Genetic and Environmental Influences on the Relationship Between Aggression and Hyperactivity-impulsivity as Rated by Teachers and Parents

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This study examined genetic and environmental contributions to the covariance between aggression and hyperactivity-impulsivity as rated by twins' teachers and parents. Sex-differences in these genetic and environmental contributions and rater bias/sibling interaction effects were of interest as well. Part of an ongoing nation-wide twin-family study of behavioral development and health habits, the sample consisted of 1636 Finnish twin pairs ascertained from five consecutive and complete twin birth cohorts. Data were collected at ages 11–12, using teacher and parental rating forms of the Multidimensional Peer Nomination Inventory. Bivariate analyses were performed using structural equation modeling allowing sex-limitation effects. Results show that, in addition to significant genetic and environmental influences specific to each behavior, aggression and hyperactivity-impulsivity share common genetic and environmental etiology. Results provide evidence that both genetic and environmental factors are important in creating the observed correlation between aggression and hyperactivity-impulsivity.

Externalizing problems in children's behavior comprise several kinds of behaviors, including aggression, hyperactivity and impulsivity. An association between these behaviors has been demonstrated in both clinical and epidemiological samples, as reviewed by Biederman et al. (1991) and Jensen et al. (1997), and the association yields a factor of behavioral or externalizing problems. Using an epidemiological sample of adolescent twins, the present study examined the extent to which covariation between aggression and hyperactivity-impulsivity is attributed to shared genes and environmental factors, and whether the pattern of these genetic and environmental effects differs between boys and girls.

We previously reported significant genetic, common environmental and unique environmental effects on teacher- and parent-rated aggression at ages 11 to 12 years (Vierikko et al., 2003); the sample for that report included 1651 Finnish twin pairs drawn from the population-based FinnTwin12 study. Estimates of genetic and environmental effects differed, depending on the twin's sex and whether the informant was a teacher or parent. In both teacher and parental ratings, boys showed lower heritabilities (.14–.27) and higher estimates of common environment (.66–.75) than did girls (.54–.62 and .25–.37, respectively). Further, the teacher rating data suggested the presence of either sex-specific common environmental effects or sex-specific additive genetic effects. Finally, results for both teacher- and parent-rated aggression among boys suggested a significant sibling contrast effect, either at the behavioral level (reflecting reciprocal interactions of twin brothers) or as rater bias (such that the rated behavior of one twin brother affects the rated behavior of the other).

Children's aggression was measured using teacher and parental rating forms of the Multidimensional Peer Nomination Inventory (MPNI; Pulkkinen, 1987; Pulkkinen et al., 1999), in which the subscales for aggression and hyperactivity-impulsivity correlated and comprised part of the main factor for externalizing problem behavior (Pulkkinen et al., 1999). Because research has repeatedly demonstrated moderate to high heritability and low to moderate environmental effects on aggression (Chodsian-Carpey & Baker, 1987; Edelbrock et al., 1995; Hudziak et al., 2000; O'Connor et al., 1980; Schmitz et al., 1995; Van den

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Oord et al., 1996), as well as on hyperactivity (Gjone et al., 1996; Goodman & Stevenson, 1989; Kuntsi & Stevenson, 2001; Nadder et al., 1998; Silberg et al., 1996; Stevenson, 1992; Thapar et al., 1995), and because a significant observed correlation exists between these behaviors, the present study asked to what extent the correlation was due to genetic and environmental effects common to both traits.

Genetic and environmental correlations between different behaviors can be estimated by bivariate structural modeling of twin data (Neale & Cardon, 1992). Bivariate models estimate the genetic and environmental influences common to two behaviors, and the genetic and environmental influences specific to each behavior. A strong genetic correlation has been found both in categorical measures of attention deficit hyperactivity disorder (ADHD) and opposi-tional-defiant disorder/conduct disorder (ODD/CD) (Nadder et al., 2002; Nadder et al., 1998; Silberg et al., 1996; Young et al., 2000), and in dimensional measures of aggression and attentional problems, which include items for hyperactivity and impulsivity (Schmitz & Mrazek, 2001). A genetic correlation of .17 was found between dimensional measures of symptoms of ADHD and CD obtained from interviews of 11-year-old twins and their mothers (Burt et al., 2001). The correlations of unique environmental factors, which affect only one twin of a pair, have been somewhat lower than genetic correlations. In addition, although the magnitude of common environmental effects was low, the two latter studies showed a common environmental correlation of 1.00 and .99, respectively, indicating that almost entirely the same family environmental factors contribute to these behaviors.

Studies considering the extent and magnitude of gender differences in genetic and environmental influences on behavioral problems have yielded inconclusive results. Different etiologies for aggression have been found for boys and girls in both childhood and adolescence (Vierikko et al., 2003) as well as in categorical ADHD (Eaves et al., 2000; Nadder et al., 2001; Steffensson et al., 1999; Rhee et al., 1999). In contrast, a similar pattern of genetic and environmental effects on boys and girls has been reported for children's and adolescents' maternal rated aggression (Eley et al., 1999) and parent- and teacher-rated ADHD (Nadder et al., 1998; Thapar et al., 2000). Mixed results have been found concerning the causes of covariation between hyperactivity and conduct problems, as well. In analyses of symptom counts obtained from age 14 interview data of the same Finnish twins studied in this report, the covariation of three childhood externalizing disorders was largely attributed to common genes (Dick et al, 2003). And the covariation between hyperactivity and conduct problems has been almost entirely accounted for by the same genes in boys and girls at age 8 to 11 (Burt et al., 2001; Silberg et al., 1996), although at ages 12 to 16, different sets of genes have contributed to the covariation between hyperactivity and conduct problems in boys and girls (Silberg et al., 1996). Recent studies have provided some evidence of developmental change in genetic effects underlying ADHD (Nadder et al., 2002) and antisocial behaviors (O'Connor et al., 1998), which in turn may create variability in the estimates of genetic and environmental effects across studies using samples differing in the age of the subjects.

There is consistent evidence of significant and substantial genetic effects on aggression and attentional problems in children and adolescents, but estimates of genetic effects vary considerably, and especially so for aggression. One reason for that variation may be sibling effects, attributable either to systematic bias in the behavioral ratings of aggression, or, alternatively, to reciprocal interactions between the twin siblings. A positive sibling interaction arises from sibling imitation or cooperation, while a negative interaction arises from sibling competition. And alternatively, rater bias, rather than sibling interaction, can create parallel effects: the rated behavior of one twin could increase (or decrease) the rated behavior of the co-twin. In earlier studies, we have found negative sibling effects in both teacher and parental ratings of aggression. A negative sibling effect is a common finding in maternal ratings of hyperactivity (Eaves et al., 2000; Kuntsi & Stevenson, 2001; Nadder et al., 1998; Silberg et al., 1996; Simonoff et al., 1998; Thapar et al., 1995), and often it is interpreted as a bias in maternal ratings of hyperactivity, rather than a consequence of behavioral interaction between the co-twins. That interpretation of sibling effects raises questions about the validity of maternal ratings and suggests the utility of multiple informants.

The primary aim of this study was to investigate to what extent genetic and environmental influences on aggression and hyperactivity-impulsivity are correlated. We expected to find high heritability for hyperactivity-impulsivity, similar to effects we found for aggression, and we expected to observe a high genetic correlation between these behaviors (Nadder et al., 2002; Nadder et al., 1998; Schmitz & Mrazek, 2001; Silberg et al., 1996; Young et al., 2000). Both sources of environmental effects were expected to be of modest or moderate magnitude, but the common environmental correlation was hypothesized to be close to unity (Burt et al, 2001; Schmitz & Mrazek, 2001), and the correlation from unique environments moderate at most (Nadder et al., 2002; Nadder et al., 1998; Schmitz & Mrazek, 2001; Silberg et al., 1996; Young et al., 2000).

A secondary purpose of this study was to examine whether the same genetic and environmental factors influence aggression, hyperactivity-impulsivity, and their correlation in boys and girls, and whether the magnitude of these influences is similar for each sex. Because significant sex effects were found in aggression

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(Vierikko et al., 2003), sex-differences were expected also in genetic and environmental correlations between aggression and hyperactivity-impulsivity. The third and final aim of our analyses was to examine the significance of sibling effects in teacher and parental ratings of hyperactivity-impulsivity, because earlier research has supported the hypothesis of negative sibling effects for hyperactivity (Kuntsi & Stevenson, 2001; Nadder et al., 1998; Silberg et al., 1996; Simonoff et al., 1998; Thapar et al., 1995).

Materials and Methods

Participants

This study is part of a longitudinal population-based twin-family study FinnTwin12 (FT12), of the behavioral development and health habits of five consecutive Finnish twin cohorts born in 1983–1987, studied from baseline age 11–12 (Kaprio et al., 2002; Rose et al., 2001). A subsample of FT12 was included in our earlier report on sex differences in genetic and environmental effects on teacher- and parent-rated aggression (Vierikko et al., 2003), and that sample is the basis for analyses reported here. Initially, teacher ratings were sent to 2677 teachers, and 2488 (93%) completed ratings. Parental rating questionnaires were sent to 2695 families, and 2470 (92%) completed questionnaires were returned. In total, either teacher or parental ratings were obtained from 2614 twin pairs. For the current analyses, the sample was restricted to those twin pairs for whom items measuring aggression and hyperactivity-impulsivity were obtained from both teacher and parental ratings (N = 1993 twin pairs). In parental rating data, mother (58%), father (3%), both parents jointly (38%), or several persons together (1%) rated the twins' behavior. Co-twins in 87% of these twin pairs were in the same class and, therefore, rated by the same teacher. Twin pairs in which co-twins were in different classes and rated by different teachers (N = 251 pairs) were analyzed in separate analyses, and those analyses appear in the Results section.

Zygosity of the same-sex twin pairs was determined from their perceived similarity and confusability of appearance as separately reported by the twins and their parents in postal questionnaires completed late in the year each twin cohort reached age 11. Further information for determination of zygosity was based on school photographs, parental interviews, and placental information supplied by parents. In a few cases, zygosity could not be assigned, because of missing or ambiguous information, and until their zygosity is confirmed with DNA samples, these pairs (N = 106) have been excluded from analyses. The final sample for analyses here reported consisted of 1636 twin pairs: 556 monozygotic (260 male, MMZ, and 296 female, FMZ), 567 same-sex dizygotic (290 male, MDZ, and 277 female, FDZ) and 513 opposite-sex dizygotic (OSDZ).

Measures and Variables

Children's social behavior was assessed with the teacher and parental rating form of the Multidimensional Peer Nomination Inventory (MPNI; Pulkkinen et al., 1999). The 38-item teacher and parent rating forms of MPNI were presented in the form of statements (e.g., "Is able to sort things out by talking"). The items were rated on a 4-point-Likert-type scale: 0 = *does not apply*, 1 = *applies sometimes, but not consistently*, 2 = *certainly applies, but not in a pronounced way*, 3 = *applies in a pronounced way*. The scale for aggression consisted of six items: "tries to leave other kids out of the company of others (e.g., by saying, 'Let's not play/hang around with him/her'"; "teases other kids or attack them for no reason at all"; "calls people names when … angry with them"; "may hurt other kids when … angry (e.g., by hitting, kicking, or throwing things at them)"; "bullies smaller and weaker kids"; and "goes around telling people's secrets to others". The coefficient alpha for aggression from teacher ratings was .87 for boys and .85 for girls and from parental ratings .69 for boys and .66 for girls.

The hyperactivity-impulsivity scale consisted of seven items: "is restless, unable to sit in class"; "is hyperactive"; "is talkative"; "has difficulties in waiting for their turn"; "is disobedient at school/home"; "often acts rashly (i.e., without thinking about the possible consequences)"; and "runs about and climbs everywhere in spite of warnings". The coefficient alpha for hyperactivity-impulsivity from teacher ratings was .92 for boys and .89 for girls, and from parental ratings .80 and .75, respectively.

Data Analyses

SPSS 10.0 for Windows (SPSS Inc., 2000) and STATA (Stata Corp., 1999) were used for phenotypic analysis and twin correlations. Mean scores for aggression and hyperactivity-impulsivity, separately for teacher and parental ratings, were computed for each twin. As the distributions of both behavioral scales were highly skewed, the data were log (x + 1) transformed prior to the analysis in order to reduce non-normality.

To explore the sources of variation and covariation of aggression and hyperactivity-impulsivity, structural equation modeling was performed using the statistical package Mx (Neale et al., 1999). Model fitting employed variance-covariance matrices and maximum-likelihood methods of estimation. The variance-covariance matrices and Pearson correlations between co-twins (twin 1 and twin 2) were computed separately for the five groups of twins. Co-twins from same-sex pairs were randomly assigned as twin 1 and twin 2. For the OSDZ twins, boys were coded as twin 1 and girls as twin 2.

Both the univariate and bivariate models partitioned the total variance into additive genetic variance (A), non-additive genetic variance (D), common environmental variance (C), and unique environmental

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variance (E) using the standard approach to twin data (Neale & Cardon, 1992) based on the differing degree of genetic similarity of MZ and DZ pairs. In addition to the basic twin model, two paths were added between the co-twins to reflect sibling effects(s). Sibling interactions affect both variances and covariances between siblings. Positive sibling effects increase both MZ and DZ correlations, with DZ correlations increased to a greater extent. In contrast, negative sibling interaction decreases the twin correlations, affecting the DZ correlation more that the MZ correlation. Negative sibling effects also increase phenotypic variances for both MZ and DZ twins, but again, with a greater effect on the DZ twins. In practice, on the basis of twin correlations alone, positive sibling effects are difficult to distinguish from common environmental effects because both increase DZ twin correlations. Further, negative sibling effects may be confused with a non-additive genetic effect, because both decrease DZ twin correlations. Simultaneous analyses of differences in MZ and DZ variances and covariances permit estimates of sibling effects; sibling effects cause differences in MZ and DZ variances, but A, C, D and E effects do not.

Univariate Analyses

Because we had data on OSDZ twins, the univariate modeling started with a full general sex-limitation model (Neale & Cardon, 1992), which enables the assessment of sex differences in genetic and environmental effects by allowing different paths for boys and girls. The significance of sex-specific genetic or environmental effects can be estimated by allowing either an additional genetic or common environmental variable for either boys or girls (A'/C'). If the estimates of these sex-specific effects are significant, the set of genes or environments that influences a trait is not identical between the sexes.

Based on the twin correlations, the full sex-limitation model included the A, C, E, and a sex-specific A or C variation and allowed their paths to differ for boys and for girls. Because, in parent-rated hyperactivity-impulsivity, the low twin correlation for DZ girls, relative to that for MZ girls, suggested significant dominant genetic effects, or possibly sibling effects, the significance of the dominance effects and sibling interaction was tested for girls. The effects of genetic dominance and common environment were estimated for girls in separate models (ACE- or ADE-model), since these effects are confounded in data with twins reared together. The sibling effect parameter was added in equal magnitude for MZ and DZ girls.

To obtain the most parsimonious model with fewer parameters, a series of reduced models was fitted to the data and compared to the full (general sex-limitation) model using the Chi-square (χ^2) difference test. The reduced models tested whether the parameters of the full model could be dropped or constrained to be equal for boys and girls. The reduced model was rejected whenever the χ^2 difference exceeded the critical value of χ^2 determined by the difference in the number of degrees of freedom (*df*) between the full and modified model. The model fit was also assessed by the Akaike's Information Criterion (AIC = $\chi^2 - 2df$), in which larger negative values indicate better fit (Akaike, 1987). Chi-square values with associated *p*-values greater than .05 indicate a good fit, though it should be noted that with large sample sizes significant χ^2 values can be the result of rather trivial failures of the model.

Bivariate Analyses

The relative importance of genetic and environmental factors in the covariance between aggression and hyperactivity-impulsivity was initially examined by comparing cross-twin cross-trait correlations (e.g., aggression of the first twin with hyperactivity-impulsivity of the second twin). As in the univariate case, additive genetic effects are indicated by a DZ cross-twin, cross-trait correlation less than MZ cross-twin, cross-trait correlation, and non-additive genetic effects by a DZ cross-twin, cross-trait correlation less than half that found for MZs. Common environmental effects are indicated by a DZ correlation greater than half the MZ correlation.

Bivariate model fitting using a Cholesky decomposition model (Neale & Cardon, 1992) was applied to examine to what extent shared genetic and environmental factors influence aggression and hyperactivity-impulsivity and to explain the correlation between aggression and hyperactivity-impulsivity (Figure 1). The significance of the sibling effect parameter was tested by adding two reciprocal paths between the co-twins, separately for aggression and hyperactivity-impulsivity (for simplicity not shown in Figure 1).

When Cholesky decomposition models are used for the analyses of sex-limitation effects on data using same-sex and opposite-sex twin pairs, the order in which the variables enter into the analysis can affect the fit of the models (Neale, 2002). We examined this possibility in our data by testing the effect of altering the order of the variables. In the data from teacher ratings, the order of the variables in the analysis affected neither the fit of the model nor the parameter estimates. In parental ratings, in contrast, the estimate of genetic effects increased for the variable entered first in the model, while that for common environmental effects decreased. Although the goodness of fit and the parameter estimates slightly changed, the interpretation of results remained unchanged. And it was apparent that the ordering effects were due to OSDZ twins, because excluding them from the model fitting eliminated any effect from the order in which variables entered into the analysis. But ordering effects were modest and did not alter inferences made, so we chose to retain the full sample to make results from teacher and parental ratings comparable.

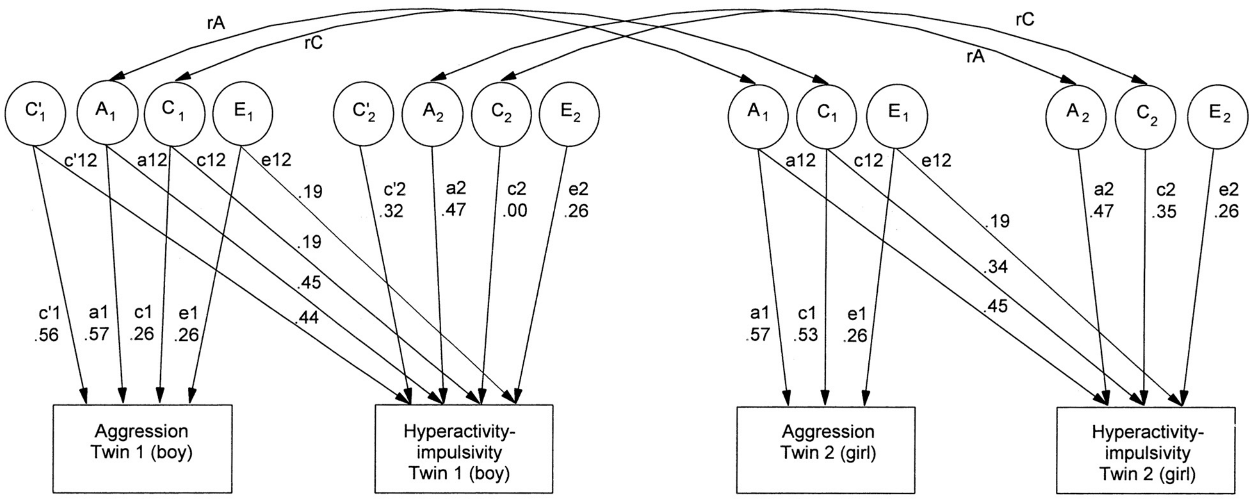

Figure 1

Bivariate Cholesky decomposition model for teacher-rated aggression and hyperactivity-impulsivity for opposite-sex DZ twins and model fitting results.

Note: $\chi^2(36) = 28.173$, p = .821, AIC = -43.827; parameter estimated to be .00 is fixed to zero; A = additive genetic variance; C = common environment; E = unique environment; A′/C′ = sex-specific genetic or common environmental variance; A_1, C_1, E_1, A'/C'_1 = genetic and environmental factors shared by aggression and hyperactivity-impulsivity; A_2, C_2, E_2, A'/C'_2 = genetic and environmental factors unique to hyperactivity-impulsivity; a_1, c_1, e_1, and a'/c'_1 = paths for A, C, E, and A′/C′ in aggression. ; a_2, c_2, e_2, and a'/c'_2 = paths for A, C, E, and A′/C′ in hyperactivity-impulsivity; a_{12}, c_{12}, e_{12}, and a'/c'_{12} = paths for A, C, E, and A′/C′ shared by aggression and hyperactivity-impulsivity. A is correlated (rA) 1.0 for MZ, 0.5 for DZ twins; C is correlated (rC) 1.0 for both MZ and DZ twins; A′/C′ is fitted, and thus correlated only for the same-sex male twin pairs (thus not depicted here) 1.0 for MZ, 0.5/1.0 for DZ twins.

Accordingly, the OSDZ pairs are included in the analysis of both ratings.

To obtain the most appropriate explanation of the data, the bivariate model fitting began based on univariate results. Modeling proceeded by testing the significance of each factor by removing the corresponding path from the model and testing whether the paths can be set equal for boys and girls. The adequacy of different reduced models was evaluated comparing the change in χ^2 and using AIC.

Results

Descriptive Statistics

Intercorrelations between aggression and hyperactivity-impulsivity were substantial and significant at the $p < .01$ level for both sexes and in both teacher and parental ratings; Pearson correlation coefficients were .72 for boys and .52 for girls in teacher ratings, and .70 and .44 in parental ratings. Means, variance-covariance matrices, and twin correlations by gender and zygosity are shown in Table 1 for aggression and in Table 2 for hyperactivity-impulsivity. The descriptive statistics for aggression were obtained from data ($N = 1651$) used in univariate analysis of aggression presented in Vierikko et al. (2003). The means, standard deviations, covariances and twin correlations did not differ for the present sample ($N = 1636$), except for the differences due to rounding error. For aggression, adjusted Wald tests indicated that boys' scores were significantly higher than girls' scores: $F(1650) = 88.12$, $p < .01$ on teacher ratings, $F(1650) = 28.71$, $p < .01$ on parental ratings. Significant mean differences were also found between boys and girls for hyperactivity-impulsivity. The mean of hyperactivity-impulsivity for boys was higher than that for girls on teacher ratings, $F(1, 1635) = 269.78$, $p < .001$, and parental ratings $F(1, 1635) = 61.64$, $p < .001$. No mean differences by zygosity were observed. For aggression for boys, DZ variance was larger than MZ variance on teacher ratings, $F(527, 1100) = .82$, $p = .01$, and on parental ratings, $F(527, 1100) = .76$, $p < .01$. In addition, for hyperactivity-impulsivity, variance was greater for boys than for girls on teacher ratings, $F(1612, 1658) = 1.129$, $p < .05$, and variance for DZ twin brothers was greater than for MZ twin brothers on parental ratings, $F(519, 1092) = .84$, $p < .05$).

Twin Correlations

The pattern of twin correlations (Table 1) in teacher ratings of hyperactivity-impulsivity indicated substantial genetic effects, and evidence of common environmental effects was present in teacher ratings as well. For parent-rated hyperactivity-impulsivity, correlations for MZ twins are considerably larger than those for DZ twins consistent with large genetic influences both for boys and for girls, and low or negligible common environmental influences for boys and nonexistent for girls. In particular, the DZ correlation for girls in parent-rated hyperactivity-impulsivity was much lower than half of MZ correlation, suggesting either genetic effects due to dominance or sibling interaction in addition to additive genetic effects.

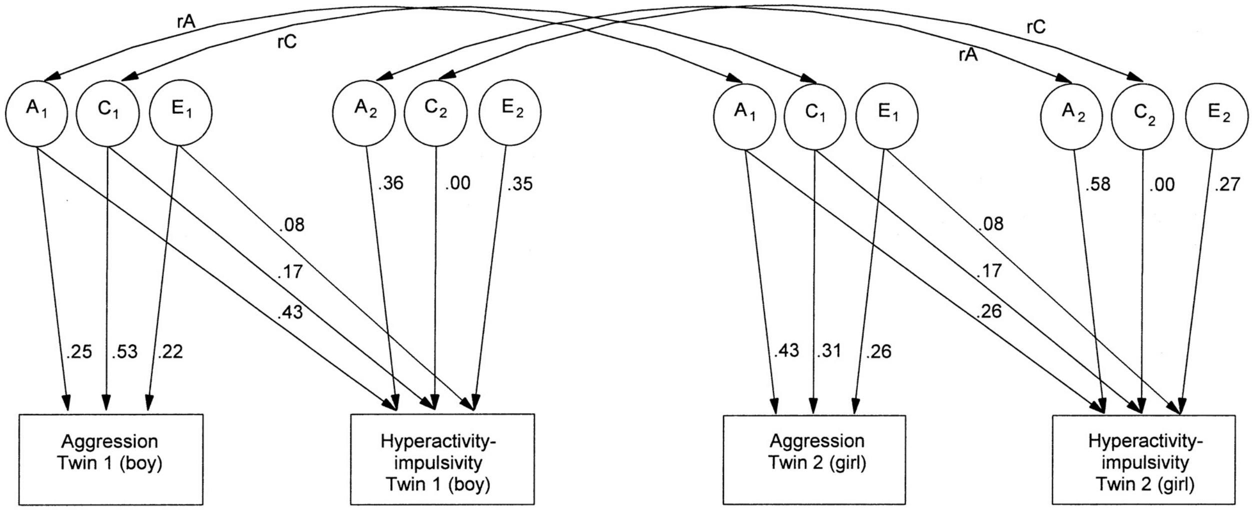

Figure 2

Bivariate Cholesky decomposition model for parent-rated aggression and hyperactivity-impulsivity for opposite-sex DZ twins and model fitting results.

Note: $\chi^2(34) = 45.907$, $p = .083$, AIC = -22.093; A = additive genetic variance; C = common environment; E = unique environment; D = dominant genetic variance; A_1, C_1, E_1, D_1 = genetic and environmental factors shared by aggression and hyperactivity-impulsivity; A_2, C_2, E_2, D_2 = genetic and environmental factors unique to hyperactivity-impulsivity. A is correlated (rA) 1.0 for MZ, 0.5 for DZ twins; C is correlated (rC) 1.0 for both MZ and DZ twins.

Possible sex-specific effects were suggested in teacher ratings both in aggression and hyperactivity-impulsivity by lower OSDZ correlations (.43 in aggression; .37 in hyperactivity-impulsivity) compared to those for same-sex DZ twins (.72 for boys and .64 for girls in aggression; .61 for boys and .60 for girls in hyperactivity-impulsivity). The larger difference between MZ and DZ correlations in teacher- and parent-rated aggression in girls (difference = .27 and .25, respectively; correlations for girls in teacher ratings: MZ .91, DZ .64; in parental ratings: MZ .78, DZ .53) compared to that for boys (difference = .17 and .13, respectively; in teacher ratings: MZ .89, DZ .72; in parental ratings: MZ .72, DZ .59) suggests greater contribution from genetic effects for girls.

Table 3 provides descriptive data for the sample of twins rated by the same teacher versus the smaller sample rated by different teachers. There were no significant differences in either means or variances between the two samples, but the twin correlations were lower for twins rated by different teachers. But it is of interest that the pattern of twin correlations suggests significant genetic and environmental effects, and sex-specific variance for both samples. The twin pairs rated by different teachers were excluded from the analysis, because of small sample sizes once twin pairs were divided by sex and zygosity.

Cross-twin Cross-trait Correlations

The cross-twin cross-trait correlations (e.g., aggression of the twin 1 with hyperactivity-impulsivity of the twin 2) are shown in Table 4 for teacher and parental ratings. The pattern of these correlations in teacher ratings for boys and girls and in parental ratings for boys suggests additive genetic ($r_{mz} > r_{dz}$) and common environmental influences ($r_{dz} > \frac{1}{2}r_{mz}$) on the association between aggression and hyperactivity-impulsivity. In parental ratings for girls, the DZ correlations were less than one-half of the MZ correlations suggesting additive and/or dominant genetic effects, but no common environmental effects.

Cross-twin Cross-rater Correlations

The phenotypic correlations between the ratings (cross-rater within-twin correlations) were significant at the $p < .01$ level: .28 for boys and .17 for girls in aggression and .35 for boys and .28 for girls in hyperactivity-impulsivity. The cross-twin cross-rater correlations (e.g., correlation between twin 1 rated by teacher and twin 2 rated by parent) were further computed to assess the extent to which teacher and parental ratings share the same genetic and environmental factors (Table 4). The pattern of these correlations indicated significant genetic effects ($r_{mz} > r_{dz}$) and negligible common environmental effects ($r_{dz} < \frac{1}{2}r_{mz}$) in correlations between teacher and parental ratings for both sexes.

Univariate Results

Teacher- and Parent-rated Aggression

The univariate genetic analyses of aggression and hyperactivity-impulsivity were carried out separately for teacher and parental ratings, and are reported for aggression in detail in Vierikko et al. (2003). To summarize, for teacher-rated aggression, the general sex-limitation model allowing either a sex-specific A or C, and a sibling parameter for MZ and DZ twin brothers (in equal magnitude), fit the data very well. Further, either the E effect, or alternatively, both E

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Table 1

Means, Variance-covariance Matrices, and Twin Correlations for Teacher and Parental Ratings on Aggression by Sex and Zygosity Group

	N pairs	Teacher Ratings Mean *(SD)*	Variance-covariance Matrix (lower diagonal) and Correlation (upper corner)		Parental Ratings Mean *(SD)*	Variance-covariance Matrix (lower diagonal) and Correlation (upper corner)	
MMZ	264	1.44 (0.79)	0.67	0.89	1.46 (0.49)	0.27	0.72
			0.59	0.65		0.20	0.29
MDZ	292	1.35 (0.81)	0.76	0.72	1.45 (0.49)	0.29	0.59
			0.55	0.78		0.18	0.33
FMZ	300	1.01 (0.79)	0.64	0.91	1.26 (0.54)	0.31	0.78
			0.59	0.67		0.25	0.33
FDZ	278	1.07 (0.74)	0.64	0.64	1.34 (0.52)	0.35	0.53
			0.43	0.72		0.19	0.36
OSDZ	517	M 1.26 (0.92)	0.84	0.43	M 1.27 (0.64)	0.41	0.58
		F 1.00 (0.83)	0.33	0.69	F 1.23 (0.60)	0.22	0.36

Note: MMZ = monozygotic males; MDZ = dizygotic males; FMZ = monozygotic females; FDZ = dizygotic females; OSDZ = opposite-sex dizygotic twins; M = male, F = female.

Table 2

Means, Variance-covariance Matrices, and Twin Correlations for Teacher and Parental Ratings on Hyperactivity-Impulsivity by Sex and Zygosity Group

	N (twin pairs)	Teacher Ratings Mean *(SD)*	Variance-covariance Matrix (lower diagonal) and Correlation (upper corner)		Parental Ratings Mean *(SD)*	Variance-covariance Matrix (lower diagonal) and Correlation (upper corner)	
MMZ	260	1.67 (0.92)	0.84	0.87	1.74 (0.65)	0.40	0.73
			0.74	0.87		0.31	0.44
MDZ	290	1.64 (0.92)	0.84	0.61	1.68 (0.69)	0.47	0.37
			0.51	0.83		0.18	0.50
FMZ	296	0.99 (0.87)	0.74	0.87	1.50 (0.68)	0.45	0.76
			0.65	0.76		0.35	0.46
FDZ	277	1.06 (0.85)	0.71	0.60	1.50 (0.67)	0.41	0.10
			0.44	0.75		0.04	0.48
OSDZ	513	M 1.59 (0.94)	0.90	0.37	M 1.65 (0.71)	0.51	0.33
		F 1.03 (0.90)	0.32	0.81	F 1.44 (0.69)	0.17	0.49

Note: MMZ = monozygotic males; MDZ = dizygotic males; FMZ = monozygotic females; FDZ = dizygotic females; OSDZ = opposite-sex dizygotic twins; M = male, F = female.

and C effects, could be set equal for both sexes without a significant decrease in fit, and both of these models provided an adequate explanation of the teacher rating data. For parent-rated aggression, testing a general sex-limitation model indicated that sex-specific A and C effects were small, and could therefore be dropped from the model. Parameter estimates (standardized to reflect the percentage of phenotypic variance accounted for) and 95% confidence intervals (CI) for the best fitting models for teacher and parental ratings are presented in Table 5. Overall, in both ratings, boys showed higher levels of common environment and lower heritabilities than did girls. The findings also provided support for negative sibling effects for twin brothers, both in teacher and parental ratings, indicating contrast effect in the ratings, or competition effects between the co-twins.

Teacher-rated Hyperactivity-impulsivity

For teacher-rated hyperactivity-impulsivity, the full general sex-limitation model, which allowed a sex-specific A or C effect and a sibling effect of equal magnitude for all zygosity groups, fit the data well. However, a model, which allowed a sex-specific C effect ($\chi^2_7 = 1.880$, $p = .966$, AIC = –12.120), provided a slightly better fit on the basis of lower AIC than a model allowing a sex-specific A ($\chi^2_7 = 1.937$, $p = .963$, AIC = –12.063). The sibling effect parameter was nonsignificant and it could be dropped from the model without significant decrease in fit ($\Delta\chi^2_1 = .966$, $p > .05$). Although the confidence interval of the C effect for boys was .00 - .09 indicating that the effect may be nonsignificant, fixing that path to zero resulted in significant worsening of fit ($\Delta\chi^2_1 = 4.602$, $p < .05$). Thus, we retained the C effect for both sexes in the model, but the A and E effects could be constrained to be equal for boys and girls ($\Delta\chi^2_2 = 2.543$, $p > .05$).

The standardized estimates of genetic and env0ironmental effects for teacher-rated hyperactivity-impulsivity were rather similar for both sexes,

Table 3

Descriptive Statistics and Twin Correlations for the Same versus Different Teacher Ratings

	Same Teacher				Different Teachers			
	N Pairs	Mean	*SD*	*r*	*N* Pairs	Mean	*SD*	*r*
Aggression								
MZ	556	2.43	1.64	0.91	63	2.53	1.72	0.59
SSDZ	568	2.42	1.58	0.69	79	2.54	1.49	0.36
OSDZ	513	2.25	1.48	0.43	88	2.67	1.34	0.23
Hyperactivity-Impulsivity								
MZ	556	2.62	1.85	0.87	63	2.99	1.94	0.70
SSDZ	568	2.72	1.69	0.64	79	2.90	1.74	0.36
OSDZ	513	2.62	1.53	0.37	88	2.92	1.43	0.21

Note: MZ = monozygotic twins; SSDZ = same-sex dizygotic twin pairs; OSDZ = opposite-sex dizygotic twin pairs.

Table 4

Cross-twin Cross-trait Correlations and Cross-twin Cross-rater Correlations

	Cross-twin Cross-trait Correlations		Cross-twin Cross-rater Correlations	
	Teacher Ratings	Parental Ratings	Aggression	Hyperactivity-Impulsivity
MMZ	0.63	0.47	0.31	0.29
MDZ	0.52	0.30	0.17	0.17
FMZ	0.62	0.34	0.19	0.29
FDZ	0.42	0.11	0.04	0.00
OSDZ	0.32	0.32	0.09	0.04

Note: MMZ = monozygotic males; MDZ = dizygotic males; FMZ = monozygotic females; FDZ = dizygotic females; OSDZ = opposite-sex dizygotic twins.

although the C effects for boys were divided into shared and sex-specific effects (Table 5). The genetic effects were about half of the variance and the common environmental effects approximately one third of the total variance. Residual variance consisted of the unique environmental variance and the error.

Parent-rated Hyperactivity-impulsivity

For parental ratings, the full general sex-limitation model, which allowed either a sex specific A or C effects and sibling interaction in equal magnitude for MZ and DZ girls, provided an acceptable fit to the data (max. $\chi^2_7 = 13.287$, p = .065, AIC = –.713). However, the sex specific A or C effects and the C effect shared by boys and girls were nonsignificant and dropping them from the model did not deteriorate the fit significantly ($\chi^2_{10} = 15.298$, $p = .122$, AIC = –4.702). In addition, the A effects could be set equal for boys and girls ($\Delta\chi^2_1 = 3.532$, $p > .05$). Because the pattern of twin correlations ($r_{dz} > ½r_{mz}$) for girls was suggestive of genetic effects due to dominance in addition to additive genetic effects, the significance of the D effect was tested for girls by adding dominant genetic effects for MZ and DZ girls in the AE-model above. That model provided a good fit to the data ($\chi^2_{10}= 15.896$, $p = .103$, AIC = -4.104), but did not increase the fit significantly. Thus, the univariate modeling of parental rating data resulted in AE-model for both sexes and sibling interaction for MZ and DZ girls.

The standardized estimates of genetic and unique environmental effects for parent-rated hyperactivity-impulsivity can be seen in Table 5. Approximately four-fifths of the total variance were attributable to additive genetic factors and one-fifth to unique environmental factors both in boys and girls. The sibling effect for girls was negative: –.11.

Bivariate Results

Teacher Ratings

Given evidence of significant sex differences in genetic and environmental effects, bivariate model fitting began by allowing the variance components for A, C, and E to differ for boys and girls. Based on the univariate results, the bivariate modeling for teacher rating data began by fitting a model, which allowed a sibling interaction parameter for MZ and DZ twin brothers (in equal magnitude) only for aggression and a sex-specific C component for both behaviors, added for boys in this case. This model provided a good fit to the data ($\chi^2_{28} = 16.794$, $p = .953$, AIC = 39.206). However, the sibling effect parameter was nonsignificant and dropping it from the model did not worsen the fit significantly ($\Delta\chi^2_1 = .825$, $p > .05$). Also the C effect for hyperactivity-impulsivity for boys (path c_2 in Figure 1) could be omitted from the model without significant loss of fit ($\Delta\chi^2_1 = .054$, $p > .05$). All other parameters were significant and could not be dropped from the model without significant reduction in fit. However, the A and the E effects could be constrained

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to be equal for boys and girls in both behaviors ($\Delta\chi^2_6$ = 1.500, p > .05).

The resultant model and its path coefficients are presented in Figure 1 for OSDZ twin pairs. Because boys in OSDZ pairs were coded as twin 1 and girls as twin 2, the left part of the figure shows the model fitting results for boys and the right part for girls. The magnitude of genetic and unique environmental factors on both behaviors was equal across sexes. Unequal paths for boys and girls in Figure 1 indicate sex-differences in magnitude of common environmental effects on aggression and hyperactivity-impulsivity. The importance of sex-specific C' indicates that partly different factors in shared environment affect boys and girls in both behaviors.

The total variance of each phenotype is the sum of all squared paths leading to that phenotype. The proportion of variance of each behavior due to genetic effects is the sum of all squared a paths leading from A to the phenotype. For example in Figure 1, the proportion of A effects for aggression in boys is $.57^2 : (.56^2 + .57^2 + .26^2 + .26^2) = .42$. The proportions of environmental variances can be calculated in a similar way. The relative proportions of A, C, and E effects for hyperactivity-impulsivity in both boys and girls are calculated by summing the paths from factor shared by aggression and hyperactivity-impulsivity and factor specific to hyperactivity-impulsivity. For example, the relative proportion of C effects for hyperactivity-impulsivity in boys is a sum of C effects shared by aggression and hyperactivity-impulsivity (path c_{12}) and C effects specific to hyperactivity-impulsivity (c_2). Although the path c_2 for hyperactivity-impulsivity in boys is fixed to zero, path c_{12} is estimated as 0.19. Accordingly, the proportion of C effects for hyperactivity-impulsivity in boys is $(.19^2 + .00^2)\colon (.44^2 + .45^2 + .19^2 + .19^2 + .32^2 + .47^2 + .00^2 + .26^2) = .04$.

Parameter estimates (standardized to reflect the percentage of phenotypic variance), 95% confidence intervals (CI), and the genetic and environmental correlations for the best fitting bivariate model for teacher ratings of aggression and hyperactivity-impulsivity are presented in Table 6. The parameter estimates for aggression and hyperactivity-impulsivity were rather similar for both sexes in the preferred bivariate model. The largest proportion of the total variance could be attributed fairly equally to genetic effects and common environmental effects, and rest of the variance, about 10%, to unique environment. The genetic and environmental correlations were high both for boys and for girls. For both sexes, half of the genetic effects, over half of the common environmental effects, and almost half of unique environmental effects influencing hyperactivity-impulsivity are the same factors, which are influencing aggression.

Parental Ratings

On the basis of the univariate results, bivariate model fitting for parental ratings started with ACE-model in aggression, and AE-model in hyperactivity-impulsivity. The variance components were allowed to differ across sexes. In addition, the model included sibling interaction for MZ and DZ boys in aggression and MZ and DZ girls in hyperactivity-impulsivity. Although that model was based on the univariate results, it provided a poor fit to the data (χ^2_{32} = 58.075, p = .003, AIC = -5.925). A model allowing A, C and E effects for both behaviors and for both sexes provided better fit (χ^2_{30} = 40.455, p = .096, AIC = -19.545). Common environmental effects specific to hyperactivity-impulsivity could be dropped from the model for both sexes (path c_2) without significant deterioration in model fit ($\Delta\chi^2_2$ = 2.478, p > .05), but the common environmental effects shared by aggression and hyperactivity-impulsivity (path c_{12}) were inevitable in order to have an acceptable model. Further, the A and E effects shared by aggression and hyperactivity-impulsivity (paths a_{12} and e_{12}) could be

Table 5

Standardized Parameter Estimates for Genetic and Environmental Effects on Teacher and Parental Ratings of Aggression and Hyperactivity-Impulsivity of the Preferred Univariate Models

	Males				Females							
	A	C	E	C'	A	C	E	s	χ^2	*df*	*p*	AIC
Aggression												
Teacher ratings	.27	.15	.07	.51	.54	.37	.09	–.09	3.832	8	.872	–12.168
E m = f	(.18–.39)	(.06–.27)	(.06–.09)	(.40–.60)	(.43–.68)	(.23–.48)	(.08–.11)	(–.17– –.02)				
Teacher ratings	.24	.23	.07	.46	.62	.29	.09	–.11	6.131	9	.727	–11.869
E,C m = f	(.17–.34)	(.16–.29)	(.06–.08)	(.37–.55)	(.53–.71)	(.20–.38)	(.08–.11)	(–.18– –.05)				
Parental ratings	.14	.75	.11	—	.54	.25	.21	–.25	4.509	8	.808	–11.491
	(.07–2.6)	(.60–.85)	(.07–.17)		(.46–.62)	(.18–.32)	(.17–.24)	(–.37– –.13)				
Hyperactivity-impulsivity												
Teacher ratings	.49	.03	.12	.36	.55	.32	.14	—	5.389	10	.864	–14.611
A, E m = f	(.40–.59)	(.00–.09)	(.11–.14)	(.27–.44)	(.45–.66)	(.20–.42)	(.12–.16)					
Parental ratings	.78	—	.22	—	.81	—	.19	–.11	18.830	11	.064	–3.170
A m = f	(.74–.81)		(.19–.26)		(.77–.85)		(.15–.23)	(–.17– –.06)				

Note: A = additive genetic variance; C = common environment; E = unique environment; C' = sex-specific common environmental variance; s = sibling effect; m = males; f = females. The confidence intervals are shown in parentheses.

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Table 6

Relative Importance (%) of Genetic and Environmental Influences on Teacher-Rated and Parent-Rated Aggression and Hyperactivity-Impulsivity of the Preferred Bivariate Models

	Aggression				Hyperactivity-Impulsivity				Genetic and Environmental Correlation			
	A	C	E	C′	A	C	E	C′	rA	rC	rE	rC′
Teacher												
Boys	.42 (.35–.51)	.09 (.03–.16)	.08 (.07–.10)	.40 (.32–.48)	.50 (.41–.59)	.04 (.01–.10)	.12 (.11–.14)	.34 (.25–.42)	.70 (.62–.76)	Fixed to be 1[a]	.59 (.53–.64)	.81 (.72–.88)
Girls	.49 (.41–.58)	.41 (.32–.50)	.10 (.08–.11)	—	.55 (.46–.66)	.31 (.21–.41)	.13 (.12–.15)	—	.70 (.62–.76)	.70 (.56–.80)	.59 (.53–.64)	—
Parent												
Boys	.16 (.10–.25)	.71 (.60–.80)	.12 (.08–.18)	—	.66 (.60–.72)	.06 (.03–.10)	.27 (.23–.33)	—	.77 (.62–.91)	Fixed to be 1[b]	.21 (.14–.28)	—
Girls	.54 (.45–.62)	.27 (.19–.34)	.20 (.17–.24)	—	.79 (.73–.83)	.06 (.03–.10)	.15 (.12–.20)	—	.41 (.33–.49)	Fixed to be 1[c]	.27 (.19–.36)	—

Note: A = additive genetic effect; C = common environment; E = unique environment; C′ = sex-specific common environmental variance. The confidence intervals are shown in parentheses.
[a]Fixed to be 1, because it was estimated as .99 (.77–1.00).
[b]Fixed to be 1, because it was estimated as .89 (.62–1.00).
[c]Fixed to be 1, because it was estimated as .64 (.34–1.00).

constrained to be equal for boys and girls ($\Delta\chi^2_2$ = 2.974, p > .05).

Since the ordering of the variables in the bivariate analysis of parental rating data have been found to be crucial for the model fitting results, we tested whether changing the order of the variables affected the fit of the model and the parameter estimates. Compared to the full sex-limited ACE-model fitted initially, a comparable model in which hyperactivity-impulsivity was entered first in the analysis provided worse fit indexes (χ^2_{30} = 48.297, p = .019, AIC = –11.703). However, although the confidence intervals were somewhat larger in the latter model, the estimates of the two models were rather similar. Moreover, the interpretation of the results was the same regardless of the ordering of the variables. Further, the estimates remain quite similar when parameters (which are nonsignificant) are dropped from the model or set equal for boys and girls.

Table 6 presents the parameter estimates (standardized to reflect the percentage of phenotypic variance), 95% confidence intervals (CI), and the genetic and environmental correlations for the best fitting model. As in the preferred univariate model, in bivariate model for aggression for boys, the largest proportion of the total variance was attributed to common environmental factors (71%) and the rest of the variance was fairly equally partitioned to additive genetic (16%) and unique environmental (12%) factors. For girls, about half of the variance (54%) was attributable to additive genetic effects, while the other half was partitioned to common environmental (27%) and unique environmental (20%) factors. In hyperactivity-impulsivity, the largest proportion of the total variance was attributable to additive genetic factors, 66% for boys and 79% for girls. The rest of the variance was attributed to the unique and common environmental factors. The common environmental correlation between aggression and hyperactivity-impulsivity was 1.00 for both sexes because the common environmental effects specific to hyperactivity-impulsivity were nonsignificant, indicating that all common environmental factors affecting hyperactivity-impulsivity are the same as those affecting aggression. Additionally the genetic effects correlated considerably for both sexes (.77 for boys; .41 for girls). The unique environmental correlation was lower, but significant (.21 for boys; .27 for girls).

Discussion

These results, from a large, population-based twin sample, confirm through both univariate and bivariate analyses contributions of genetic and environmental factors to both teacher- and parent-rated aggression and hyperactivity-impulsivity. Moreover, bivariate results provide evidence that both genetic and environmental factors are important in creating the correlation between aggression and hyperactivity-impulsivity. It is apparent, however, that the sex of the rated twin and the informant who rated the twin's behavior also affected the results.

Genetic and Environmental Correlations

The genetic correlation between aggression and hyperactivity-impulsivity in both teacher and parental ratings was rather high, indicating that to a large degree that the same genes affect these behaviors. This result was in line with the hypothesis based on an earlier finding of a large genetic correlation between behavioral problems (Nadder et al., 2002; Nadder et al., 1998; Silberg et al., 1996; Schmitz & Mrazek, 2001; Thapar et al., 2001; Young et al., 2000). In addition, the common environmental correlation was high in both ratings. Moreover, the common environmental correlation of 1.00 for boys in teacher ratings and for both sexes in parental ratings suggested that

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the same factors in the family environment affected both behaviors. The finding of high common environmental correlations between aggression and hyperactivity-impulsivity are consistent with previous reports of common environmental correlations close to unity in studies using dimensional approaches on these behaviors (Burt et al., 2001; Schmitz & Mrazek, 2001). The unique environmental correlation was high in teacher ratings and lower, but significant in parental ratings. Usually the unique environmental factors have correlated moderately at most (Burt et al., 2001; Nadder et al., 2002; Nadder et al., 1998; Schmitz & Mrazek, 2001; Silberg et al., 1996; Thapar et al., 2001; Young et al., 2000). Future studies are needed to resolve what factors affect one twin only and contribute to co-occurrence of behavioral problems.

Sex Differences

Significant sex-specific effects were found in teacher ratings for both aggression and hyperactivity-impulsivity. However, neither univariate nor bivariate models could distinguish whether the models allowing sex-specific A effect or C effect fit better to the data. Further, the importance of sex-specific genetic and environmental factors could not be tested at the same time in a single model. The models with additional sex-specific C were chosen to be the preferred models because of slightly better fit indexes, which suggested that different factors in the twins' shared environment are contributing to teacher-rated aggression in boys and girls. However, the parental rating data provided no evidence of sex-specific effects. Rarely has the existence of sex-specific genetic and environmental influences been tested. Recent studies have found some evidence that the genetic factors influencing problem behaviors, such as conduct disturbance, aggression, and hyperactivity, may be quite similar, but not identical for boys and girls (Eaves et al., 2000; Rhee et al., 1999; Silberg et al., 1996; Vierikko et al., 2003). The qualitative sex differences in genetic and environmental effects are clearly a question to be addressed in future research. The range of genetic effects on aggression reported in the present study is slightly lower than in earlier studies of aggression (Edelbrock et al., 1995; Hudziak et al., 2000; Schmitz et al., 1995). However, the magnitude of genetic effects on hyperactivity-impulsivity in our results is consistent with studies reporting heritabilities ranging between .70–.82 in parental ratings (Eaves et al., 1997; Gjone et al., 1996; Kuntsi & Stevenson, 2001; Neuman et al., 2001; Silberg et al., 1996; Simonoff et al., 1998; Thapar et al., 2000) and between .50–.62 in teacher ratings (Eaves et al., 1997; Kuntsi & Stevenson, 2001; Simonoff et al., 1998; Thapar et al., 2000).

Our results emphasized the role of common environmental effects on teacher- and parent-rated aggression and teacher-rated hyperactivity-impulsivity. For parent-rated aggression and hyperactivity, many earlier studies found no significant common environmental effects (Chodsian-Carpey & Baker, 1987; Edelbrock et al., 1995; Gjone et al., 1996; Goodman & Stevenson, 1989; Hudziak et al., 2000; Kuntsi & Stevenson, 2001; Nadder et al., 1998; O'Connor et al., 1980; Schmitz et al., 1995; Silberg et al., 1996; Stevenson, 1992; Thapar et al., 1995; Van den Oord et al., 1996). Reports of common environmental effects are more usual for studies of non-aggressive antisocial behavior (Eley et al., 1999). The finding of unexpectedly high common environmental effect for both behaviors in the present study may reflect rater bias in teacher ratings (a possibility suggested in separate analysis of co-twins rated by independent teachers).

Sibling Effect/Rater Bias

Our results suggested either sibling interaction or effects of rater bias in preferred univariate and bivariate models for parent-rated aggression (for MZ and DZ boys) and hyperactivity-impulsivity (for MZ and DZ girls), and univariate model for teacher-rated aggression (for MZ and DZ boys), but these effects could be dropped from the bivariate teacher rated model. No such evidence was found in teacher ratings of hyperactivity-impulsivity. Rater bias (or, alternatively, sibling effects) on aggression has been tested or found only rarely (Hudziak et al., 2000). However, unexpectedly low (or even negative) DZ correlations in the context of high MZ correlations and variance differences between zygosity groups, which suggest rater bias and/or sibling interaction effects, have been reported repeatedly in maternal ratings of hyperactivity (Eaves et al., 2000; Eaves et al., 1997; Kuntsi & Stevenson, 2001; Nadder et al., 1998; Sherman et al., 1997; Simonoff et al., 1998; Thapar et al., 1995), but not for teacher ratings (Eaves et al., 1997; Kuntsi & Stevenson, 2001; Nadder et al., 1998; Sherman et al., 1997; Simonoff et al., 1998). The frequent finding of low or negative DZ correlations has been interpreted as a reflection of rater bias, particularly when found in maternal ratings (Simonoff et al., 1998). On the other hand, maternal contrast effects have been shown to vary for different measures and items assessing ADHD, suggesting that these contrast effects may be a feature of the scales used in behavioral assessments, rather than a bias in maternal ratings (Thapar et al., 2000).

A different bias may operate in teacher ratings, especially when the same teacher rates both co-twins. In Finnish culture, twin children are usually placed in the same classrooms; in this study, initially 87% of the co-twins were in the same class. Earlier research has found larger twin correlations for co-twins rated by the same teacher than for those rated by separate teachers (Simonoff et al., 1998; Towers et al., 2000). As Simonoff et al. (1998) have found, teachers may have difficulties in attributing behavior to a correct twin because of confusing the twins with each other. In addition, teachers may be biased to observe similarity in children who come from the same family, particularly when they are of the same sex. Comparing the correlations for co-twins rated by the same teacher against those rated by different teachers, reveals, as expected, lower twin correlations for twins rated by different teachers. In addition, the high twin correlations of both MZ and DZ same-sex twin pairs rated by the same teachers support the confusion effect in our data. It is possible that the results of remarkably common environmental influences, particularly for girls, reflect teachers' biasing effects. The present results require further study with multiple, independent informants to separate the rater bias effects from the common environmental component. However, it is possible that the twins in separate classrooms are truly more dissimilar, which may be why they were placed separately (e.g., behavior problems or special aptitudes in one twin).

Effects of Different Informants

The present study suggests that assessments made by different informants reflect different patterns of genetic and environmental influences. Teacher and parental ratings of both aggression and hyperactivity-impulsivity correlated only moderately, suggesting that these two informants, making observations in different contexts, have somewhat different perceptions of a target child's behavior. Assessing different phenotypes, in turn, may be associated with the differences between the ratings. The cross-rater correlations suggested that the phenotypic correlation between the ratings is mainly due to shared genetic and unique environmental factors. The role of common environmental factors on the correlation between the raters appeared to be negligible.

Limitations

In order to estimate qualitative sex differences the sample size must be large enough to have a power to estimate the sex specific effects, and OSDZ twin pairs must be included in the sample. With a relatively large twin sample, we found significant sex-specific effects, but it was impossible to distinguish whether these effects were genetic or environmental. The power for distinguishing A' or C' in our data in univariate analyses was low at the .05 significance level (with 1 *df*): .050 for detecting A' in aggression and hyperactivity-impulsivity, .19 for detecting C' in aggression, and .057 for detecting C' in hyperactivity-impulsivity.

Comparing separate models fit to independent data obtained from teachers and parents, we have inferred differences in parameter estimates of the sources of variation and covariation in aggression and hyperactivity-impulsivity. We recognize, however, that our inferences of these differences are just that: inferences only; to confirm them, we plan a follow-up to include both sets of ratings in the same models with appropriate constraints to rigorously test the differences inferred from our current analyses.

To understand discrepancies between the results that we report here and earlier findings by other investigators, differences in both design and twin samples may be relevant. Differences in the measurement and definition of behavior problems have proved critical for behavioral genetic analyses and results. Specific and narrow personality traits may be more sensitive to consistent sex differences than broader measures. Here, the definition of behavioral problems was based on a model of emotion regulation and behavior control (Pulkkinen, 1995), in which aggression and hyperactivity-impulsivity are defined as quite limited aspects of behavior problems. In addition, some studies have had relatively small sample sizes (Goodman & Stevenson, 1989; Thapar et al., 1995), and some have been restricted to same-sex twin pairs (Rhee et al., 1999). Including OSDZ twin pairs, as in the univariate and bivariate analyses of teacher- and parent-rated data reported here, yields valuable information regarding sex differences.

Summary

The high phenotypic correlation we report between aggression and hyperactivity-impulsivity is a common finding in both clinical and epidemiological studies (Biederman et al., 1991; Jensen et al., 1997; Pulkkinen et al., 1999). Our analyses show that the correlation is due to extensive genetic and common environmental overlap between the two behaviors. However, roughly half of the variance in hyperactivity-impulsivity was accounted for by genetic and environmental factors specific to it, suggesting somewhat different etiologies for these two correlated behaviors.

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