorder is often preceded by ODD, suggest the hypothesis that ODD may only be a risk factor for sociopathy if it is a predecessor to a conduct syndrome. This idea is not supported by our data, because the ODD component was found to be an independent risk factor. Furthermore, examination of the joint distribution of estimated component scores in males (not shown) demonstrated that many adoptees had elevations in the ODD component only. We therefore fit these models over a range of data appropriate for checking this conjecture.

If our male model is correct, one would still expect correlation between conduct disorder and ODD in the general population. This is due to the expected association between adverse environment and antisocial parenthood in nonadoption populations.[47,48] Thus, our findings are not inconsistent with the high comorbidity that is frequently reported between conduct disorder and ODD,[7,36,49] or some of the overlap seen in nonadoption factor analyses.[7]

We studied features of ODD in adolescents. The symptoms were either especially persistent or began at a relatively late age. This may be crucial to the strong link to adult sociopathy. Our data also contained a retrospective measure of preadolescent aggressivity, which presumably also correlates highly with ODD in that age range. This measure was not very predictive of adult antisocial personality. This may be due to the frequency with which, in preadolescence, these behaviors represent a less severe and self-limiting form of disruptive behavior.[34]

The apparent role of biological and environmental background varies by gender, particularly in the prediction of ODD components. This is consistent with previous observations that the predominantly male literature regarding these influences may not be entirely relevant to females.[50,51] The significant interactions in our female model suggest that a confluence of multiple factors may be needed to put them at risk. This is broadly consistent with the conclusions of Sigvardsson et al[12] from the Swedish Adoption Study. We make this hypothesis with some caution however, because the frequency and severity of antisocial symptoms were substantially lower in females.

Finally, we note that we have ignored the potentially confounding influences of a third *DSM* disruptive behavior diagnosis, attention-deficit/hyperactivity disorder (ADHD). The significant comorbidity between ADHD and both conduct disorder and ODD is well known.[4,32,49,52,53] It is possible that some of our proposed biological or environmental associations are illusory and created by associations with ADHD instead. However, studies reported by Lahey et al suggest that both paternal psychopathy[54] and general maternal personality pathology[55] are specifically linked to conduct disorder and not ADHD. Furthermore, results of the family study by Faraone et al,[4] although somewhat ambiguous, are also consistent with the idea that it is conduct disorder and ODD, not ADHD, that serve as risk factors for antisocial behavior.

One should keep several potential limitations of this data in mind. The Adverse Adoptive Environment Scale obviously does not cover all environmental influences that may be relevant to the development of behavior disorders. For example, we have not examined the possible roles of socioeconomic status or peer influences. We therefore make no claims to a comprehensive analysis of possible biological-genetic interactions.

Furthermore, we cannot be sure that environmental influences are similar if comparable events occur in a home where a parent is antisocial.[45] There may be a qualitative aspect to such an interaction that cannot be captured in a statistical model of adoption data.

Our measurement of the influences that we did study is surely imperfect. The identification of antisocial personality in the biological parents via prison and hospital records probably led to a fairly severe threshold for identifying parental antisocial personality. Less severe (or more successful) cases may have gone undetected and been incorrectly classified. The use of *DSM* symptom counts is obviously an imperfect approximation of the "quantity" of sociopathy present in the adult adoptees. Similar limitations apply to the other scales.

The percent variance explained throughout the path models, while often statistically significant, is also relatively small. This is likely to be due in part to a combination of the above factors, ie, omission of other causal factors and imperfect measurement. However, given the difficulties inherent in quantification of these variables, we do not believe that a small percent of variance explained necessarily translates into a lack of practical significance.

All of the behavioral ascertainments were retrospective. Most of the conduct items were rated by a consensus of information collected from the adoptee and the adoptive parents, but the ODD items were rated on the basis of parental recall alone. (The developers of the aggressivity scale assumed that an authority figure such as a parent would be needed to reliably rate these items. Although we lack hard evidence, we still believe this to be the case.) It is possible that the separation of these items in our principal component analyses reflects these differences in informant. An attempt to analyze possible informant bias for the conduct symptoms was inconclusive. A related concern is that the parents of adults with antisocial personality disorder might be more prone to retrospectively recall the extent of early oppositional traits. However, potential respondent and recall bias do not explain why a specific association between the male ODD component and having antisocial biological parents exists.

The danger of other possible biases inherent in an adoption study, such as selective placement, time-to-placement effects, nongenetic biological effects, and induction of an adverse environment by the disruptive behavior of the adoptee, have been discussed previously by Cadoret et al.[18] Although our ability to examine nongenetic biological effects (eg, in utero exposures) was limited, there was no evidence to suggest that any of these

potential effects significantly confound the relationships studied here.

Was there sufficient power to truly test the biological-environmental interaction in males? The sensitivity of our analysis is reflected in the 95% confidence intervals for the relevant interaction terms in our initial, saturated models. These were –0.16 to 0.50 for the ODD component, –0.29 to 0.39 for the conduct component, –0.66 to 0.04 for the early experimenting component, and –0.45 to 0.99 for adult antisocial symptoms (units are additional principal component SDs or adult antisocial symptoms per Adverse Adoptive Environment Scale point in presence of antisocial parent). The results suggest that large interactions are unlikely. We may have missed more moderate effects. Nonetheless, the evidence is much more consistent with no-interaction models.

Several reviewers have suggested that tetrachoric correlation factor analyses[56] would have been preferable to principal components analyses. Such analyses proved impossible with this data because the estimated tetrachoric matrices were not of the proper mathematical form (ie, they were not positive definite). Although not strictly interpretable as a factor analysis, we still believe that the principal components approach is useful. We briefly note some of the differences between these methods.

Although they both describe pairwise relationships between variables, principal components and factor analyses are based on different underlying models. In contrast to factor analysis, principal components analysis does not remove estimated specific variance ("error") terms associated with each variable. Also, a factor analysis is predicated on the assumption that there is no information about the interrelationships between the variables that is not reflected in a description of the pairwise relationships. A principal components analysis requires no such assumption. However, the analysis still cannot detect any 3-way or higher interrelationships that might exist.

We emphasize that this was an exploratory analysis. Our results are dependent on behavior components that were produced by inherently exploratory methods. These models should be considered hypotheses, suggested by one adoption data set, but in need of further testing.

Our analysis supports the concept of ODD in adolescents as a biologically influenced phenomenon that may be distinct from conduct disorder and comparably predictive of adult antisocial behavior. This prognostic importance suggests a change in diagnostic criteria for antisocial personality disorder. At present, *DSM-IV* criteria[57] require a retrospective diagnosis of conduct disorder before age 15 years. Our findings suggest that a diagnosis of either conduct disorder or ODD may be more appropriate.

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Reprints: Douglas R. Langbehn, MD, PhD, Psychiatry Service, 9W-20 VA Hospital, Highway 6, Iowa City, IA 52246.

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