

Conduct Problems in Children and Adolescents

A Twin Study

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Background: Evidence supports a genetic influence on conduct problems as a continuous measure of behavior and as a diagnostic category. However, there is a lack of studies using a genetically informative design combined with several different informants and different settings.

Objectives: To examine genetic and environmental influences on conduct problems rated by parent and teacher reports and self-reports and to determine whether their ratings reflect a common underlying phenotype.

Design: A twin study design was used to examine conduct problem scores from ratings by teachers, parents, and twins themselves.

Setting: General community.

Participants: Twins aged 5 to 17 years participating in the Cardiff Study of All Wales and North England Twins (CaStANET) project.

Main Outcome Measures: Conduct problem scale from the Strengths and Difficulties Questionnaire.

Results: Conduct problem scores were significantly heritable based on parent and teacher reports and self-reports. Combining data from all 3 informants showed that they are rating a common underlying phenotype of pervasive conduct problems that is entirely genetic, while teacher ratings show separate genetic influences that are not shared with other raters.

Conclusions: Conduct problems are significantly heritable based on parent and teacher reports and self-reports, and are also influenced by environmental effects that impinge uniquely on children from the same family. There is a cross-situational conduct problems' phenotype, underlying the behavior measured by all informants, that is wholly genetic in origin. No significant influence of shared environmental effects was found.

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IN A RECENT GOVERNMENT SURVEY¹ of the mental health of children in the United Kingdom, conduct disorder emerged as the most common child psychiatric disorder and one of the most frequent reasons for referral to specialist services. Children with conduct disorder are more likely to have comorbid substance abuse, depression, and anxiety,^{2,3} and in adult life are much more likely to develop antisocial personality disorder and other psychiatric disorders.^{4,5} Follow-up studies have found high rates of premature death, often from violent causes,⁶ and up to 46% of those who attempt suicide have had conduct disorder.⁷ Even those without a frank psychiatric disorder in adulthood have pervasive social difficulties compared with their peers.⁸

There has been increasing awareness in recent years that genetic influences have a role in the development of conduct problems, whether measured as

a continuum of behavior or as a diagnostic category. A recent meta-analysis⁹ reported a heritability of 41% (additive and nonadditive genetic effects) for antisocial behavior measured in various ways. It is unlikely that there will turn out to be genes for these behaviors, in the sense that a specific genetic constellation would provide necessary and sufficient cause. Rather, the path from genes to behavior is likely to be a complex one involving environmental stresses and individual differences in liability. There is evidence supporting a genetic influence on conduct disorder as a diagnostic category^{10,11} and on continuous measures of conduct problems and antisocial behavior,¹²⁻²³ although findings vary among studies.

Discrepant findings are likely to be the result of different sample characteristics and measurement strategies and the fact that these behaviors probably represent a heterogeneous group.^{24,25}

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Because children's behavior can vary in different settings, the most valid measurements are those that include data from more than one informant and from more than one setting. Behavioral genetic approaches can indicate the extent to which different informants are rating the same substrate of behavior and the degree of unique perspective that different informants contribute. The present investigation examines genetic and environmental influences on a dimensional measure of conduct problems using parent and teacher reports and self-reports from the Strengths and Difficulties Questionnaire (SDQ),²⁶ a measure widely used in the United Kingdom clinically and epidemiologically.

METHODS

SAMPLE

The Cardiff Study of All Wales and North England Twins (CaStANET) is a population-based twin register of all twin births in Wales and greater Manchester, England. This investigation is based on the subsample of families from south Wales: 1109 twin pairs aged 5 to 17 years, consisting of all twin births between January 1, 1980, and December 31, 1991, in the former counties of Mid and South Glamorgan. The methods used to identify and trace twins have been described elsewhere.²⁷

MEASURES

Data collection was by mail. Parents were asked to complete a packet of questionnaires about their twins, including the SDQ.²⁶ Three reminders were sent, and telephone reminders were used when numbers could be traced. Parental consent was obtained to contact a teacher, who was then asked to complete the teacher version of the SDQ. Self-report versions were sent to twins 11 years and older. The SDQ is a brief behavioral screening questionnaire that can be completed in 5 minutes. Its brevity and its inclusion of strengths as well as difficulties make it particularly suitable for use in general population samples. It includes a continuous measure of behavior problems, the Conduct Problems Scale. The following 5 items contribute to the Conduct Problems Scale: "Often has temper tantrums or hot tempers; generally obedient, usually does what adults request; often fights with other children or bullies them; often lies or cheats; steals from home, school or elsewhere." Parents were asked to report behavior observed in the past 6 months, and items are scored "not true" (0), "somewhat true" (1), or "certainly true" (2), with reverse scoring when appropriate. This conduct scale correlates highly with the conduct/externalizing scales of the longer-established Rutter Scales and the Achenbach Child Behavior Checklist.^{26,28} Zygosity was assigned using a twin similarity questionnaire that is more than 90% accurate in distinguishing monozygotic (MZ) from dizygotic (DZ) twins.^{29,30}

ANALYSES

Univariate Analyses

The influence of latent genetic factors (the sum of effects of individual alleles at a locus), shared environmental factors, and unique environmental factors on conduct problem scores from parents, teachers, and adolescents were first estimated in univariate models. Because MZ twins share 100% of their genes and DZ twins share, on average, 50%, additive genetic effects are reflected in a pattern of correlations by which the MZ correlation

is twice the DZ correlation. When DZ correlations are less than half the MZ correlations, nonadditive or dominance genetic effects can be examined. This refers to interaction effects between alleles at the same locus. Dizygotic twins share only 25% of non-additive or dominance genetic effects, so these will be reflected in a DZ correlation that is less than half the MZ correlation. Alternatively, when DZ correlations are less than half the MZ correlation, a sibling interaction model can be tested; this model examines genetic and environmental influences in the presence of sibling interactions. Shared environmental influences are those that tend to make twins more similar, and will inflate DZ correlations relative to MZ correlations, while unique environmental influences are those that make twins different from one another. They are seen in the extent to which the MZ correlation is less than 1.0. The overall acceptability of a model is evaluated using a χ^2 test (assessing goodness of fit) and Akaike Information Criterion (assessing parsimony), which has a more negative value the better the fit. The significance of individual parameters within a model is assessed by constraining their effects to 0 and then calculating the change in χ^2 for the model. A significant deterioration in the goodness of fit implies a significant influence from the constrained parameter. A commercially available software program (SPSS for Windows) was used³¹ to obtain descriptive statistics and the covariance matrices for genetic analysis. A software package (Mx)³² was used for genetic model fitting. The methods and principles of the model-fitting procedure are described in detail by Neale and Cardon.³³ Before model fitting, scores were transformed to approximate normality using $\log(\text{score} + 1)$.

Multivariate Analyses

Data from all informants were subsequently examined together in the adolescent sample, in which 3 sources of data were potentially available. For these analyses, scores were first standardized for age and sex so that any influence of age or sex would not bias parameter estimates. To maximize the available data, a raw data approach in the software package (Mx) was used at this stage. This removes the need to summarize data in covariance matrices across all informants, which would result in the exclusion of any twin pairs in which data from one informant were missing. This raw maximum likelihood approach generates a value ($-2 \times \log$ likelihood), which is then compared with a saturated model to generate a goodness-of-fit χ^2 .³²

First, an independent pathway model was tested (**Figure 1**). This tests the extent to which genetic and environmental influences are shared across informants, while also allowing for discrete genetic and environmental influences on ratings from each informant. Next, a more stringent model, described as a psychometric or common pathway model,³³ was tested (**Figure 2**). This type of model implies that parents, teachers, and adolescents are all rating the same underlying conduct problems' phenotype (with contributing genetic and environmental influences), but allows for rater-specific estimates of genetic and environmental influences. The arrows from the latent phenotype, "P," represent the factor loadings of the latent phenotype on each of the observed measures. This type of model was chosen because it best represents the sources of data herein, with each rater basing his or her appraisal on different circumstances (ie, teachers rating behavior in school, parents rating behavior primarily at home, and adolescents rating behavior across settings). Finally, a simple multiple rater model was tested (**Figure 3**). This again allows the 3 observed measures (parent, teacher, and adolescent ratings) to be influenced by a latent conduct problems' phenotype. It also allows for variation in the extent to which different raters contribute to the latent phenotype and for variation in the error associated with each rater, but it does not allow for discrete genetic and shared environmental influences on each observed measure.

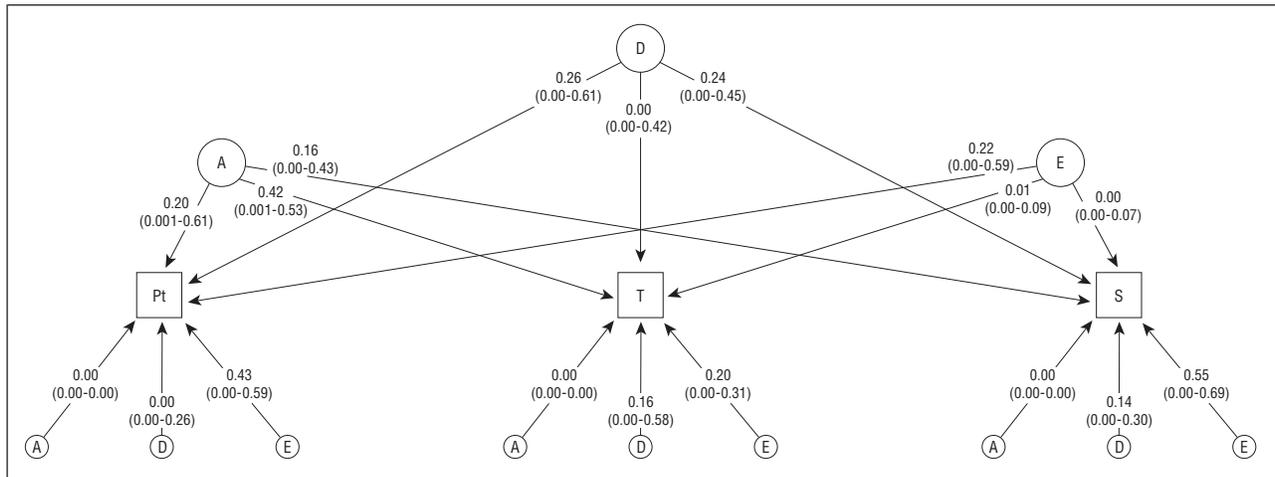


Figure 1. Independent pathway model. Data are shown as components of variance (95% confidence interval). The fit of an ADE model is as follows: χ^2_{24} goodness of fit=14.52, $P=.93$, AIC=-33.48. The fit of an ACE model is as follows: χ^2_{24} goodness of fit=19.23, $P=.74$, AIC=-28.77. A indicates additive genetic effects; AIC, Akaike Information Criterion; C in ACE, shared environmental effects; D, nonadditive or dominance genetic effects; E, unique environment (which includes measurement error); Pt, parent report; S, adolescent self-report; and T, teacher report.

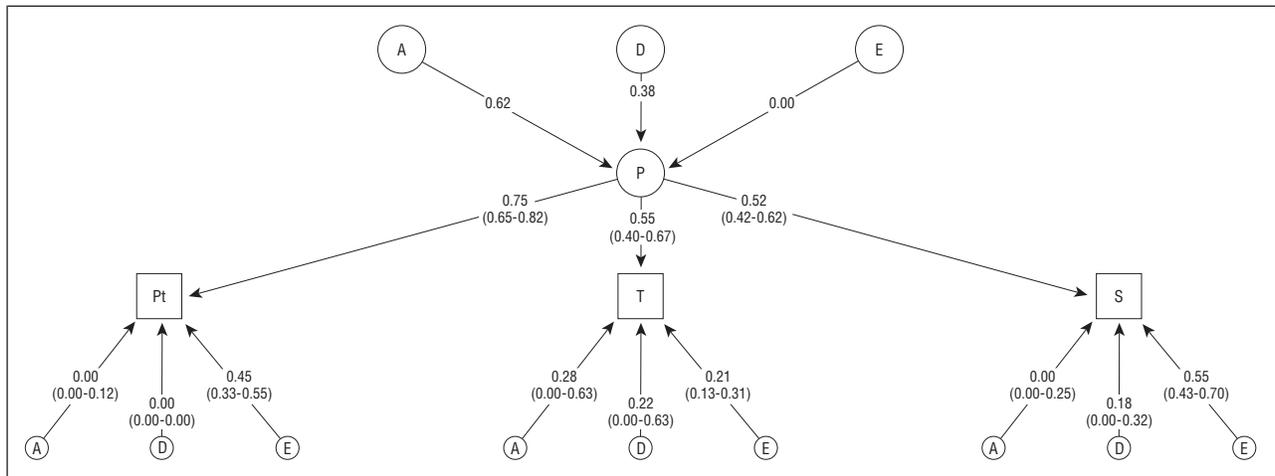


Figure 2. Common pathway model. Data are shown as components of variance (95% confidence interval). The variance of the latent variable, "P," was constrained to equal 1.00. The fit of an ADE model is as follows: χ^2_{28} goodness of fit=16.81, $P=.95$, AIC=-39.19. The fit of an ACE model is as follows: χ^2_{28} goodness of fit=19.52, $P=.88$, AIC=-36.48. A indicates additive genetic effects; AIC, Akaike Information Criterion; C in ACE, shared environmental effects; D, nonadditive or dominance genetic effects; E, unique environment (which includes measurement error); P, conduct problems' phenotype common to all raters; Pt, parent report; S, adolescent self-report; and T, teacher report.

RESULTS

Replies were received from 682 families, a response rate of 61.5% from an initial sample of 1109. There were 12 families in which either the parent or one of the twins failed to return a questionnaire, leaving 670 families who returned parent- and twin-rated measures. Of these families, permission to contact a teacher was given by 561 (83.7%), and 443 teachers replied (79.0% of those families giving consent). Adolescent self-report packs were sent to 570 twin pairs aged 11 to 17 years, and were returned by 286 twin pairs, a response rate of 50.2%.

The age of the twins ranged from 5 to 17 years, with a mean for both sexes of 10.6 years. There were 14 pairs in which zygosity could not be determined because of missing or inconsistent data.

A comparison of responders and nonresponders using Townsend Scores,³⁴ which are based on National Cen-

sus data for each electoral ward, revealed no significant ($P=.10$) sociodemographic differences between the 2 groups, suggesting that the sample is representative of the local population in sociodemographic terms. Furthermore, a comparison with population norms for the SDQ showed that mean conduct problem scores for this sample are not significantly different from those in the general population. For twins, the mean (95% confidence interval) scores were as follows: parent report, 1.83 (1.39-2.27); teacher report, 0.98 (0.51-1.45); and self-report, 2.41 (1.85-2.97). For singletons, the mean (95% confidence interval) scores were as follows: parent report, 1.60 (1.35-1.85); teacher report, 0.90 (0.64-1.16); and self-report, 2.20 (1.88-2.52).

Mean conduct problem scores, standard deviations, correlations, and final sample sizes of twin pairs with complete data are shown in **Table 1**. Conduct problem scores were higher in male than female twins, in keep-

ing with the existing literature, for parent and teacher reports and self-reports (parent-rated $t_{1300}=3.3, P<.001$; teacher-rated $t_{831}=3.4, P<.001$; and adolescent-rated $t_{530}=2.6, P=.01$). There were no significant differences in mean scores between MZ and DZ twins for any of the measures (parent-rated $t_{1273}=0.65, P=.50$; teacher-rated $t_{809}=1.6, P=.10$; and adolescent-rated $t_{519}=-0.2, P=.90$). The F ratio of variance in MZ/DZ pairs was not significant for any of the measures (parent-rated $F=1.4, P=.20$; teacher-rated $F=2.4, P=.20$; and adolescent-rated $F=.02, P=.90$). The correlation between conduct problem scores and age was significant for parent report (Spearman

$\rho=-0.09, P=.02$), but not for other informants. The distribution of conduct scores was positively skewed so scores were transformed to approximate normality before model fitting using $\log(\text{score}+1)$. After transformation, skewness and kurtosis indices were between -1.0 and 1.0 , implying that not much distortion is to be expected.³⁵

MODEL FITTING: UNIVARIATE ANALYSES

Table 2 presents the results of univariate model fitting for the 3 informants. For results from different informants to be comparable (because self-reports are only available for 11- to 17-year-old subjects), results for children (those aged <11 years) and adolescents (those aged 11-17 years) are presented separately.

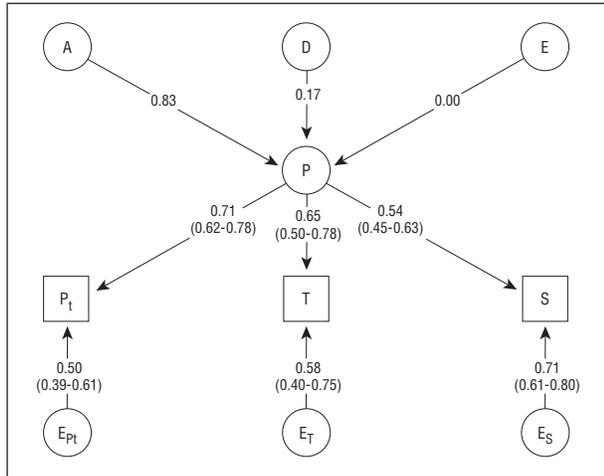


Figure 3. Simple multiple rater model. Data are shown as components of variance (95% confidence interval). The variance of the latent variable, "P," was constrained to equal 1.00. The fit of an ADE model is as follows: χ^2_{34} goodness of fit=58.48, $P=.006$, AIC=-9.52. The fit of an ACE model is as follows: χ^2_{34} goodness of fit=58.62, $P=.005$, AIC=-9.38. A indicates additive genetic effects; AIC, Akaike Information Criterion; C in ACE, shared environmental effects; D, nonadditive or dominance genetic effects; E, unique environment (which includes measurement error); E_{Pt} , E_S , and E_T , error for parent rating, self-rating, and teacher rating, respectively; P, conduct problems' phenotype common to all raters; P_t , parent report; S, adolescent self-report; and T, teacher report.

Parent-Rated Conduct Problems

Complete data were available from 621 twin pairs for these analyses. The pattern of correlations (0.63 MZ/0.16 DZ in male pairs and 0.53 MZ/0.33 DZ in female pairs) suggested genetic influence on the variance of scores, and this was supported by model fitting. The best-fitting models show that more than half the variance was accounted for by additive genetic influences, while the remainder was influenced by unique environmental factors. Shared environmental influences and nonadditive genetic influences were insignificant.

Teacher-Rated Conduct Problems

The same model-fitting procedure was applied to teacher-rated SDQ conduct problems. There were 39 twin pairs in which the twins were rated by different teachers, and these were excluded from the present analyses. Complete data were available from 387 twin pairs. The pattern of correlations (0.69 MZ/0.13 DZ in male pairs and

Table 1. Data for Conduct Problem Scores in MZ and DZ Pairs

| Variable | MZ Pairs | | DZ Pairs | | | |
|-----------------------|----------|--------|----------|--------|--------------|--------|
| | Male | Female | Male | Female | Opposite Sex | |
| | | | | | Male | Female |
| Parent Report | | | | | | |
| Mean score | 2.18 | 1.68 | 2.06 | 1.79 | 1.96 | 1.80 |
| SD | 1.75 | 1.82 | 1.92 | 1.54 | 1.82 | 1.85 |
| No. of pairs | 117 | 148 | 95 | 78 | 183 | |
| Correlation | 0.63 | 0.53 | 0.16 | 0.33 | 0.17 | |
| Teacher Report | | | | | | |
| Mean score | 1.15 | 0.86 | 1.32 | 0.77 | 0.98 | 0.92 |
| SD | 1.94 | 1.64 | 2.12 | 1.23 | 1.74 | 1.65 |
| No. of pairs | 74 | 92 | 54 | 49 | 118 | |
| Correlation | 0.69 | 0.75 | 0.13 | 0.26 | 0.23 | |
| Self-report | | | | | | |
| Mean score | 2.73 | 2.09 | 2.54 | 2.06 | 2.38 | 2.16 |
| SD | 2.22 | 1.70 | 1.94 | 1.59 | 1.67 | 1.69 |
| No. of pairs | 47 | 63 | 37 | 35 | 72 | |
| Correlation | 0.39 | 0.53 | 0.05 | 0.17 | 0.18 | |

Abbreviations: DZ, dizygotic; MZ, monozygotic.

Table 2. Results of Model Fitting Using SDQ Conduct Problem Scores From Different Raters

| Model | Additive Genetic Variance* | Common Environmental Variance* | Dominance Genetic Variance* | Unique Environmental Variance* | Model χ^2 (df) | P Value | AIC |
|-------------------------------------|----------------------------|--------------------------------|-----------------------------|--------------------------------|-----------------------|---------|--------|
| Parent Report (Adolescents) | | | | | | | |
| ACE | 0.54 (0.27 to 0.64) | 0.00 (0.00 to 0.00) | ... | 0.46 (0.36 to 0.59) | 20.45 ₍₁₂₎ | .06 | -3.55 |
| ADE | 0.33 (0.00 to 0.64) | ... | 0.22 (0.00 to 0.65) | 0.45 (0.34 to 0.58) | 19.92 ₍₁₂₎ | .07 | -4.08 |
| AE† | 0.54 (0.41 to 0.64) | ... | ... | 0.46 (0.36 to 0.59) | 20.45 ₍₁₃₎ | .08 | -5.56 |
| Teacher Report (Adolescents) | | | | | | | |
| ACE | 0.77 (0.41 to 0.84) | 0.00 (0.00 to 0.32) | ... | 0.23 (0.16 to 0.34) | 10.06 ₍₁₂₎ | .61 | -13.90 |
| ADE | 0.75 (0.00 to 0.84) | ... | 0.01 (0.00 to 0.79) | 0.23 (0.16 to 0.34) | 10.06 ₍₁₂₎ | .61 | -13.90 |
| AE† | 0.77 (0.66 to 0.84) | ... | ... | 0.23 (0.16 to 0.34) | 10.06 ₍₁₃₎ | .69 | -15.90 |
| Self-report (Adolescents) | | | | | | | |
| ACE | 0.35 (0.00 to 0.48) | 0.00 (0.00 to 0.00) | ... | 0.65 (0.52 to 0.79) | 16.26 ₍₁₂₎ | .18 | -7.73 |
| ADE | 0.22 (0.00 to 0.48) | ... | 0.14 (0.00 to 0.49) | 0.64 (0.50 to 0.79) | 16.12 ₍₁₂₎ | .19 | -7.88 |
| AE† | 0.35 (0.20 to 0.48) | ... | ... | 0.65 (0.52 to 0.79) | 16.26 ₍₁₃₎ | .24 | -9.74 |
| Parent Report (Children) | | | | | | | |
| ACE | 0.58 (0.35 to 0.66) | 0.00 (0.00 to 0.18) | ... | 0.42 (0.34 to 0.53) | 15.57 ₍₁₂₎ | .21 | -8.43 |
| ADE | 0.42 (0.00 to 0.68) | ... | 0.17 (0.00 to 0.67) | 0.41 (0.31 to 0.55) | 9.33 ₍₁₂₎ | .68 | -14.68 |
| AE† | 0.58 (0.45 to 0.68) | ... | ... | 0.42 (0.32 to 0.55) | 9.59 ₍₁₂₎ | .73 | -16.41 |
| Teacher Report (Children) | | | | | | | |
| ACE | 0.70 (0.59 to 0.78) | 0.00 (0.00 to 0.08) | ... | 0.30 (0.22 to 0.41) | 29.40 ₍₁₂₎ | .003 | 5.40 |
| ADE | 0.00 (0.00 to 0.00) | ... | 0.73 (0.28 to 0.79) | 0.27 (0.21 to 0.36) | 20.54 ₍₁₂₎ | .06 | -3.46 |
| AEs‡ | 0.81 (0.71 to 0.87) | -0.12 (-0.20 to -0.03)‡ | ... | 0.19 (0.13 to 0.29) | 20.90 ₍₁₂₎ | .05 | -3.10 |
| AE | 0.70 (0.60 to 0.78) | ... | ... | 0.30 (0.22 to 0.41) | 29.40 ₍₁₃₎ | .006 | 3.40 |
| DE† | ... | ... | 0.73 (0.64 to 0.79) | 0.27 (0.21 to 0.36) | 20.54 ₍₁₃₎ | .08 | -5.46 |

Abbreviations: A, additive genetic effects; AIC, Akaike Information Criterion; C, shared environmental effects; D, nonadditive or dominance genetic effects; E, unique environmental effects; SDQ, Strengths and Difficulties Questionnaire.

*Data are given as standardized components of variance (95% confidence interval). Ellipses indicate that the parameter is fixed at 0 and not estimated in the model.

†The best-fitting model.

‡The sibling interaction effect.

0.75 MZ/0.26 DZ in female pairs) suggested genetic influences. The results of model fitting are summarized in Table 2, and show that, in adolescents, almost three quarters of the variance in scores was attributable to genetic influences, with the remainder accounted for by unique environmental factors. In children, the same proportion of variance was due to genetic influence, but in this case the genetic influence was nonadditive. A sibling interaction model was also tested, but fitted the data less well than the nonadditive or dominance genetic effects and unique environmental effects model. Again, the influence of shared environment was insignificant.

Adolescent Self-rated Conduct Problems

Complete data were available from 254 twin pairs for these analyses. The pattern of correlations (0.39 MZ/0.05 DZ in male pairs and 0.53 MZ/0.17 DZ in female pairs) suggested genetic influence. The same model-fitting procedure was applied, and results are summarized in Table 2. Of the variance in scores, 35% was accounted for by additive genetic influences, with the remainder due to influences of the twins' unique environment. Again, the influences of the shared environment and the nonadditive genetic effects were insignificant.

MULTIVARIATE MODELS COMBINING PARENT-, TEACHER-, AND ADOLESCENT-RATED SCORES

Results for the independent pathway model are shown in Figure 1. A model including dominance genetic effects is shown, but a model including additive genetic effects, shared environmental effects, and unique environmental effects was also tested; fit statistics for both models are given in the legend to Figure 1. The model including additive genetic effects, nonadditive or dominance genetic effects, and unique environmental effects provides a slightly better fit.

Results for the common pathway model are shown in Figure 2. Again, a model including dominance genetic effects is shown, and fit statistics for the model including additive genetic effects, nonadditive or dominance genetic effects, and unique environmental effects and the model including additive genetic effects, shared environmental effects, and unique environmental effects are given in the legend to Figure 2; the model including additive genetic effects, nonadditive or dominance genetic effects, and unique environmental effects gives a slightly better fit with the data. A model including additive genetic effects, shared environmental effects, and unique environmental effects estimated all shared environmental influences at 0. The common pathway model, which tests whether a shared underlying phe-

notype accounts for similarities in ratings from different observers, is more psychologically meaningful and a more stringent representation of the data. It can be compared with the more general independent pathway model (change in $\chi^2=2.29$, $P=.68$), revealing a nonsignificant change of fit. This suggests that the common pathway model is a better explanation of the data than the independent pathway model. The common pathway model indicated that a wholly heritable common phenotype (genetic influence = additive genetic effects + nonadditive or dominance genetic effects = 1.00) underlies cross-situational conduct problems that are rated by different informants in different settings. In addition, the common pathway model suggests discrete genetic effects on teacher ratings (additive and nonadditive) and on self-ratings (nonadditive). When these discrete effects were constrained to 0 and the fit compared with a full model including additive genetic effects, nonadditive or dominance genetic effects, and unique environmental effects, the genetic effects on self-ratings were not significant (change in $\chi^2=5.37$, $P=.07$), but discrete genetic effects on teacher ratings were significant (change in $\chi^2=16.02$, $P<.001$).

Results for the simple multiple rater model are shown in Figure 3. This model provides a poor fit with the data. The multiple rater model is more stringent than the other 2 models because it offers fewer parameters and, in particular, does not allow for any separate influences, other than error, on each measure. This restriction results in a significant deterioration of fit compared with the common pathway model (change in $\chi^2=41.67$, $P<.001$), indicating that the presence of discrete rater influences provides a better explanation of the data.

COMMENT

The results of this investigation lend further support to existing evidence suggesting that conduct problems in children and adolescents have a heritable component. In this community sample of twins, a substantial genetic influence has been shown using a dimensional measure that is widely used in clinical practice and epidemiology.²⁶ Parent, teacher, and adolescent reports all provided significant heritability estimates. There was no significant effect of the shared environment in any of these models, and only in the teacher-report scores for adolescents were the confidence intervals fairly wide around this estimate (0%-32%), suggesting that a larger sample might have detected a significant effect.

When conduct scores from all informants were combined in a single model, the best results were achieved for a model that included an underlying phenotype that was common to all informants and separate genetic and environmental influences acting on each measure. This suggested that all informants were, to a considerable extent, rating a common underlying phenotype that was wholly influenced by genetic factors, additive and nonadditive. This highly genetic phenotype refers only to aspects of behavior that are common to all informants and, therefore, pervasive across settings. However, ratings by teachers also showed a significant specific genetic influence, suggesting that different genes may be influencing

the behavior that teachers were rating. Conduct problems occurring in school seem to be different from those that are pervasive across settings. This is a phenomenon that has also been observed in ratings of antisocial behavior in younger children³⁶ and in childhood hyperactivity.^{37,38} It supports the value of SDQ data from teachers because they are clearly contributing a measure of behavior that is not tapped by parent reports or self-reports. It also has implications for phenotype definition in molecular genetic research, implying that different genes are influencing measures from different informants.

Another approach to data from multiple informants is to examine the extent of rater bias, but this approach is better suited to multiple raters of behavior in one setting (eg, mothers and fathers rating behavior at home) and does not easily generalize to multiple raters in different settings. Given that children may behave differently at home and at school, it is possible that parents and teachers provide valid ratings of behavior in their respective domains, and to what extent this represents rater bias or true phenotypic differences is difficult to disentangle.

The genetic literature on conduct/externalizing problems in children and adolescents is unusual insofar as more than any other behavioral trait, it has shown a significant effect of shared environment.²⁴ However, the lack of shared environmental influence shown herein is a far from unique finding. These results are in agreement with those of Rowe,¹⁴ who used a self-report delinquency scale in adolescents; Schmitz et al,¹⁸ who used the delinquency subscale of the Child Behavior Checklist in 4- to 18-year-old subjects; McGuffin and Thapar,²⁰ who used the Olweus adolescent self-report scale; Slutske et al,¹¹ who made retrospective *DSM-III-R* conduct disorder diagnoses in adults; Eaves et al,¹⁰ who used interview-based diagnoses of conduct disorder and oppositional defiant disorder in children and adolescents; and Miles et al,²³ who used interview-derived symptom counts. The only other twin study³⁶ we are aware of that has used multiple informants in different settings also found a highly heritable shared latent phenotype with no significant influence from the shared environment. Arsenault et al³⁶ used data from mothers, teachers, observers, and children themselves, and showed that heritability was higher (82%) for a shared latent phenotype than for corresponding single measures of antisocial behavior, as found in this investigation. In their sample of 5-year-old twins, a common pathway model was the best explanation of the data, as found herein. Their results and ours support the notion that conduct problems that are pervasive across settings are a highly genetic phenotype in young children and adolescents. This is in keeping with a theory of antisocial development³⁹ that suggests that pervasive antisocial behavior that develops in childhood is a result of heritable neurodevelopmental abnormalities and is likely to persist into adolescence and adulthood, although longitudinal cross-situational data are required to examine this question further.

The lack of significant shared environmental influences in this investigation warrants some consideration. When shared environmental influences depend on

genotype (as in gene \times environment interactions), then their effects are subsumed in the estimate of genetic influences in twin analyses. There is some evidence^{40,41} suggesting both gene \times environment interaction and correlation in conduct problems and related behaviors, and this may have contributed to the lack of any significant shared environmental influence in the present investigation.

The estimates for unique environmental effects represent those environmental influences that are not shared and tend to make individuals different from one another, while also including measurement error. Unique environmental influences emerged for each informant, but not for the shared underlying phenotype. This is in keeping with another twin study¹⁹ that used data from mothers, fathers, and twins, and found that the influence of the nonshared environment reduced and disappeared when comparing single- with multiple-rater models. This observation is likely to reflect a reduction in error variance when data from multiple informants are combined. For observed phenotypes, nonshared environment estimates include all measurement error, but with a latent phenotype, as used in the multiple-rater models herein, much of this error is taken up elsewhere. Although the present investigation does not measure which specific environmental influences are at work for each informant, a significant role for unique environmental influences is in keeping with other literature. There is evidence^{42,43} suggesting that children, while growing up in the same family, can nevertheless experience quite different environments, and Reiss et al⁴⁴ have shown a strong association between child-specific parental conflict within families and children's behavior problems.

The finding that conduct problems are highly heritable is not in any way a suggestion that these behaviors are wholly genetically determined and, therefore, not amenable to intervention. Environmental influences of the kind that tend to make twins different from one another had a significant influence in this investigation, and appreciating genetic influence is only a small step in understanding the complex pathway from genes to behavior. Complex gene-environment correlation and interaction suggests that individuals with a genetic propensity to certain behaviors will seek out environments that exacerbate the behavior. Nature and nurture are inextricably entwined.

Three limitations of the present study warrant discussion. First, the findings presented are based on questionnaire data; diagnostic interviews are obviously preferable, but are expensive and were not a feasible option for this investigation. Second, another limitation is the response rate: 61.5% of a population-based sample of 1109 twin pairs. However, comparison of responders with non-responders revealed no significant differences in socio-demographic measures reflecting neighborhood social deprivation. Because these are the type of environmental stresses associated with conduct problems, it was assumed that the sample adequately represents the general population and there was no systematic bias with regard to conduct problems. In addition, a comparison of mean conduct problem scores with population norms revealed no significant difference. However, we cannot

completely discount the possibility of a response bias. Third, the main measure of conduct problems^{26,28} used is a relatively new and brief measure, with 5 items that generate the Conduct Problems Scale. Despite this, its brevity and its inclusion of strengths as well as difficulties were seen as an advantage in a general population postal survey of this sort.

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