

Parent-Child Conflict and the Comorbidity Among Childhood Externalizing Disorders

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Background: Previous research has suggested that substantial comorbidity exists among childhood externalizing disorders, specifically attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD). Moreover, parent-child conflict predicts each of these disorders. Our goals were to determine whether parent-child conflict was associated with the comorbidity among ADHD, CD, and ODD, and to explicitly examine the etiology of this association via a genetically informative design.

Methods: We compared the fit of the following 2 biometric models: the 2-factor common-pathway model, which examined genetic and environmental contributions to the relationship between conflict and the covariation among the 3 disorders, and the Cholesky model, which examined the relationship between conflict and each disorder individually. The sample consisted of 808 same-sex 11-year-old twin pairs from the Minnesota Twin Family Study, a population-based sample of Minnesota twins and their families. Main outcome measures in-

cluded symptom counts for ADHD, CD, and ODD, obtained from structured interviews administered to twins and their mothers. Parent-child conflict was assessed via mother and twin reports of the Parental Environment Questionnaire.

Results: The 2-factor model provided a better fit to the data. These results indicated that conflict accounted for 33% of the covariation among the disorders, via genetic and environmental factors.

Conclusions: Parent-child conflict appears to act as a common vulnerability that increases risk for multiple childhood disorders. Furthermore, this association is mediated via common genetic and environmental factors. These findings support the idea that the comorbidity among these disorders partially reflects core psychopathological processes in the family environment that link putatively separate psychiatric disorders.

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R ESEARCH HAS confirmed high levels of comorbidity among attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD).¹⁻³ Of individuals with 1 of these diagnoses, 29% to 71% had at least 1 other diagnosis in epidemiological and clinical samples.⁴⁻⁶ Although no firm consensus exists regarding the meaning of this comorbidity, research^{3,4,7} has generally supported 2 interrelated conceptualizations: First, there exists a gradient upon which those with multiple disorders have more serious clinical courses with poorer outcomes and higher levels of the relevant genetic and/or environmental influences than individuals with single disorders. Second, these influences likely take the form of common vulnerabilities. However, the nature (ie, genetic or environmental) of these vulnerabilities remains unresolved.

In an effort to understand comorbidity among childhood externalizing disorders, the etiology of these common vulnerabilities was examined in a previous study via the classic twin method.⁸ Using data from the 11-year-old cohort of the Minnesota Twin Family Study (MTFS), we fit a biometric model to the data. The results showed that although genetic factors contributed substantially to each disorder individually, they accounted for only 25% of the covariance among ADHD, ODD, and CD, whereas 50% resulted from a single shared environmental factor. Given the importance of this shared environmental factor, our goal in the present study was to begin to explicitly identify this factor. We focused on parent-child conflict as a recent study,⁹ and subsequent reanalysis¹⁰ found that social interactions between parents and their children were strongly influenced by the shared environment.

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Studies of putatively “environmental” influences on externalizing disorders are typically conducted on non-twin family members who share genes and a familial environment (ie, the traditional family design).¹¹⁻¹³ Although such studies provide important information, they do not allow for explicit estimation of the distinct contributions of environmental factors, as genetic and environmental effects are confounded. The use of genetically informative samples, such as twins, allows for more definitive identification of environmental factors. As such, studies that examine psychosocial indicators within genetically informative designs advance our understanding of the environmental factors important for a given disorder.

Nevertheless, few studies have examined the relationship between a psychosocial measure and a specific disorder using genetically informative designs.¹⁴⁻²⁰ Furthermore, to our knowledge, no previous study has examined the etiology of the relationship between a psychosocial variable and the *comorbidity* among the externalizing disorders. Previous studies have examined the relationship between parenting and the occurrence of juvenile antisocial behavior,^{15,17-20} but have not examined how parenting is related to the co-occurrence of ADHD and CD. Thus, the present study is advantageous in 2 ways. First, we examined a psychosocial variable within a genetically informative design. Second, we focused specifically on the comorbidity of ADHD, CD, and ODD, thereby expanding our understanding of the impact of explicit environmental factors on childhood externalizing. Specifically, we entered parent-child conflict, ADHD, CD, and ODD into a multivariate biometric model to determine whether conflict accounted for part of the shared environmental factor common to ADHD, CD, and ODD.

METHODS

PARTICIPANTS

Participants were male and female twin pairs who participated in the MTFs, a population-based, longitudinal study of adolescent twins born in the state of Minnesota, and their parents. Twin families were ascertained from birth records and located using public databases. More than 90% of twin births from 1971 through 1985 have been located. Families were excluded from the study if either twin had a cognitive or physical handicap that would preclude completing our daylong, in-person assessment, or if the family lived more than 1 day's drive from our Minneapolis laboratory. Of the eligible families, 83% agreed to participate. Parents in participating families had slightly, albeit significantly, more years of education (0.25 years) than parents in nonparticipating families. In socioeconomic status and self-reported mental health problems, however, no significant differences existed between participating and nonparticipating families.²¹ Participating families were generally representative of the Minnesota population at the time of the twins' birth; approximately 98% were Caucasian. Children gave informed assent, whereas parents gave informed consent for themselves and their children. Research protocol was approved by the University of Minnesota Institutional Review Board. Further information regarding all aspects of MTFs recruitment is detailed elsewhere.²¹

The participants ranged in age from 10 to 12 years (average age, 11 years), during their intake visit. The original MTFs

11-year-old cohort consisted of 753 same-sex, reared-together monozygotic (MZ) and dizygotic (DZ) twin pairs: 373 male ($n_{MZ}=253$; $n_{DZ}=120$) and 380 female pairs ($n_{MZ}=233$; $n_{DZ}=147$). In an effort to increase our sample size, we have begun augmenting our sample with twins born from 1989 through 1991. To date, we have added 55 additional pairs, bringing the sample to 384 male ($n_{MZ}=259$; $n_{DZ}=125$) and 424 female twin pairs ($n_{MZ}=263$; $n_{DZ}=161$). This yields a total sample of 1616 participants.

Monozygotic twins are slightly more common than dizygotic twins in the population from which our sample was drawn.²² From 1971 to 1984, there were 4.09 MZ and 2.60 like-sex DZ twin pairs per 1000 births for an MZ/DZ ratio of 1.57:1. The preponderance of MZ twin pairs reflects this, as well as a slight bias in recruitment, with an MZ/DZ ratio of 1.82:1.

ZYGOSITY DETERMINATION

Zygosity was determined by the agreement of several separate estimates. First, parents completed a standard zygosity questionnaire. Second, MTFs staff evaluated visage, hair color, and face and ear shape for physical similarity. Third, ponderal and cephalic indexes and fingerprint ridge counts were measured. A serological analysis was performed when the 3 estimates did not agree. This method was found to be highly accurate.⁸

ASSESSMENT OF MENTAL DISORDERS

During their intake visit, participants and their parents underwent in-person assessment by trained bachelor's- and master's-level interviewers for lifetime *DSM-III-R* mental disorders (the *DSM-III-R* was current at the onset of the study). Lifetime ADHD, CD, and ODD were assessed using the Diagnostic Interview for Children and Adolescents-Revised.²³ The MTFs version of this instrument contained supplementary probes and questions, added after consultation with one of its authors, to ensure complete coverage of each symptom. Mothers reported on symptom presence in both twins, whereas twins reported on symptom presence in themselves only.

Before the assignment of mental disorder symptoms, a clinical case conference was held in which the evidence of every symptom was discussed by at least 2 advanced clinical psychology doctoral students (S.A.B.). As necessary, audiotapes from the interview were replayed or the participant was recontacted for clarification. Only symptoms that were judged to be clinically significant in both severity and frequency were considered present. The reliability of the consensus process was good, with $\kappa=0.77$, $\kappa=0.79$, and $\kappa=0.67$ for diagnoses of ADHD, CD, and ODD, respectively.

After symptom assignment, computer algorithms were used to create symptom counts corresponding to the criteria for *DSM-III-R* disorders. These include the following: (1) the 9 criterion-A symptoms of ODD, (2) 12 of the 13 criterion-A symptoms of CD (symptom 9, “has forced someone into sexual activity,” was not assessed to avoid potential mandated reporting), and (3) the 14 criterion-A symptoms of ADHD listed in the *DSM-III-R*. Symptom duration rules were omitted. We used a best-estimate approach to compute symptom counts, in which a symptom was considered present if it was endorsed by the mother or the child. Symptoms endorsed by both were counted as a single symptom. Previous studies have indicated that each type of informant contributes a considerable amount of valid information not contributed by other informants,^{8,24} allowing for a more complete assessment of symptomatology. Symptom counts, rather than diagnoses, were used primarily to increase statistical power, as diagnostic prevalence rates in community-based samples such as the MTFs are lower than in clinically referred samples. Also, previous studies have found

that patterns of genetic and environmental influence are similar for categorical and dimensional models of psychopathology.²⁵⁻³⁰

ASSESSMENT OF THE FAMILY ENVIRONMENT

The Parental Environment Questionnaire (PEQ) was administered to tap perceptions of mother-twin and father-twin relationships. The mother rated her relationship with each twin, whereas the twins independently rated their relationships with their mother. Each informant rated 50 items assessing aspects of their relationships on a 4-point scale (1 being definitely true). Items were essentially the same for mothers and twins, with alterations in wording for particular raters. This scale, which was developed by the MTF, has been factor analyzed and shown to reliably assess the following 5 dimensions of parent-child relationships: parent-child conflict, parental involvement with child, child regard for the parent, parent regard for the child, and structure provided by the parent.^{31,32} The internal consistencies for the 12-item Parent-Child Conflict Scale (**Table 1**) ranged from 0.81 to 0.88 for twin and parent informants. If 1 item was missing, that item was prorated (ie, the average of the other 11 items was used as the missing item's score) and added to the scale score. If 2 or more items were missing, the scale was considered missing. More information on PEQ development is detailed elsewhere.³¹

The PEQs were mailed to families before their intake assessment. Participants were asked to bring their completed PEQ to their in-person visit. If a completed PEQ was not obtained by the end of the intake assessment, participants were asked to complete it at home and return it by mail. One telephone prompt was made if a PEQ was still not received. Of the 848 female participants, we received twin PEQ reports for 787 and mother PEQ reports for 727. Of the 768 male participants, we received twin PEQ reports for 670 and mother PEQ reports for 640. Mother and twin informant reports of conflict were all moderately and significantly correlated with each other, ranging from 0.21 to 0.30 ($P < .001$).

In creating the conflict variable, we averaged all informant reports. However, to ensure that twin reports were not weighted more heavily than mother reports, we first averaged the twin report of mother and that of the father. We allowed 1 of these reports to be missing. We then averaged the twin "composite" and the mother report of twin. To maximize the number of participants with conflict data, we allowed for missing twin or mother data. As a result, 109 (81 male and 28 female) of the 1616 participants were missing conflict data.

Our choice to average mother and twin reports is bolstered by the results of partial correlations between the disorders and each informant's report of conflict, controlling for the effects of the other informant. These reflect the extent to which the mother and twin provided predictive information that was not provided by the other informant. Partial correlations between ADHD, CD, and ODD and the mother report (controlling for the twin report) of conflict were 0.16, 0.24, and 0.32, respectively ($P < .001$). Partial correlations between ADHD, CD, and ODD and the twin "composite" (controlling for the mother report) of conflict were 0.20, 0.18, and 0.23, respectively ($P < .001$). These results suggest that the mother and twin are providing unique and predictive information and support the inclusion of both informants.

Those subjects with conflict data (returners) were compared with those subjects without conflict data (nonreturners) on the 3 symptom counts, using the Mann-Whitney test (a nonparametric test). Male nonreturners did not differ from the returners on any symptom count. Female nonreturners ($n=28$) exhibited more symptoms of CD than did female re-

Table 1. Items Constituting the Parental Environment Questionnaire Parent-Child Conflict Scale*

1. My parent often criticizes me.
2. Before I finish saying something, my parent often interrupts me.
3. My parent often irritates me.
4. Often there are misunderstandings between my parent and myself.
5. I treat others with more respect than I treat my parent.
6. My parent often hurts my feelings.
7. My parent does not trust me to make my own decisions.
8. My parent and I often get into arguments.
9. I often seem to anger or annoy my parent.
10. My parent often loses her or his temper with me.
11. My parent sometimes hits me in anger.
12. Once in a while I have been really scared of my parent.

*These items constituted the twin version of the questionnaire. For the parent version, items were essentially the same, with alterations in wording appropriate for parental informants (eg, "I sometimes hit my child in anger"). Parents and twins rated these items on a 4-point scale (1 indicated definitely true; 4, definitely false).

turners (Mann-Whitney $z = -2.33$; $P = .02$, 2-tailed), but did not differ on ODD or ADHD symptom counts. However, when the significance level was corrected for multiple tests via the Bonferroni method ($\alpha < .008$), the CD symptom count difference for female participants was no longer significant.

To determine whether conflict was directly associated with the disorders, we also examined several other psychosocial variables. We examined the remaining PEQ scales: parental involvement with the child, child regard for the parent, parent regard for the child, and structure provided by the parent. We averaged mother and twin reports. Next, we examined the Family Adaptability and Cohesion Scale—Third Edition,³³ which consisted of 20 true/false items and assesses 2 aspects of overall family functioning: cohesion and adaptability. Mother and twin reports were averaged. We examined marital discord, as assessed via 2 items on the Spanier Dyadic Adjustment Scale³⁴ (ie, "How often do you and your spouse quarrel?" and "How often do you and your spouse get on each other's nerves?"). These items were rated on a scale of 1 to 6, with 6 corresponding to "all the time." Reports of the mother and father were averaged for each item, after which the items were averaged. Divorce of the twins' biological parents, as reported by the mother via a single item, was examined (1 indicated married and 2, divorced). Finally, we examined parental income, as reported by the mother and father. Parents rated their income on a scale of 1 to 13, with 13 corresponding to an annual income of less than \$10,000 and 1 corresponding to an annual income of greater than \$85,000. Mother and father reports were averaged.

STATISTICAL ANALYSES

As is typical in population-based samples, the symptom count distributions were positively skewed for each disorder. To better approximate normality, the symptom count variables were first ranked, which replaces raw symptom counts with their rank values. Ties were assigned the mean rank of the tied values. The ranked symptom counts were then Blom transformed and standardized to normalize each scale's distribution and give each scale a mean of 0 and an SD of 1. The adjustments were conducted separately by sex, but without regard to zygosity. This procedure was found to optimize model selection.³⁵

The structural equation modeling in the present study uses twin study methods. Twin studies make use of the difference in the proportion of genes shared between MZ twins, who share 100% of their genetic material, and DZ twins, who share an average of 50% of their segregating genetic material. The MZ and

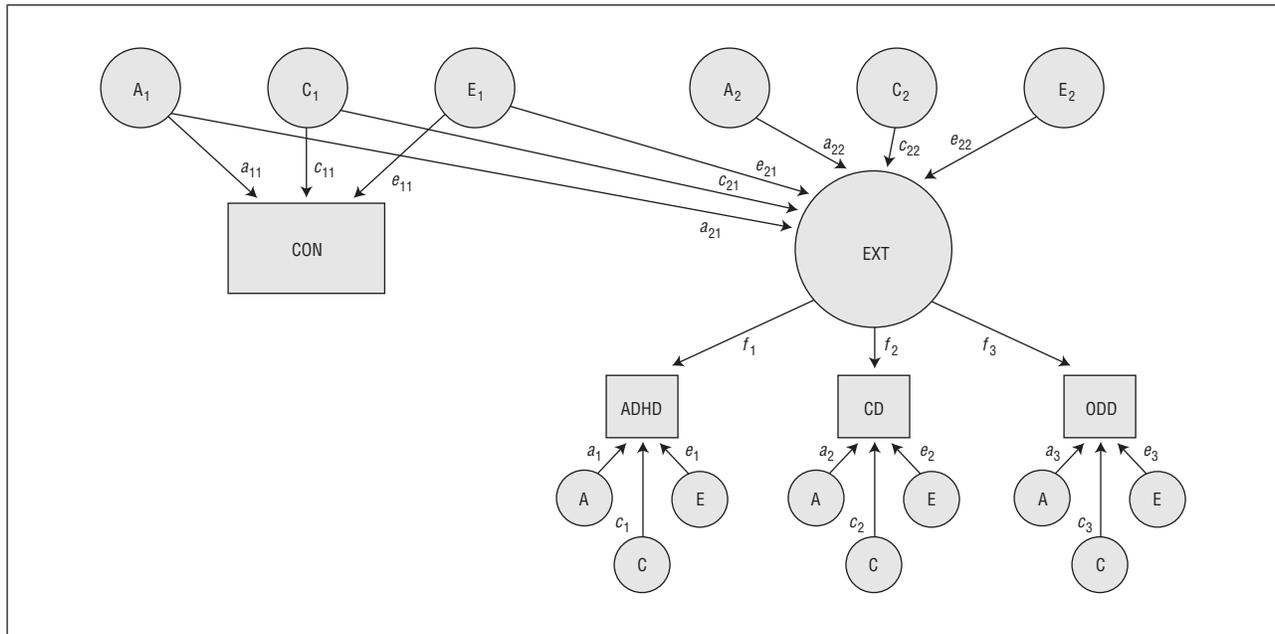


Figure 1. Path diagram of a 2-factor common pathway model for parent-child conflict (CON) and the latent externalizing factor (EXT) underlying attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), and oppositional defiant disorder (ODD). The genetic, shared, and nonshared environmental effects that are common to CON and EXT are represented by A_1 , C_1 , and E_1 , respectively. The genetic, shared, and nonshared environmental effects that are unique to EXT are represented by A_2 , C_2 , and E_2 , respectively. Paths are squared to estimate the proportion of variance. Paths to the factors are represented by lowercase letters followed by 2 numerals (eg, a_{11} , a_{21} , and a_{22}). The disorder-specific paths (ie, those effects unique to each disorder) are represented by lowercase letters followed by 1 numeral (eg, a_1 , a_2 , and a_3).

DZ twin correlations are compared to estimate the relative contributions of additive genetic (a^2), shared environmental (ie, factors that make family members similar to each other; c^2), and nonshared environmental effects plus measurement error (ie, factors that make family members different from each other; e^2) to variance within and covariance among observed behaviors or characteristics (phenotypes). More information on twin studies is provided elsewhere.³⁶

We used Mx, a structural-equation modeling program,³⁷ to perform the model-fitting analyses. Because of the missing PEQ data, we used full-information maximum-likelihood raw data techniques, which correct for statistical biases due to missing data. Specifically, when data are missing, the full-information maximum-likelihood raw data techniques impute a value and then adjust for the imprecision of the imputed value. When fitting models to raw data, their variances, covariances, and means are first freely estimated to get a baseline index of fit (minus twice the natural log-likelihood; $-2\ln L$). The $-2\ln L$ under this unrestricted baseline model is then compared with $-2\ln L$ under more restrictive biometric models. This yields a likelihood-ratio χ^2 test of goodness of fit, which is converted to the Bayesian information criterion (BIC; $BIC = \chi^2 - \Delta df / \ln N$, in which N equals the number of twin pairs).³⁷ The BIC, which measures model fit relative to parsimony, is used to determine the best-fitting model. Better-fitting models have more negative values. A difference in BIC of 10 corresponds to the odds being 150:1 that the model with the more negative value is the better-fitting model and is considered very strong evidence in favor of the model with the more negative BIC value.³⁸

Fitting a 2-factor common pathway model (**Figure 1**) with Mx allowed us to examine genetic and environmental contributions to the association between conflict and the overlap among the childhood externalizing disorders. In this 2-factor model, the covariation among the disorders is conceptualized as a latent externalizing factor (EXT) that loads on all 3 disorders. We then assessed the relationship between conflict and

EXT, parsing the variance within and the covariance between conflict and EXT into genetic, shared, and nonshared environmental effects. In this way, we were able to examine the sources of covariation between conflict and the *comorbidity* among ADHD, CD, and ODD. In addition, we were able to examine the genetic and environmental factors *unique* to each disorder.

However, to determine whether the etiology of the relationship between conflict and the disorders was instead disorder specific, we also tested a Cholesky model (**Figure 2**). As this model allows conflict to load on each disorder individually, conflict could conceivably have a primarily genetically mediated relationship with, for example, CD, but a largely environmentally mediated relationship with ADHD. Of note, the 2-factor model is nested within the Cholesky model, allowing a direct comparison of model fit statistics.

RESULTS

DESCRIPTIVE STATISTICS

Families of male twins had significantly higher mean levels of several psychosocial indicators than did the families of female twins (**Table 2**). The families of male twins reported significantly more conflict and significantly less involvement, regard, and parental income than the families of female twins. Mean levels of structure, cohesion, adaptability, divorce, and marital discord did not vary across sex. Boys also had significantly more symptoms of each disorder than did girls.⁸ Lifetime prevalences of ADHD, CD, and ODD for boys in this sample were 6.7%, 7.2%, and 8.9%, respectively. For girls, lifetime prevalences were 3.5%, 1.1%, and 4.5%, respectively. These rates are upper estimates of the possible disorder prevalences in our sample, because duration requirements were omitted and the best-estimate procedure for symptom as-

signment was used. Correlations among maternal, child, and best-estimate symptom count variables are presented elsewhere.⁸

CORRELATIONS

Phenotypic correlations between the psychosocial variables and the disorders are presented for male and female twins in **Table 3**. For male and female twins, the results indicate that all but 2 of the psychosocial variables, structure and adaptability, were correlated with at least 1 of the disorders. Structure and adaptability were dropped from all subsequent analyses. Conflict was most highly correlated with all 3 of the disorders for male and female twins.

We computed partial correlations between the disorders and parent-child conflict, controlling for all other measured psychosocial variables, to evaluate whether conflict was directly related to the disorders. For female participants, the partial correlations were 0.21, 0.18, and 0.32 for ADHD, CD, and ODD, respectively ($P < .01$). For male participants, the partial correlations were 0.22, 0.28, and 0.33, respectively ($P < .01$). These partial correlations are comparable to the phenotypic correlations between conflict and the disorders, indicating the association between conflict and the disorders was generally not mediated via other psychosocial variables. These results suggest that conflict is directly related to ADHD, CD, and ODD and collectively bolster our choice of conflict as the variable of interest.

MULTIVARIATE MODELING

We initially estimated variances, covariances, and means for the raw data to get a baseline index of fit (**Table 4**), which is necessary to compute and compare fit indices. We tested a 2-factor common pathway model, both allowing for sex differences in parameter estimates and constraining parameter estimates to be equal across sex. We also tested a Cholesky model, which allowed conflict to load on each disorder individually. Again, we fit a sex-differences and a no-sex-differences model. The best-fitting model (ie, that which resulted in the most negative BIC value) was the 2-factor, no-sex-differences model. The improved fit of the no-sex-differences model indicates that parameter estimates do not vary across sex, and apply to both male and female participants. Furthermore, the improved fit of the 2-factor model compared with the Cholesky model indicates that the relationship between conflict and the externalizing disorders is best conceptualized via a common latent externalizing factor rather than independently for each disorder. These results suggest that conflict may act as a common vulnerability that underlies and unites the externalizing behaviors, uniformly increasing risk.

The full 2-factor no-sex-differences model is illustrated in **Figure 3**. The loadings of EXT onto each disorder range from 0.47 to 0.70, suggesting that the latent factor accounts for a moderate proportion (22%-49%) of the variance within each disorder. Thus, these results indicate that at least a moderate amount of the variance in each disorder springs from the same source.

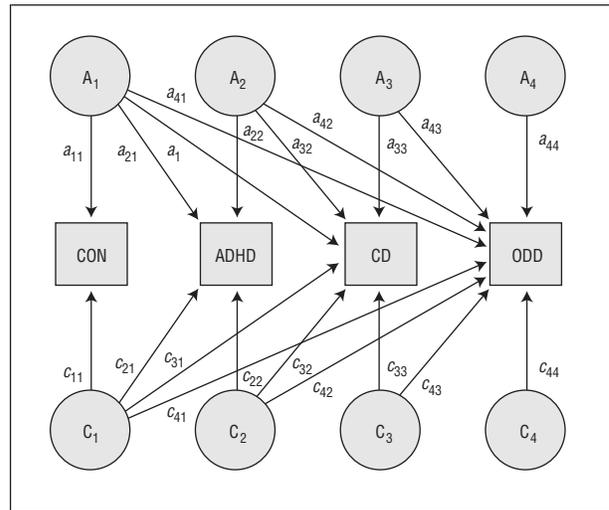


Figure 2. Path diagram of a Cholesky model for parent-child conflict (CON), attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), and oppositional defiant disorder (ODD). The variance in CON and each disorder is parsed into that which is due to additive genetic effects (A₁, A₂, A₃, and A₄), shared environmental effects (C₁, C₂, C₃, and C₄), and nonshared environmental effects (e) are not represented here for ease of presentation. If shown, however, they would follow the same pattern as the genetic and shared environmental effects. Paths, which are squared to estimate the proportion of variance accounted for, are represented by lowercase letters followed by two numerals (eg, a₁₁, a₂₁, a₃₁, and a₄₁).

Variance estimates for conflict can be obtained by squaring its path coefficients. These results indicated that conflict was influenced similarly by all 3 parameters ($a^2=0.38$; $c^2=0.35$; $e^2=0.27$; $P < .05$). The respective contributions of a^2 , c^2 , and e^2 to the co-occurrence among ADHD, CD, and ODD (ie, EXT) can be calculated by squaring and then summing the paths that load on EXT. For example, the proportion of variance in EXT that is due to the shared environment is calculated by individually squaring c_{21} and c_{22} , and then summing the resulting numbers. Thus, shared environmental factors account for the majority (ie, 51%) of the disorder overlap, while genetic and nonshared environmental factors account for only 31% and 18%, respectively ($P < .05$). Moreover, as all of the shared environmental disorder-specific paths (ie, c_1 , c_2 , and c_3) are estimated to be 0, these results further indicate that all shared environmental effects are held in common by the disorders.⁸ In contrast, the genetic and nonshared environmental disorder-specific paths (ie, a_1 , a_2 , a_3 , e_1 , e_2 , and e_3) are all significant. These results collectively indicate that although some of the genetic and nonshared environmental factors are held in common by the disorders, many of the effects are unique to each disorder.⁸ That these results held with the addition of 55 twin pairs further increases our confidence in the findings reported in the introduction.⁸

An examination of the genetic and environmental covariance paths (ie, a_{21} , c_{21} , and e_{21}) reveals the origins of the association between EXT and conflict. These results suggest that, of the shared environmental variance (51%) in EXT, the shared environmental factors contributing to conflict account for roughly 23%. This corresponds to approximately 12% of the total variance in EXT. The remaining shared environmental effects on the

Table 2. Descriptive Statistics for Psychosocial Measures

Variable Scales	Participants							
	Male				Female			
	No. of Participants	Mean Score (SD)	Minimum Score	Maximum Score	No. of Participants	Mean Score (SD)	Minimum Score	Maximum Score
Parent-child conflict	687	21.44 (4.88)*	12.00	40.25	820	18.93 (4.45)*	12.00	34.75
Low parent involvement†	686	7.50 (3.80)*	1.00	21.00	821	6.35 (3.62)*	1.00	21.00
Low twin regard for parent†	689	4.93 (2.33)*	1.00	21.50	822	3.81 (2.17)*	1.00	19.00
Low parent regard for twin†	689	2.00 (1.45)*	1.00	11.50	823	1.62 (1.06)*	1.00	8.00
Low structure†	689	2.93 (1.34)	1.00	8.00	823	2.96 (1.43)	1.00	10.50
Low cohesion†	642	20.99 (4.34)	11.50	39.00	684	20.88 (4.08)	11.00	37.00
Low adaptability†	651	34.03 (3.93)	22.78	48.89	700	33.82 (4.03)	21.67	44.44
Marital discord	730	2.77 (0.62)	1.50	5.50	772	2.77 (0.71)	1.00	6.00
Low parental income†	730	5.87 (3.09)*	1.00	13.00	753	5.45 (3.07)*	1.00	13.00
Divorce	734	1.17 (0.37)	1.00	2.00	774	1.15 (0.35)	1.00	2.00

* $P < .01$, mean differences across sex by independent samples t test.

†Indicates that the item has been reverse scored, such that a high score indicates more dysfunction. Reverse scoring changes only the direction of correlations (ie, from negative to positive), and is performed strictly to ease data presentation. It does not change any other properties of the scale.

Table 3. Phenotypic Correlations Between Psychosocial Measures and Disorder Symptom Counts and Parent-Child Conflict

Variable	Participants, Correlation (No. of Participants)							
	Male				Female			
	ADHD	CD	ODD	Conflict	ADHD	CD	ODD	Conflict
Parent-child conflict	0.28 (687)*	0.33 (687)*	0.37 (687)*	NA	0.25 (820)*	0.24 (820)*	0.37 (820)*	NA
Low parental involvement	0.14 (686)*	0.21 (686)*	0.23 (686)*	0.59 (684)*	0.14 (821)*	0.10 (821)*	0.21 (821)*	0.53 (819)*
Low twin regard for parent	0.12 (689)*	0.17 (689)*	0.18 (689)*	0.56 (687)*	0.06 (822)	0.10 (822)*	0.17 (822)*	0.48 (819)*
Low parent regard for twin	0.16 (689)*	0.19 (689)*	0.18 (689)*	0.53 (687)*	0.12 (823)*	0.10 (823)*	0.12 (823)*	0.42 (820)*
Low structure	0.00 (689)	0.06 (689)	0.04 (689)	0.25 (687)*	0.01 (823)	0.04 (823)	0.02 (823)	0.12 (820)*
Low cohesion	0.18 (642)*	0.23 (642)*	0.21 (642)*	0.32 (616)*	0.05 (684)	0.08 (684)†	0.11 (684)*	0.30 (678)*
Low adaptability	-0.02 (651)	-0.01 (651)	0.07 (651)	-0.03 (625)	-0.06 (700)	0.03 (700)	-0.02 (700)	0.12 (700)*
Marital discord	-0.04 (730)	0.09 (730)†	0.10 (730)†	0.13 (653)*	0.05 (772)	0.08 (772)†	0.16 (772)*	0.14 (764)*
Low parental income	0.03 (730)	0.05 (730)	0.11 (730)*	0.07 (657)	-0.02 (753)	0.07 (753)	0.12 (753)*	0.11 (742)*
Divorce	-0.04 (734)	0.09 (734)†	0.09 (734)†	0.03 (653)	0.05 (774)	0.06 (774)	0.13 (774)*	0.01 (766)

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; CD, conduct disorder; NA, not applicable; ODD, oppositional defiant disorder.

* $P < .01$.

† $P < .05$.

Table 4. Test Statistics for Models*

Model	-2lnL	df	χ^2 on df	BIC
Baseline	20 200.43	6149	NA	NA
Cholesky, sex-differences	20 327.71	6249	127.28 on 100	-541.72
Cholesky, no-sex-differences	20 361.78	6279	161.35 on 130	-708.35
2-Factor, sex-differences	20 346.71	6269	146.28 on 120	-656.52
2-Factor, No-Sex-Differences	20 373.13	6289	172.70 on 140	-763.90

Abbreviations: BIC, Bayesian information criterion; NA, not applicable; -2lnL, minus twice the natural log-likelihood.

*The baseline model is used to compute and compare fit indices. The best-fitting model is determined by the most negative BIC value. Using this criterion, the 2-factor common pathway no-sex-differences model fit best (indicated by boldface type).

externalizing factor (ie, c_{22}) do not overlap with those of conflict. In contrast, the genetic factors contributing to conflict account for all of the significant genetic vari-

ance in EXT, as the remaining genetic effects (ie, a_{22}) are not statistically significant. Finally, the nonshared environmental factors contributing to conflict account for roughly 5% of the nonshared environmental variance in EXT, which is about 1% of its total variance. In total then, the genetic and environmental factors contributing to conflict account for roughly 33% of the total variance in EXT.

COMMENT

The results presented herein support 3 interrelated conclusions. First, parent-child conflict appears to act as a common vulnerability that increases risk for multiple childhood disorders. Second, the genes that are common to the disorders may be many of the same genes that influence conflict. Third, the shared environmental factor that influences conflict accounts for roughly a quarter of the shared environmental effect common to ADHD, CD, and ODD.⁸ This last conclusion is consistent with those of a previous study of parental negativity and antisocial behavior, which found that genetic factors largely

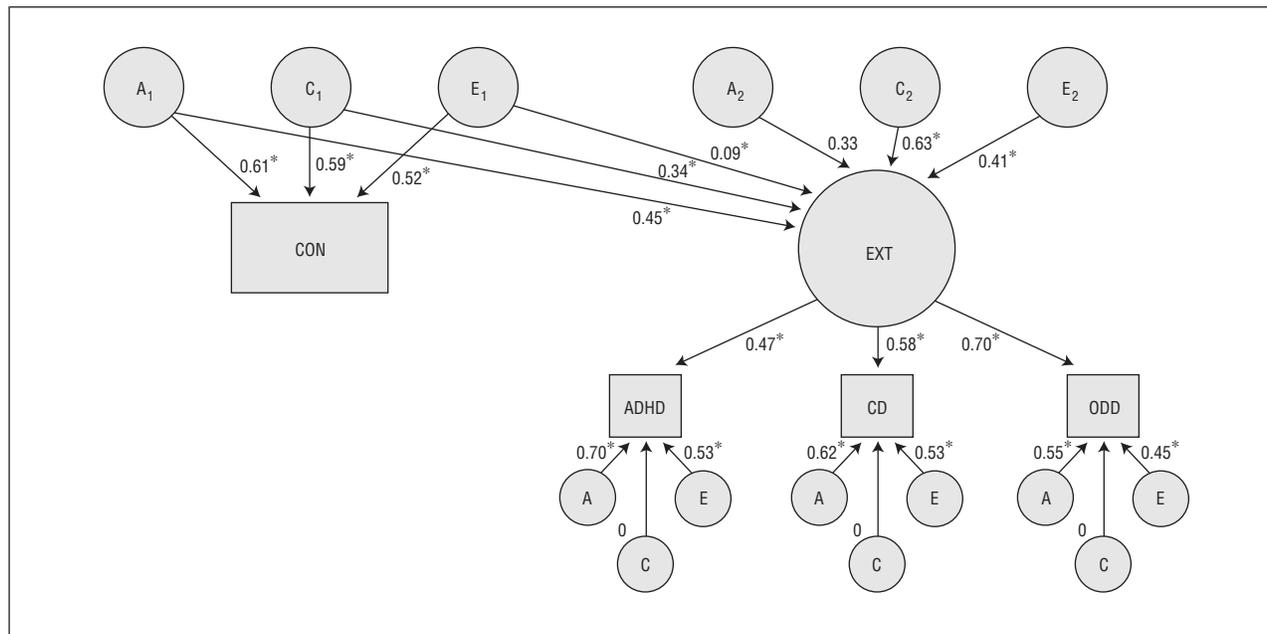


Figure 3. Standardized path estimates from the 2-factor common pathway model for parent-child conflict (CON) and the latent externalizing factor (EXT) underlying attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), and oppositional defiant disorder (ODD). Standardized path estimates of the genetic, shared, and nonshared environmental contributions to the variance within each factor, the covariance between the factors, and variance unique to each disorder are illustrated. Asterisk indicates significant paths ($P < .05$). Other abbreviations are explained in the legend to Figure 1.

accounted for their covariation, although shared environmental factors still made a significant contribution (20%-26%).¹⁹

Several limitations must be kept in mind when interpreting the results of this study. First, we are unable to determine the direction of causation between conflict and the disorders. It may be that conflict exacerbates the disorders, causing the child to manifest even more severe externalizing symptomatology than he or she otherwise would, or alternately, that the oppositional component of externalizing psychopathology induces parent-child conflict. Finally, it may be that both of these forces work in unison, such that conflict both influences and is influenced by ADHD, CD, and ODD.¹⁵ Regardless, the model used herein cannot determine direction of causation. Future research should address this concern, perhaps using a longitudinal twin design. A second limitation concerns the statistically nonsignificant but non-zero contribution made by the second genetic factor (a_{22}) to the latent externalizing factor. It may be that we did not have the power to detect these effects as significant. Should that be the case, future studies may find that other psychosocial variables account for some of the common genetic influence on ADHD, CD, and ODD. A third limitation concerns general limitations of the additive twin model. Specifically, these models do not reveal the contributions of gene-environment correlations or gene \times environment interactions. Instead, these effects are indirectly included in the reported genetic and environmental parameter estimates. Also, although this study relies on the equal-environments assumption for interpretation, that assumption was not directly evaluated herein. Such analyses are beyond the scope of the present study. However, the equal-environments assumption appears tenable for many mental disorders.³⁹⁻⁴² A fourth limita-

tion concerns the use of family-reported, as opposed to observer-rated, conflict. Observer ratings are potentially advantageous in that they remove the dispositional characteristics of the reporter(s). However, observer ratings are generally based on only 1 to 2 hours of observation, whereas family reporters have direct and long-term knowledge of their within-family relationships. Regardless, it is unclear what effect observer reports may have on the estimates reported herein. Finally, the value of conflict as a psychosocial indicator may vary with characteristics of the sample. For example, these results apply only to children aged 10 to 12 years and not to younger or older populations. It may be that other psychosocial variables, such as parental monitoring,⁴³ become more salient as children transition through adolescence. Also, estimates of genetic influence often increase with age,^{44,45} a phenomenon that may arise because individuals have an increasingly greater impact on their environments as they age.⁴⁶ As such, the results from the present study should not be applied to externalizing behaviors at other ages.⁴⁷ It also remains unclear how these results might vary by ethnicity.

Despite these limitations, the results of the present study have some important implications. The first relates to our understanding of the conceptual basis behind psychiatric comorbidity. The comorbidity of psychiatric disorders, once thought to be the exception, now seems to be the rule,^{48,49} although the mechanisms underlying it have remained unclear. The source of comorbidity among mental disorders has been ascribed to measurement error, to overlapping symptoms within the DSM system, and as a window into broad latent factors linking mental disorders.⁴⁹⁻⁵¹ The data presented herein support the last hypothesis. Parent-child conflict appears to act as a common vulnerability that increases risk for mul-

tiple childhood disorders, supporting the notion that core psychopathological processes link putatively separate psychiatric disorders.

Second, the findings presented herein begin to bridge the ideological chasm between those who focus on genetic influences on childhood psychiatric disorders and those who point to etiologically relevant environmental influences. Traditional family designs are frequently used to examine psychosocial influences on childhood behavioral disorders. Positive results are often reported as evidence of significant environmental influence. However, as this design does not allow for the examination of environmental influences independent of genetic influences, such conclusions may be premature. In contrast, twin studies do allow for such disambiguation. However, twin studies have not typically sought to examine explicit environmental influences, and have generally limited their analyses to parsing variance and covariance. To date, only a handful of twin and adoption studies have examined specific psychosocial influences on childhood disorders.¹⁴⁻²⁰ These studies are in the unique position of more conclusively demonstrating specific environmental influences on childhood psychiatric disorders, and are beginning to fill the gap between studies of environmental influences and studies of genetic influences.

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