

Heritability and the Comorbidity of Attention Deficit Hyperactivity Disorder With Behavioral Disorders and Executive Function Deficits: A Preliminary Investigation

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The heritability and comorbidity of attention deficit hyperactivity disorder (ADHD) with conduct disorder (CD), oppositional defiant disorder (ODD), and executive function (EF) deficits were examined in 224 child twins (140 monozygotic and 84 dizygotic). The Coolidge Personality and Neuropsychological Inventory for Children (Coolidge, 1998), a standardized, 200-item, *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994) aligned, parent-as-respondent inventory, assessed psychopathology. Structural equation model fitting revealed that the individual scale heritabilities were substantial: .82 for ADHD, .74 for CD, .61 for ODD, and .77 for EF deficits. The results of the multivariate twin analyses suggest that ADHD shares most of its genetic liability with CD, ODD, and EF deficits. Thus, the findings argue for a common biological risk underlying these commonly comorbid externalizing behavior problems and cognitive deficits. The residual genetic variance provides preliminary support for additional genetic influences underlying CD, ODD, and EF that are independent of ADHD.

Increasing attention was given to the genetic bases of the attention deficit hyperactivity disorder (ADHD), and heritability estimates range from .39 to .91 of the total variance (e.g., Levy, Hay, McStephen, Wood, & Waldman, 1997; Sherman, Iacono, & McGue, 1997; Willerman, 1973). These estimates appear to vary as a function of the method for obtaining the heritability estimate (e.g., Falconer's, 1990, estimate; structural equation modeling; and so on), the behavioral raters (teachers or parents; Sherman et al., 1997), and whether ADHD is viewed dimensionally or categorically (Levy et al., 1997). ADHD's specific mode of inheritance is still undetermined, although it has been suggested that it may be transmitted in a major locus fashion (Pennington & Ozonoff, 1996).

Recent evidence also suggests at least three important comorbidity issues: conduct disorders (CD), oppositional defiant disorders (ODD), and executive function (EF) deficits of the frontal lobes. A review of 29 studies of the comorbidity between ADHD and CD (Biederman, Newcorn, & Sprich, 1991) concluded that these two disorders are comorbid in approximately 30% to 50% of all cases. Kuhne, Schachar, and Tannock (1997) investigated three groups of children: 33 ADHD children, 46 ADHD children with ODD, and 12 ADHD children with CD. The children were compared on various measures of school-related behavior, emotional well-being, and parental psychopathology. Kuhne et al. found that the comorbid condition of ADHD with CD led to higher aggression scores, higher anxiety levels, decreased self-esteem, and significantly higher ratings of maternal psychopathology. They also found a distinctively different disruptive profile for ADHD with ODD. The latter was characterized by significantly greater social withdrawal. Their findings support earlier work by Biederman et al. (1991), who argued that ADHD with ODD may be a distinct subtype from ADHD alone and ADHD with CD. These findings also indirectly support the distinction between ODD and CD. As previously noted, Lahey, Loeber, Quay, Frick, and Grimm (1992) demonstrated that many children with ODD do not develop later CD, and the emergence of CD in early adolescence appears to be independent of ODD. Kuhne et al. noted that there are limited statistical evaluations of the covariations of the symptoms of ODD and CD. Their study of 33 "pure" ADHD children, 46 ADHD plus ODD children, and 12 ADHD plus CD children led them to the hypothesis that ODD could not be ruled out as a milder form of CD, but that a linear relation between the two was probably too simplistic. They came to the conclusion that the comorbid disorders do share some common vulnerabilities, but that their comorbid differential effects on ADHD suggested that they are distinct subtypes with differing clinical paths.

The term EF has been defined in the literature (e.g., Luria, 1966; Pineda et al., 1998; Reader, Harris, Schuerholz, & Denckla, 1994; Welsh & Pennington, 1988; Weyandt & Willis, 1994) as a unique domain of cognitive abilities that involves organization in space and time, selective inhibition, response preparation, goal attainment, planning, and cognitive flexibility. This set of functions is thought to be

relatively independent from other cognitive functions, such as sensation, perception, language, and memory, yet thought to overlap with attention, reasoning, and problem solving. The neural substrate for the EF has not been precisely delineated, but they are thought to be mediated by prefrontal cortices of the brain (e.g., Pennington & Ozonoff, 1996; Stuss, 1992). Reader et al., in a study of 48 ADHD children (mean age 9.5 years), found that ADHD children with and without reading disabilities were similarly impaired on some but not all measures of EF. They concluded that ADHD children seem to be at risk for EF deficits. Pennington and Ozonoff, in a review of 18 studies of EF deficits in ADHD, found that 15 studies showed a significant difference between ADHD children and controls on at least one or more measures of EF. They concluded that ADHD children may have a mix of specific deficits (like an essential EF deficit) and some general deficits (such as a general cognitive inefficiency).

The heritability estimates for CD were found to be similar to the range of heritability estimates for ADHD, from about .27 to .78 (e.g., Cadoret, Leve, & Devor, 1997; Coolidge, Thede, & Jang, in press; Eaves et al., 1997; Slutske et al., 1997). There appears to be good evidence for the comorbid heritability of ADHD and CD and emerging evidence for the comorbid heritability of ADHD and ODD (e.g., Comings, Chen, Wu, & Muhleman, 1999; Hewitt et al., 1997; Nadder, Silberg, Eaves, Maes, & Meyer, 1998; Silberg et al., 1996). Heritability studies of EF deficits are more scarce. One reason is that, unlike the diagnoses of ADHD, CD, and ODD, established criteria for EF deficits have not appeared in any version of the *Diagnostic and Statistical Manual of Mental Disorders*. Thus, a standard syndromal description and consensus of criteria are lacking. Nevertheless, heritability studies of ADHD and comorbidity evidence of ADHD with EF deficits indirectly suggest a strong and perhaps primary genetic link. However, no single study to date has provided heritability estimates for the comorbidity of ADHD with CD, ODD, and EF deficits.

A predominant research model of heritability is the study of twins. Twin studies provide estimates of the relative contributions from genetic and environmental effects on behavior by contrasting the similarities of monozygotic (MZ) twin pairs (who share 100% of their segregating genes) with dizygotic (DZ) twin pairs (who share, on average, 50% of their segregating genes). The purpose of this article was to examine child and adolescent behavior in a twin model to determine the relative contributions of genetic and environmental factors to the comorbidity of ADHD with CD, ODD, and EF deficits. Prior studies also suggested that there is sufficient variation in nonreferred, community-based samples to assess the heritability of psychopathologies (Jang, Livesley, & Vernon, 1998; Jang, Livesley, Vernon, & Jackson, 1996). The twins were assessed with the Coolidge Personality and Neuropsychological Inventory (CPNI; Coolidge, 1998; Coolidge, Aksamit, & Becker, 1994; Coolidge et al., 1990; Coolidge, Reilman, Becker, Cass, & Coolidge, 1992; Coolidge et al., in press), a 200-item, parent-as-respondent question-

naire designed to assess psychopathology according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM-IV]; American Psychiatric Association, 1994) as well as neuropsychological dysfunction. It was hypothesized that ADHD, CD, ODD, and EF deficits would yield sizable heritability coefficients as well as sizable comorbid heritability estimates for ADHD with CD, ODD, and EF deficits.

METHOD

Participants

The parents of twins were recruited through advertisements on the Internet, in local newspapers, and through students in psychology classes at a midwestern university who earned extra credit by identifying parents of twins. Participating parents completed the CPNI on each child as well as a demographic survey. Informed consent was obtained.

The final sample consisted of 224 twins, 70 MZ pairs (34 male pairs and 36 female pairs) and 42 DZ pairs (14 male pairs, 15 female pairs, and 13 male-female pairs). The mean age of the MZ pairs was 8.6 ($SD = 3.1$), and the mean age of the DZ pairs was 8.9 ($SD = 3.1$). The mean age of the parents was 38.2 years ($SD = 6.4$), and 86% of the parents had attained a level of education beyond high school. The mean maternal age at time of birth was 29.0 years ($SD = 5.7$), and ethnicity was as follows: MZ twins: White (82%), Hispanic (6%), Asian (5%), African American (2%), or other (5%); DZ twins: White (98%) and Asian (2%). Conception followed the use of fertility drugs for 15.2% of the twins. A small proportion of parents reportedly exposed the twins to potentially harmful substances prior to birth such as alcohol (4%), tobacco (9%), injury (1%), serious illness (1%), and prescription drugs (13%).

Zygosity was diagnosed using a 10-item questionnaire based on a study by Cohen, Dibble, Grawe, and Pollin (1975) and contained items regarding physical similarities (e.g., height, weight, and hair and eye color) and confusion of the twins by parents, family, and strangers. Their questionnaire was demonstrated to be approximately 90% reliable (compared to blood typing). Sixty-eight mothers and 2 fathers completed the questionnaires on their MZ twins, and 41 mothers and 1 father completed the questionnaires on their DZ twins.

Materials

Personality disorders were assessed with the 200-item, parent-as-respondent CPNI (Coolidge, 1998). The CPNI was designed with a threefold purpose: (a) to

assess the 10 personality disorders on Axis 2 of the *DSM-IV* and the 2 personality disorders in its appendix (depressive and passive-aggressive), (b) to measure neuropsychological dysfunction, and (c) to assess *DSM-IV* Axis 1 separation anxiety disorder, ODD, ADHD, and other clinical scales. The CPNI uses a 4-point Likert scale ranging from 1 (*strongly false*) to 2 (*more false than true*) to 3 (*more true than false*) to 4 (*strongly true*), and it is designed to be filled out by a primary caregiver who is intimately acquainted with the child's behavior. This normative sample consists of 329 children, 5 to 17 years of age, and their raw scores were used to establish *T* scores on the personality disorder scales (for greater details, see Coolidge, 1998). The 12 personality disorder scales have a median internal scale reliability (Cronbach's alpha) of .67 and a median test-retest reliability of .81 (4- to 6-week interval). The scale reliability is .61 for the 15-item CD scale, and the test-retest reliability is .87. The scale reliability is .84 for the 8-item ODD scale, and the test-retest reliability is .67. The scale reliability is .91 for the 18-item ADHD scale, and the test-retest reliability is .83. The scale reliability is .86 for the 8-item EF deficits scale, and test-retest reliability is .81. Preliminary validity studies support the use of the CPNI in a variety of clinical and community settings (e.g., Coolidge et al., 1994; Coolidge et al., 1990; Coolidge et al., 1992; Coolidge et al., in press; Friedman, 1998; Philbrick, 1990; Reilman, 1993). Recent studies also provided empirical support for the validity of parent ratings of children's executive functions (Gioia, Isquith, Guy, & Pratt, 1999; Silver, Benton, Goulden, Molho, & Clark, 1999).

Procedure

The CPNI was mailed to the parents along with the demographic survey containing the zygosity questionnaire and informed consent. The parents were instructed to complete the two forms for their twins on separate days to reduce any contrast effects (a preconceived view that MZ twins are more alike than DZ twins) or effects from repeating the same procedure simultaneously.

Statistical Analyses

In a classic twin study, individual differences (i.e., variability in behavioral measures) are decomposed into genetic and environmental sources. First, there are additive genetic influences (a^2), which means that alleles from different loci in the genome contribute in an additive fashion to the liability for a behavioral trait or disorder. Second, there are dominant genetic influences (d^2), which are interactions between alleles at a single locus that influence a trait or disorder. (Note that interactions among alleles at different loci are epistatic effects not modeled in this study.) Third, there are shared environmental influences (c^2) that, by definition, contribute to twin similarity or correlation. Fourth, there are nonshared environmental influ-

ences (e^2) that represent factors unique to a single twin and do not contribute to their similarity.

Univariate twin model. Figure 1 shows the classic twin path model for a univariate analysis. In this case, squares represent observed behavioral phenotypes for a pair of twins. The circles represent unmeasured latent influences on the observed measures; *A* represents latent additive genetic effects, *C* represents latent shared environmental effects (i.e., environmental influences that, by definition, make twins more similar), *D* represents genetic dominance effects, and *E* represents latent unique (individual) environmental effects and measurement error. Double-headed arrows represent the correlations between the latent factors. MZ twins are genetically identical; thus, correlations are fixed at unity for both their *A* and *D* genetic effects. On average, DZ twins share half of their *A* effects and 25% of their *D* effects. The correlation between *C* effects is fixed at 1.0 for both MZ and DZ twins, implying equal *C* effects for the different twin types. Other implicit assumptions of the model are that *A* and *C* effects are uncorrelated, and random mating is operating in the parent generation (not shown).

The *C* and *D* effects are confounded in studies of twins raised together (Neale & Cardon, 1992) and cannot be estimated simultaneously. Thus, in any given analysis, the influence of one of these sources of variation must be assumed to be absent (i.e., fixed to zero). The model is chosen based on the plausibility of the source's influence and the pattern of observed twin correlations.

Maximum likelihood estimation procedures operationalized in Mx, software designed for structural equation modeling of genetically informative data (Neale,

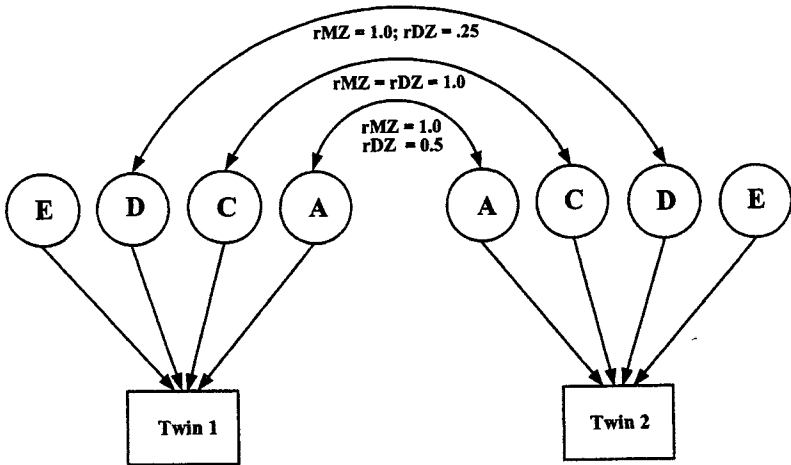


FIGURE 1 Basic twin model.

1999), provided estimates for each parameter specified in the model. Chi-square, goodness-of-fit statistics are used to assess how well the models fit the data. A smaller chi-square value and corresponding higher p value indicate better correspondence between the model and the observed variances and covariances. Significance tests of the individual path coefficients are carried out by constraining paths to zero and applying a chi-square difference test or by estimating the 95% confidence intervals on individual parameters.

Raw data were rank normalized to correct for skewness. Small sex effects were detected for each of the four measures. Because the inclusion of opposite-sex twin pairs can artificially reduce the DZ correlations when sex effects are operating, the normalized scores were corrected for sex effects using standard linear regression methods.

For the multivariate twin analyses, a Cholesky triangular decomposition model was employed (Neale & Cardon, 1992). It is a descriptive, multivariate twin model that provides information about the genetic and environmental sources of variation and covariation among behavioral indexes. The model, as illustrated in Figure 2, allows for two "first" factors, one for additive genetic influences (A) and one for nonshared environmental influences (E). First factors load on each of the four measures. The second A and E factors influence CD, ODD, and EF; the third factors influence ODD and EF only; and the fourth influences EF only. For simplicity, only Twin 1 is depicted, but the same correlations among the latent factors between twins (as shown in Figure 1) are implied. The model decomposes both the variance of each measure and covariance among measures into their genetic and environmental sources.

RESULTS

Twin Correlations

The within-pair correlations for ADHD, CD, ODD, and EF in the MZ and DZ groups are presented in Table 1. The within-trait correlations are shown on the main diagonal of the upper (MZ) and lower (DZ) portions of the table. The MZ correlations ranged from .64 to .81; the DZ correlations were consistently lower, ranging from .12 to .18. For each of the four measures, the MZ correlation was more than twice the DZ correlation, suggesting that twin resemblance was due to both additive and nonadditive (i.e., dominance) genetic effects, special MZ environmental effects, or both. The patterns of within-pair correlations suggest that shared environmental influences common to MZ and DZ twins are not important factors for these behaviors. Within-pair, cross-trait correlations, which provide information regarding the etiology of the covariation among the measures, are shown above and below the main diagonals. The greater MZ cross-trait correlations suggest that genetic influences underlie comorbidity.

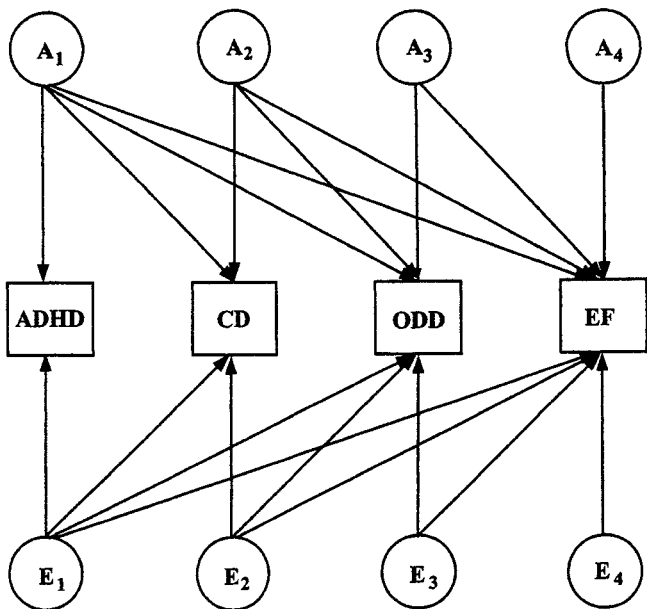


FIGURE 2 Cholesky decomposition.

TABLE 1
Twin Correlations

Twin 2	Twin 1			
	ADHD	CD	ODD	EF
MZ twins (<i>n</i> = 67)				
ADHD	.81	.37	.63	.66
CD	.39	.71	.51	.33
ODD	.54	.36	.64	.57
EF	.62	.33	.48	.79
DZ twins (<i>n</i> = 42)				
ADHD	.18	.14	.18	.16
CD	.13	.18	.12	.24
ODD	.30	.13	.12	.31
EF	.16	.31	.38	.15

Note. Within-trait twin correlations on the diagonals; cross-trait twin correlations on off-diagonals. ADHD = attention deficit hyperactivity disorder; CD = conduct disorder; ODD = oppositional defiant disorder; EF = executive functions; MZ = monozygotic; DZ = dizygotic.

Genetic Model Fitting Results

The univariate twin model (Figure 1) was fit to 2×2 (Twin 1, Twin 2) covariance matrices for each measure separately. Table 2 summarizes the full and best-fitting models including the variance components due to additive genetic (a^2), nonadditive genetic (d^2) and nonshared environmental (e^2) influences, and the corresponding statistics evaluating goodness of fit. Although the full ADE models suggest that additive genetic factors are unimportant, nonadditive genetic effects operating in the absence of additive genetic effects is not highly plausible for polygenic traits. Thus, reduced AE models were tested against the full ADE model to examine the fit of a more parsimonious explanation of the data. Results from the ADHD, CD, ODD, and EF analyses suggest that genetic dominance can be eliminated from the model (i.e., d parameters fixed at zero) without a significant decrement in model fit. The AE model fit the ADHD data well, $\chi^2(5) = 9.31, p = .10$, and estimated that .82 of the variance in ADHD could be attributed to additive genetic influences and .18 to nonshared environmental influences. The simplified models for CD, ODD, and EF tell much the same story, with heritabilities (a^2) of .73, .63, and .79.

The Cholesky decomposition (multivariate twin) model depicted in Figure 2 was fit to 8×8 covariance matrices (four measures for Twin 1 and four measures for Twin 2). The full model decomposed the covariation among the measures into four additive genetic factors (A_1 through A_4) and four nonshared environmental factors (E_1 through E_4). Univariate analyses indicated that there

TABLE 2
Full and Best Fitting Univariate Models

Scale Model	a^2	d^2	e^2	χ^2	df	p
ADHD						
ADE	.00	.84	.16	6.50	4	.17
AE	.82	—	.18	9.31	5	.10
CD						
ADE	.00	.75	.25	2.77	4	.60
AE	.73	—	.27	5.73	5	.33
ODD						
ADE	.00	.65	.35	2.34	4	.67
AE	.63	—	.37	4.08	5	.54
EF						
ADE	.00	.80	.20	2.69	4	.61
AE	.79	—	.21	6.02	5	.31

Note. ADHD = attention deficit hyperactivity disorder; ADE = additive genetic influences, genetics dominance effects, and latent unique (individual) environmental effects and measurement error; AE = additive genetic influences and latent unique (individual) environmental effects and measurement error; CD = conduct disorder; ODD = oppositional defiant disorder; EF = executive functions.

was insufficient power to detect nonadditive genetic effects, thus they were omitted from the multivariate analyses. Table 3 presents the standardized factor loadings. The finding of significant genetic factor loadings among the off-diagonal elements clearly suggests that there are important genetic influences common among these four measures. However, the magnitude of the loadings for A_2 , A_3 , and A_4 suggest that the genetic influences cannot be explained by a single common factor. In contrast, the factor loadings for nonshared environmental factors (E_1 through E_4), which include measurement error, show a more measure-specific (diagonal) pattern. That is, factors that contribute to the variability in ADHD, CD, ODD, and EF but that are unique to the individual (thus do not increase twin resemblance) are not making a strong contribution to the phenotypic overlap among the measures. The overall fit of the model was marginal, $\chi^2(52) = 74.67$, $p = .02$; however, much of the lack of fit was due to variance differences between the MZ and DZ groups.

Table 4 summarizes the contribution of genetic factors to the covariation among the measures. Heritabilities are presented on the main diagonal, ranging

TABLE 3
Standardized Factor Loadings From the Cholesky Decomposition

Scale	A_1	A_2	A_3	A_4	E_1	E_2	E_3	E_4
ADHD	.91 ^a				.42 ^a			
CD	.48 ^a	.72 ^a			.16 ^a	.49 ^a		
ODD	.66 ^a	.25 ^a	.34 ^a		.20 ^a	.16 ^a	.57 ^a	
EF	.72 ^a	-.02	.12	.49 ^a	.33 ^a	-.01	-.03	.35 ^a

Note. A = additive genetic factors; E = nonshared environmental factors; ADHD = attention deficit hyperactivity disorder; CD = conduct disorder; ODD = oppositional defiant disorder; EF = executive functions.

^aSignificantly greater than zero based on 95% confidence intervals.

TABLE 4
Phenotypic Correlations^a, Heritabilities^b, and Proportions of Phenotypic Correlations Due to Genetic Factors^c

Scale	ADHD	CD	ODD	EF
ADHD	.82	.50	.68	.79
CD	.87	.74	.60	.37
ODD	.87	.82	.61	.56
EF	.83	.88	.92	.77

Note. ADHD = attention deficit hyperactivity disorder; CD = conduct disorder; ODD = oppositional defiant disorder; EF = executive functions.

^aAbove main diagonal. ^bOn main diagonal. ^cBelow main diagonal.

from .61 (ODD) to .82 (ADHD), differing only slightly from the univariate analyses. Above the main diagonal, the phenotypic correlations are presented for each comparison. They range from .37 (CD-EF) to .79 (ADHD-EF). The observed proportion of phenotypic correlations due to genetic factors is presented below the main diagonal. They ranged from .82 (CD-ODD) to .92 (ODD-EF) suggesting that, to the extent that these behavioral and cognitive patterns are comorbid, the overlap is largely driven by genetic factors.

DISCUSSION

The goal of this study was to examine the genetic and environmental architecture of parent-reported ADHD along with two commonly associated psychiatric syndromes (CD and ODD), as well as the etiology of EF deficits, which may be cognitive markers of these disorders. For each of these traits, all within-pair correlations were at least twice as great for the MZ twin pairs as for the DZ twin pairs, strongly supporting a hypothesis of a genetic etiology. Results of the structural equation modeling showed estimates of heritability ranging from .61 (ODD) to .82 (ADHD). Although there is a large twin literature examining the etiology of general cognitive abilities, ours is among the first of twin studies to specifically address executive function deficits. Our data suggest that these cognitive factors also are largely due to genetic factors. These findings are well in line with larger twin studies in the literature that demonstrated substantial genetic influences on externalizing disorders (Eaves et al., 1997; Sherman et al., 1997; Slutske et al., 1997). Also consistent with previous research is our finding that there was no evidence of shared environmental influences on ADHD, CD, ODD, or EF. That is, family, school, and peer influences that increase twin resemblance do not appear to contribute to the variability in these behavioral characteristics.

The multivariate cross-trait analyses showed that the comorbidity of ADHD with CD, ODD, and EF deficits is largely attributable to genetic influences. In the analyses, we decomposed the variances and covariances among the measures into four additive genetic and four nonshared environmental components. If there were strong genetic influences that were unique to each measure but were not contributing to the phenotypic overlap in the measures, we would expect large factor loadings on the diagonal (see the left panel of Table 3) and near-zero loadings on the off-diagonal. This was clearly not the case in our results, which suggests that comorbidity is driven, in large part, by heritable factors. From these results, the proportion of phenotypic correlation due to common genetic factors was computed. Our findings suggest that, although the phenotypic overlap in these syndromes ranges from moderate (CD-EF) to substantial (ADHD-EF), the correlations are due almost entirely to genetic influences. We could conclude that there is a common genetic vulnerability underlying a number of childhood behavioral and cognitive problems. It also is worth noting that

our data showed no evidence that shared environmental factors contribute to the comorbidity among these behavioral characteristics.

The genetic link between ADHD and CD replicates findings by Silberg et al. (1996) who showed that conduct problems and hyperactivity share an underlying biological risk. Because of the substantial shared genetic liability between ADHD and EF and between ADHD and ODD, we might speculate that ODD may represent an emotional manifestation of a core cognitive deficit in ADHD children. The finding of the strong comorbidity of ADHD with EF deficits is also consistent with previous studies (e.g., Pennington & Ozonoff, 1996) that argued that EF deficits may be a core or definitive problem in ADHD. It is possible, however, that this finding in our study could be an artifact of the overlap in the criteria for ADHD and the general definition of EF deficits. The *DSM-IV* lists "trouble organizing tasks" as a criterion of the inattention subtype of ADHD, whereas the same criterion often appears in definitions of EF deficits. However, this criterion is the sole overlapping item on the 18-item ADHD scale and the 8-item EF deficits scale of the CPNI. Although there are no published reports of the common genetic etiology of ADHD and EF, there is mounting evidence of a genetic relation between ADHD and other cognitive markers, such as reading disabilities (Gillis, Gilger, Pennington, & DeFries, 1992; Light, Pennington, Gilger, & DeFries, 1995).

Another interesting finding in this study is the greater comorbidity between ADHD and ODD and ADHD and EF deficits than between ADHD and CD. The extent to which this comorbidity is due to genetic factors is similar across comparisons. However, our results suggest that although there is substantial overlap in the genetic liability for ADHD and the other disorders, there also may be measure-specific heritable mechanisms important to the expression of the individual disorders.

This study is limited by the relatively small number of twins (compared to adult twin studies) and the use of nonreferred, community-based samples. It should be noted, however, that the latter point also may be viewed as a strength because it has been noted that, in clinical samples, there is often an entanglement between the correlates of assistance-seeking behavior and the correlates associated with psychopathological disorders themselves (Szatmari, Offord, & Boyle, 1989). The findings in this article also may have been influenced by the use of only one rater (mothers), because there is some evidence (e.g., Sherman et al., 1997) that heritability estimates may be considerably higher for mothers' reports compared to teachers' ratings. In addition, the nature of the differences between mothers' ratings and teachers' ratings has yet to be determined, particularly with respect to the accuracy or validity of these respective sources. This study may have been strengthened by the use of teachers' ratings or self-ratings and the ratings by the childrens' fathers, stepfathers, or significant other mens' ratings. In future research, it may be useful if ADHD also is investigated with respect to its two main subtypes (inattention and hyperactivity-impulsivity). As the sample size

increases, models that examine hypotheses about ADHD and its subtypes, possible gender differences in the etiology of these traits and their overlap, and possible sibling interactions (Silberg et al., 1996) also can be tested.

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Information about the Coolidge Personality and Neuropsychological Inventory (CPNI) for Children may be obtained from Frederick L. Coolidge, Department of Psychology, University of Colorado at Colorado Springs, P. O. Box 7150, Colorado Springs, CO 80933. The CPNI is available, pro bono, for research.

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