Behavior Genetics, Vol. 34, No. 6, November 2004 (© 2004)

A Study of Genetic and Environmental Influences on Maternal and Paternal CBCL Syndrome Scores in a Large Sample of 3-year-old Dutch Twins

E. M. Derks,[1,*] J. J. Hudziak,[2] C. E. M. van Beijsterveldt,[1] C. V. Dolan,[3] and D. I. Boomsma[1]

Received 27 March 2003—Final 26 July 2004

Background. There is increasing evidence that behavioral problems are common in very young children, yet little is known about the etiology of individual differences in these problems. It is unclear to what degree environmental and genetic factors influence the development of early child psychopathology. In this paper, we focus on the following issues. Firstly, to what degree do genetic and environmental factors influence variation in behavioral problems? Secondly, to what degree are these underlying etiological factors moderated by sex and informant? We investigate these issues by analyzing Child Behavior Checklist (CBCL) data on 9689 3-year-old twin pairs. *Methods.* Rater Bias and Psychometric Models were fitted to CBCL/2-3 data obtained from mothers and fathers to determine the genetic and environmental contributions to the five CBCL syndromes:aggressive, oppositional, overactive, withdrawn, and anxious/depressed behavior. *Results.* Parental ratings are influenced by aspects of the child's behavior that are experienced in the same way by both parents and by aspects of the child's behavior that are experienced uniquely by each parent. There is evidence for high genetic contributions to all CBCL syndromes. Shared and non-shared environmental influences play significant roles as well. One exception is overactive behavior, which is influenced by genetic and non-shared environmental influences only. *Conclusions.* Variation in behavior problems in the very young shows high heritability.Individual raters offer unique perspectives that can have an impact on estimates of problem behavior and genetic architecture. Therefore, multi-informant approaches in the assessment of the very young will be useful to clinicians and researchers alike.

KEY WORDS: preschool; children; genetic; twins; problem behavior; Child Behavior Checklist; rater models.

INTRODUCTION

Little is known about the etiology of behavioral disorders in the very young. Studies in this age group have focused mainly on the assessment of problem behavior in clinically referred or at risk samples (Shaw *et al.*, 2001; Thomas and Guskin, 2001). In order to obtain a better understanding of the etiology of psychopathology in non-clinical samples of very young children, we studied problem behavior in a sample of 9821 3-year-old twin-pairs, using the Child Behavior Checklist (CBCL) (Achenbach, 1991; Achenbach, 1992).

The CBCL, completed by mothers and/or fathers, has been used in studies of childhood behavior around the world (Achenbach and Rescorla, 2000). Many studies have shown that CBCL-scores predict behavior problems in children as they age.

[1] Department of Biological Psychology, Vrije Universiteit, Amsterdam, The Netherlands.
[2] Department of Psychiatry and Medicine (Division of Human Genetics), Center for Children, Youth and Families, University of Vermont, College of Medicine, Burlington, USA.
[3] Department of Psychology, University of Amsterdam, The Netherlands.
* To whom correspondence should be addressed at Department of Biological Psychology, Vrije Universiteit, Amsterdam, The Netherlands. Tel: +31-0-20-4448743; Fax: +31-0-20-4448832. e-mail:em.derks@psy.vu.nl

For example, CBCL syndrome scores for overactive behavior at age three and attention problems at age 12 correlate 0.37 in a large Dutch twin sample (Rietveld *et al.*, 2004). The correlation between aggressive scores at age 3 and 12 is 0.41 (Van Beijsterveldt *et al.*, 2003). Hofstra *et al.* (2000) identified children with deviant CBCL-scores, and showed that 41% of these children were still classified as deviant 14 years later. A final example that demonstrates the continuity of problem behavior is the study of Achenbach *et al.* (1995). CBCL-scores were used to predict symptoms of disturbance in adolescents, such as academic problems, suicidal behavior, and substance abuse. CBCL-scores accounted for an average of 31% of the variance of symptoms of disturbance measured 3 years later.

Parents and other caregivers are the main source of information when it comes to the assessment of problem behavior in young children. Because studies on the very young are relatively scarce, little is known about how best to use such parental reports. Fathers and mothers may not agree about the nature and degree of problem behavior in their children. Different raters are confronted with different aspects of their children's behavior, and may have different internal standards for the evaluation of behavior. For example, when DSM-interview data were collected from both parents to determine the presence or absence of psychopathology in their child, the correlations between maternal and paternal data ranged from 0.13 to 0.35 (Hewitt *et al.*, 1997). Achenbach *et al.* (2000) reported maternal and paternal correlations ratings of CBCL scales in 3-year-old twins that range from 0.48 to 0.67, with a mean correlation of 0.61. While these correlations are substantial, they still suggest that each parent has a unique perspective on the behavior of their offspring.

One advantage of using a twin population to study behavior problems, is that genetic models can be used to test whether maternal and paternal ratings diverge because of different internal standards (rater bias), or if their ratings reflect different, but valid, aspects of the child's behavior. In other words, when data from multiple informants are available, a distinction can be made between:(a) variance explained by the environment that is shared between siblings, (b) variance that is explained by rater bias, (c) variance that is explained by a common perception of the parents, and (d) variance that is explained by a unique perception of each parent. To investigate if the ratings of multiple informants disagree because of different internal standards or because of the reflection of different, but valid, perceptions of the child's behavior, two structural models have been developed. The Rater Bias Model (Hewitt *et al.*, 1992; Neale and Stevenson, 1989) allows the parental ratings to be influenced by the behavior of the child and by rater bias, which gives rise to disagreement between the parents. The Psychometric Model (Hewitt *et al.*, 1992) allows the parental ratings to be influenced by aspects of the child's behavior that are experienced commonly by both parents, and by aspects of the child's behavior that are experienced uniquely by each parent.

The genetic contributions to the two broadband scales (externalizing and internalizing) of the CBCL in Dutch 3-year-olds, as well as to the seven behavioral syndromes (aggressive, oppositional, overactive, withdrawn, anxious/depressed, sleep problems, and somatic problems) were reported by Van der Valk *et al.* (1998, 2001) and by Van den Oord *et al.*, (1996). Van der Valk *et al.* (1998) studied maternal reports of externalizing and internalizing behavior in 3-year-old children, and found that additive genetic factors explained 54% of the variance in externalizing behavior, and 64% of the variance in internalizing behavior. Shared and non-shared environmental factors explained the remaining part of the variance of the CBCL broadband scales. More recently, van der Valk *et al.* (2001) combined maternal and paternal scores of externalizing and internalizing in 3-year-olds and compared the fit of the Rater Bias and Psychometric Model. The Psychometric Model fitted the data better than the Rater Bias Model. The parents were found to assess a common component in the behavior in their children, and, in addition, each parent experienced unique aspects of their children's behavior. The heritability of internalizing behavior was 66%. The heritability of 66% was mostly explained by a common view of the parents (86%), and in addition by a unique view of the parents (14%). The heritability of externalizing behavior was 54%. The common view of the parents explained 87% of the heritability and the unique view explained 13% (Van der Valk *et al.*, 2001). These results agree with the results of Hewitt *et al.* (1992), who studied internalizing behavior in an 8- to 11-year-old sample and in a 12- to 16-year-old sample of twins. The Psychometric Model fitted the data better than the Rater Bias Model in both samples. This supports the hypothesis that also at older ages, disagreement is

not caused by parental bias, but by the fact that the parents assess partially different aspects of the children's behavior.

Additive genetic and environmental influences on the seven CBCL syndromes were reported by Van den Oord *et al*. (1996) in a sample of 1358 twin pairs. For all scales, parental ratings were for the most part expressions of the same underlying trait. Therefore, Van den Oord et al. averaged parental scores in the genetic model fitting to obtain estimates of genetic and environmental influences. Additive genetic effects explained the main part of the variance of the CBCL syndromes (60–74%). Shared environmental effects influenced individual differences in oppositional and aggressive behavior (both 12%). Non-shared environmental influences explained the remaining part of the variance (19–40%). The authors acknowledged the possible influence of non-additive genetic effects on overactive behavior, but these effects were not included within the genetic model, due to inadequate power to detect these effects.

In this paper we analyze behavioral problems in over 9600 Dutch twin-pairs aged 3 years. Ratings of their behavior were obtained from mothers and fathers. Because of the large sample size, we have sufficient statistical power to detect genetic dominance, and sibling interaction effects, if present. In addition, we will investigate whether the parameter estimates of the genetic model fitting are similar in boys and girls. Van den Oord et al. found small sex differences, but did not include these in the final models. We analyze maternal and paternal ratings of aggressive, oppositional, overactive, withdrawn, and anxious/depressed behavior, because these scales represent the most common behavioral problems in the very young. To determine whether parents assess different aspects of their children's behavior, we fit Rater Bias and Psychometric Models. We focus on the behavioral syndromes instead of the broadband scales because the behavioral syndromes may form a better basis for prescribing treatment (Achenbach and Edelbrock, 1984), and may be more suitable for future gene-finding studies (Hudziak, 1997; Hudziak, 2001).

METHOD

Subjects

This study is part of an ongoing longitudinal twin study in the Netherlands. The subjects were all registered at birth with the Netherlands Twin Registry (Boomsma, 1998; Boomsma *et al*., 2002). For the present study, we analyzed data of a sample of Dutch twins, whose parents (or primary caregivers) reported on their behavior when they were 3 years old. These twins were all born between 1986 and 1997. Of the total sample of 9969 pairs, 152 pairs were excluded from statistical analyses because a health questionnaire completed at age three indicated that one or both of the twins suffered from a disease or handicap that interfered severely with daily functioning. The resulting sample comprises 9817 pairs. The zygosity status of 128 pairs was unknown; these pairs were excluded from the genetic analyses. The sample that was used for the genetic analyses consisted of 9689 pairs.

Zygosity diagnosis was assessed with the use of a 10-item questionnaire. This procedure allows an accurate determination of zygosity of nearly 95%. It is described in more detail in Rietveld *et al*. (2000). The number of twin pairs, by sex, zygosity and informant are presented in Table I.

Procedure

A survey, including the CBCL/2-3, was mailed to the fathers and mothers of the twins when the twins were 3 years old. Due to funding problems, this questionnaire was only sent to the mother of the twins born between May 1989 and November 1991. Parents who did not return the forms within 2 months received a reminder, and during some years, persistent non-responders were contacted by phone 4 months after the initial mailing. This procedure resulted in a 75.5% participation rate (Rietveld *et al*., 2004).

Table I. Number of Twin Pairs by Sex, Zygosity and Informant

Zygosity	Father	Mother	Total
MZm	1033	1519	1561
DZm	1059	1594	1635
MZf	1156	1736	1777
DZf	972	1454	1494
DOS	2030	3142	3222
Unknown	76	125	128
Total zygosity known	6250	9445	9689
Total	6326	9570	9817

Note: MZm, Monozygotic male; DZm, Dizygotic male; MZf, Monozygotic female; DZf, Dizygotic female; and DOS, Opposite sex.

Measure

The CBCL/2-3 is a standardized questionnaire for parents to report the frequency and intensity of behavioral and emotional problems exhibited by their child in the past 6 months. It contains 100 items that measure problem behavior; the items are rated on a 3-point scale ranging from ''not true'', ''somewhat or sometimes true'', to ''very true or often true''. The CBCL measures the number of symptoms of seven behavioral syndromes, which can be combined to form two broadband scales:externalizing and internalizing behavior. The seven syndromes were derived from factor analyses of the problem items. These factor analyses resulted in the formation of the problem scales oppositional (17 items), withdrawn (10 items), aggressive (9 items), anxious/depressed (9 items), overactive (5 items), sleep problems (7 items) and somatic problems (3 items) (Koot *et al.*, 1997). Sleep problems and somatic problems were not analyzed in this study, because the prevalence of these problems was very low.

Statistical Analyses

Means and standard deviations of untransformed CBCL-scores were calculated using SPSS/Windows 11.0. (SPSS, 2001). The distributions of these scores are skewed. In order to obtain a distribution that approaches normality with respect to skewness and kurtosis, normal scores were computed with Prelis (Jöreskog, 1993).

The effects of sex and zygosity on these normal CBCL-scores were tested in an ANOVA. The effects were examined in first and second born twins separately, to avoid dependency inherent in twin data. The type-I error rate was corrected for multiple testing in two ways. First, the α (type I error probability) of each test was set to equal 0.01. Second, an effect was only considered to be present if it was significant given α is 0.01, in both first and second born twins.

Twin correlations among CBCL syndrome scores were computed in Mx (Neale, 1997). Structural Equation Modeling (SEM) was employed to obtain an estimate of the genetic and environmental contributions to the observed variances and covariances. An assumption of SEM is that the data are normally distributed. Therefore, these analyses were carried out on the normal scores (Jöreskog, 1993). The genetic model fitting analyses were performed on raw data with Mx, using maximum likelihood estimation. Point estimates and confidence intervals for the estimated genetic and environmental parameters are reported (Neale, 1997; Neale and Miller, 1997). Technical details of genetic model-fitting analyses are reviewed elsewhere (Neale and Cardon, 1992).

Model Fitting

Variation in a phenotypic trait can be decomposed into latent genetic and environmental components. The decomposition of variance takes place by comparing the degree of similarity between pairs of individuals, who differ in their degree of genetic relatedness. Monozygotic twins are genetically identical, while dizygotic twins on average share half of their segregating genes. Limiting the genetic decomposition of phenotypic variance to additive genetic (A) effects and dominant genetic (D) effects, the fact that MZ twins are genetically identical implies that they share all the additive genetic and dominant genetic variance. DZ twins on average share half of the additive genetic and one quarter of the dominant genetic variance (Plomin, *et al.*, 2001). In addition to the genetic components, the phenotypic variance is decomposed into shared and non-shared environmental variance. The shared environmental variance is due to environmental effects shared by two members of a twin pair (C). These effects are by definition perfectly correlated in both monozygotic and dizygotic twins. The non-shared environmental variance is due to effects (E) which are by definition uncorrelated between twin pair members. Estimates of the non-shared environmental variance usually include measurement error (Plomin *et al.*, 2001). In fitting models to MZ and DZ twin data, it is not possible to estimate the effects of all mentioned sources of variance (A, D, C, and E). Specifically, with E and A in the model, one cannot estimate D and C simultaneously.

The Rater Bias Model (Figure 1) allows one to estimate variance due the effects of genetic and environmental factors (note that Figure 1 includes all four sources of variance A, D, C, E, even though, as mentioned, they cannot all be estimated simultaneously). In this model, the parental ratings of their children's behavior are not only influenced by the child's behavior, but also by rater bias and residual error. The influence of the child's behavior on the ratings of the fathers and the mothers may differ. To identify this model, the factor loading of the child's behavior on the maternal ratings is fixed to

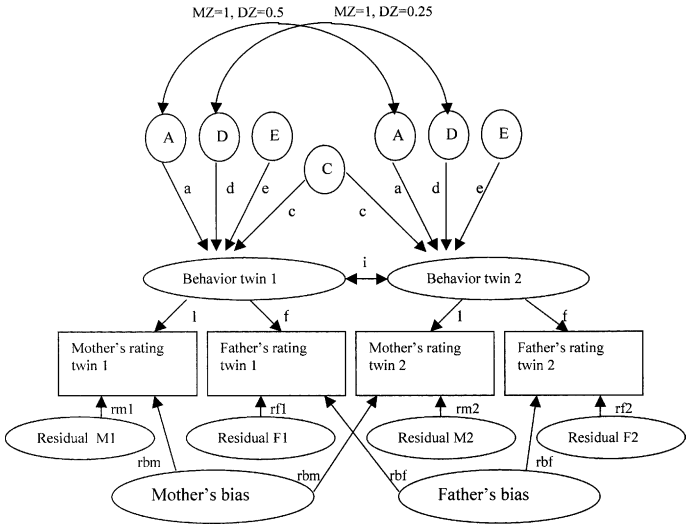

Fig. 1. Rater Bias Model. *Note*: A, additive genetic factor; D, dominant genetic factor; C, shared environmental factor; E, non-shared environmental factor; f, the factorloading from behavior of twins on father rating.

1, whereas the factor loading of the child's behavior on the paternal ratings is freely estimated. In addition, in the full model, we do not constrain the parental bias to be equal for MZ and DZ twins. This allows for the possibility that parental biases are influenced by the beliefs that parents have about their twin's zygosity (Neale and Cardon, 1992).

The Psychometric Model (Figure 2; again including all four sources of variance) allows the parental ratings to be influenced by aspects of the child's behavior that are perceived commonly by both parents, and by aspects of the child's behavior that are perceived uniquely by each parent. Unique perceptions could arise if the child behaves differentially towards his or her parents, or if the parents observe the child in different situations. The common and unique aspects are both influenced by genetic and environmental factors.

In both models, we have added a path with coefficient *i* between the CBCL-scores of the twins. This path implies an interaction that may be interpreted in two ways (Simonoff *et al.*, 1998). First, it may be considered an interaction between siblings (Carey, 1986; Eaves, 1976). Second, the path may be considered an effect introduced by the rater, who may compare the behavior of one child with the behavior of the other child. The latter may thus be interpreted as a rater contrast effect. Very low DZ correlations compared to MZ correlations give a first indication that a competitive social interaction effect or negative rater contrast effect is present. However, such a configuration of twin correlations also suggests an ADE model. One way to distinguish between these possibilities is by testing the observed variances for MZ and DZ twins. An interaction effect leads to different variances in MZ and DZ twins (Hewitt *et al.*, 1992). In case of dominance, MZ and DZ variances are expected to be equal. If a cooperative social interaction effect or a positive rater contrast effect is present, the pattern of MZ and DZ correlations resembles an ACE model (i.e., DZ correlation greater than half the MZ correlation). Again the model that includes an interaction term gives rise to differences in variances of MZ and DZ twins (Eaves, 1976), and thus may be distinguished from an ACE model by comparing variances.

Model Fitting Procedure

The first step in the model fitting procedure was to determine whether the interaction parameters were required (i.e., deviated significantly from zero). The fit of the model, in which the variances of MZ twins and DZ twins were constrained to be equal, was compared with the fit of a fully saturated model, in which all variances and covariances were freely estimated. An interaction effect was included only if the variances of MZ and DZ twins were significantly different.

The second step was to choose between an ACE or an ADE model as the starting model. As mentioned above, with data of twins reared together, the effects of dominance and shared environment cannot be estimated simultaneously. The

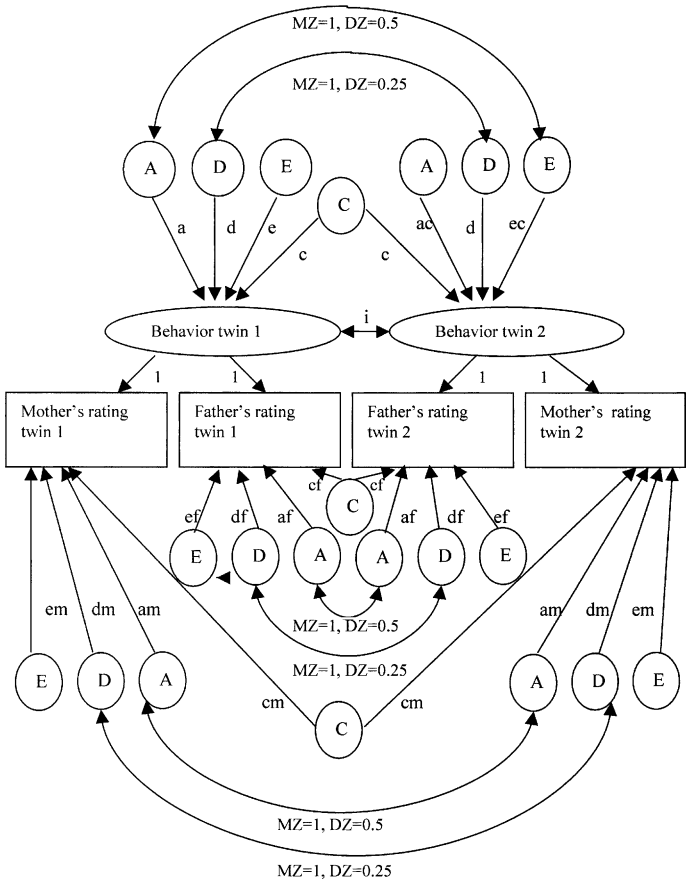

Fig. 2. Psychometric Model. *Note*: A, additive genetic factor; D, dominant genetic factor; C, shared environmental factor; E, non-shared environmental factor; ac, additive genetic common; dc, dominant genetic common; ec, non-shared environment common; cc, shared environment common; am, additive genetic maternal; dm, dominant genetic maternal; em, non-shared environment maternal; cm, shared environment maternal; af, additive genetic father; df, dominant genetic father; ef, non-shared environment father; cf, the shared environment father.

choice between the initial models was based on the phenotypic correlations: an ADE model was chosen if the MZ correlations were more than twice the DZ correlations, an ACE model was chosen if the MZ correlations were twice or less than twice the DZ correlations. The fit of the Rater Bias and Psychometric Model was then assessed by comparing the likelihood of these models to the likelihood of a fully saturated model.

In order to test for sex differences, we compared the likelihood of a model that includes estimates of parameters that vary over boys and girls with the likelihood of a model that equates all model parameters over sex. This test is also sensitive for absolute variance differences between boys and girls, because the absolute factor loadings were equated. Finally, the significance of the common and unique influences of A, and C or D was tested by means of likelihood ratio tests.

RESULTS

The means and standard deviations of the five CBCL-scales are reported by the six different zygosity groups by maternal and paternal report in Table II. Note that the final two columns in Table II give the means by sex.

Estimates of skewness and kurtosis before and after normalization in Prelis are shown in Table III. As expected, the transformation resulted in better (i.e., more normal) values of skewness and kurtosis. The transformed data were used for the tests of sex and zygosity effects, and for the genetic model fitting.

Table II. Mean CBCL-scores and Standard Deviations

Informant	MZm	DZm	MZf	DZf	DOSm	DOSf	All boys	All girls	Total
Oppositional	M	10.7 (6.5)	10.5 (6.6)	10.3 (6.5)	10.3 (6.5)	9.9 (6.5)	9.7 (6.3)	10.3 (6.5)	10.1 (6.5)
	F	10.0 (6.4)	9.9 (6.2)	9.5 (6.2)	9.7 (6.3)	9.3 (6.2)	9.1 (6.0)	9.8 (6.3)	9.4 (6.2)
Aggressive	M	4.3 (3.0)	3.9 (3.0)	2.9 (2.5)	2.8 (2.5)	3.1 (2.7)	3.0 (2.6)	4.0 (3.0)	2.7 (2.5)
	F	3.7 (2.9)	3.6 (2.8)	2.5 (2.3)	2.4 (2.3)	2.8 (2.5)	2.7 (2.5)	3.6 (2.8)	2.4 (2.3)
Overactive	M	3.0 (2.2)	2.8 (2.3)	2.6 (2.1)	2.6 (2.2)	2.6 (2.2)	2.5 (2.2)	2.9 (2.2)	2.5 (2.1)
	F	2.9 (2.2)	2.8 (2.2)	2.6 (2.1)	2.6 (2.2)	2.5 (2.2)	2.4 (2.1)	2.8 (2.2)	2.5 (2.1)
Withdrawn	M	1.2 (1.6)	1.2 (1.6)	1.1 (1.4)	1.2 (1.5)	1.1 (1.5)	1.0 (1.4)	1.2 (1.6)	1.1 (1.5)
	F	1.1 (1.5)	1.2 (1.5)	1.0 (1.3)	1.1 (1.5)	1.0 (1.4)	1.0 (1.4)	1.1 (1.5)	1.0 (1.4)
Anxious	M	3.5 (3.0)	3.5 (3.1)	3.8 (3.2)	3.8 (3.2)	3.4 (3.0)	3.2 (3.0)	3.5 (3.1)	3.5 (3.1)
	F	3.2 (2.8)	3.4 (3.0)	3.5 (3.0)	3.6 (3.0)	3.2 (2.9)	3.1 (2.9)	3.3 (3.0)	3.3 (3.0)

Table III. Skewness and Kurtosis of CBCL Scales and their Standard Errors (SE) Before and After Transformation in Prelis

	Before transformation		After transformation (normal scores)	
	Skewness (SE)	Kurtosis (SE)	Skewness (SE)	Kurtosis (SE)
Maternal				
Aggression	1.11 (0.02)	1.27 (0.04)	0.15 (0.02)	−0.33 (0.04)
Oppositional	0.59 (0.02)	−0.08 (0.04)	0.03 (0.02)	−0.13 (0.04)
Overactive	0.67 (0.02)	−0.17 (0.04)	0.20 (0.02)	−0.47 (0.04)
Withdrawn	2.39 (0.02)	8.51 (0.04)	0.56 (0.02)	−0.50 (0.04)
Anxious/Depressed	1.04 (0.02)	0.93 (0.04)	0.18 (0.02)	−0.42 (0.04)
Paternal				
Aggression	1.16 (0.02)	1.47 (0.04)	0.18 (0.02)	−0.38 (0.04)
Oppositional	0.62 (0.02)	0.03 (0.04)	0.04 (0.02)	−0.14 (0.04)
Overactive	0.68 (0.02)	−0.15 (0.04)	021 (0.02)	−0.47 (0.04)
Withdrawn	2.23 (0.02)	7.12 (0.04)	0.60 (0.02)	−0.49 (0.04)
Anxious/Depressed	1.03 (0.02)	0.92 (0.04)	0.20 (0.02)	−0.44 (0.04)

Contributions of sex and zygosity on CBCL syndrome scores were tested in a two-way ANOVA. According to reports from both parents, boys had higher scores than girls on aggression, overactive, and withdrawn. Both maternal and paternal reports revealed higher scores in MZ than DZ twins on aggression and overactive behavior. Table IV provides an overview of statistically significant main effects of sex and zygosity. No significant interaction effects of sex and zygosity were found.

The inter-parent correlations of the CBCL syndromes are high and significant (Table V). The correlations ranged from 0.54 to 0.71 for the five syndromes.

Genetic Analyses

The correlations of the twins' CBCL-scores are shown in Table VI. Based on the correlations, the ACE model served as the initial model for the genetic analyses on aggressive, oppositional, withdrawn and anxious/depressed behavior. The ADE model served as the initial model for overactive behavior.

The results of the genetic analyses are summarized in Table VII for externalizing problem behaviors (aggressive, oppositional and overactive), and in Table VIII for internalizing problems (withdrawn and anxious/depressed). The best fitting model is printed in bold.

Tests of differences in variance between MZ and DZ twins revealed no significant differences. Therefore, interaction parameters were not included. For all syndromes, the Psychometric Model provided a better fit than the Rater Bias Model. The significance of A, and C or D was therefore tested by dropping A, and C or D from the Psychometric Model. Four significance tests were performed by fixing common influences of A, unique influences of

Table IV. An Overview of the Effects of Sex and Zygosity on Transformed CBCL-scores

	Mother		Father	
	Sex	Zygosity	Sex	Zygosity
Aggressive	**	**	**	**
Oppositional				
Overactive	**	**	**	*
Withdrawn	*			
Anxious				

*$p < 0.01$; **$p < 0.001$.

Table V. Parental Correlations on Transformed CBCL-scores

	Parental Correlation		
	Boys	Girls	Opposite sex twins
Aggressive	.68	.66	.63
Oppositional	.71	.70	.71
Overactive	.68	.67	.66
Withdrawn	.53	.54	.55
Anxious	.68	.69	.68

A, common influences of C or D, and unique influences of C or D to zero. The significance of single parameters can be evaluated by considering the confidence intervals of the parameter estimates, which are reported for all parameters in the full Psychometric Model (Table IX).

The variance in aggressive behavior is explained adequately by additive genetic influences (A), shared environmental influences (C), and non-shared environmental influences (E) ($\chi^2(42) = 55.74$, $p = 0.08$). These factors were mainly explained by a common view of the parents (about 65%), but the factors that were viewed uniquely by each parent also explained a significant part of the variance. The influence of the common and unique factors differed significantly in boys and girls ($\chi^2(9) = 100.86$, $p = 0.00$). The estimates show that the genetic influences are larger in girls than in boys while the shared environmental influences are higher in boys than in girls.

The variance in oppositional behavior is explained by common and unique influences of A, C, and E ($\chi^2(51) = 53.44$, $p = 0.38$). The influence of these factors are identical in boys and girls ($\chi^2(9) = 4.48$, $p = 0.48$). In comparison with the other behavior scales, large shared environmental influences were found.

The best fitting model for overactive behavior included A (common and unique), D (common only), and E (common and unique) ($\chi^2(53) = 105.03$, $p = 0.00$). Although common influences of A were non-significant, this factor was not removed from the model, because the presence of dominant influences

Table VI. Maternal, Paternal and Cross-informant CBCL Twin-correlations by Zygosity Status

	MZM	DZM	MZF	DZF	DOS-MF	DOS-FM
Aggression						
Maternal	.83	.55	.83	.51	.48	.53
Paternal	.80	.53	.83	.51	.51	.55
Cross-rater	.57	.38	.60	.34	.30	.34
Oppositional						
Maternal	.79	.53	.79	.50	.50	.51
Paternal	.80	.57	.81	.54	.54	.57
Cross-rater	.60	.38	.57	.35	.34	.38
Overactive						
Maternal	.69	.14	.69	.15	.22	.19
Paternal	.64	.12	.67	.21	.22	.18
Cross-rater	.46	.04	.46	.08	.10	.08
Withdrawn						
Maternal	.74	.46	.72	.51	.45	.42
Paternal	.75	.50	.72	.55	.47	.49
Cross-rater	.45	.27	.43	.34	.27	.28
Anxiety						
Maternal	.68	.32	.72	.33	.38	.32
Paternal	.67	.35	.71	.43	.39	.36
Cross-rater	.49	.21	.53	.26	.24	.23

Table VII. Model Fitting Results, Externalizing Disorders

	N par	With model	Δ df	Aggression (ACE) −2 LL	Aggression (ACE) $\Delta\chi^2$	Aggression (ACE) p	Oppositional (ACE) −2 LL	Oppositional (ACE) $\Delta\chi^2$	Oppositional (ACE) p	Overactive (ADE) −2 LL	Overactive (ADE) $\Delta\chi^2$	Overactive (ADE) p
1. Fully saturated	84	–	–	132867.7	–	–	186356.3	–	–	125599.6	–	–
2. Equal variances[a]	76	1	8	132876.7	9.0	.34	186370.5	14.2	.08	125611.6	12.0	.15
3. Rater bias, unequal bias MZ/DZ	44	1	40	133109.7	242.0	.00	186469.3	113.0	.00	125825.8	226.2	.00
4. Rater bias, equal bias MZ/DZ	40	3	4	133125.5	15.8	.00	186487.0	17.7	.00	125871.3	45.6	.00
5. Psychometric + sex differences	42	1	42	**132923.5**	**55.7**	**.08**	186405.3	49.0	.21	125687.3	87.8	.00
6. Psychometric − sex differences	33	5	9	133024.3	100.9	.00	**186409.8**	**4.5**	**.48**	125702.7	15.3	.08
7. Psychometric unique A excluded[b]	38/31[b]	5/6[c]	4/2	133160.5	237.1	.00	186520.0	110.3	.00	125808.8	106.1	.00
8. Psychometric common A excluded[b]	40/32[b]	5/6[c]	2/1	133574.4	651.0	.00	187006.9	597.2	.00	125702.7	.0	–
9. Psychometric unique C/D excluded[b]	38/31[b]	5/6[c]	4/2	133054.7	131.2	.00	186748.1	338.3	.00	**125704.6**	**1.9**	**.38**
10. Psychometric common C/D excluded[b]	40/32[b]	5/6[c]	2/1	132977.7	54.27	.00	186470.5	60.8	.00	125918.5	215.8	.00

[a]The variances of MZ and DZ twins were fixed on similar values, to test if social interaction is plausible.
[b]The number of parameters varies because sex differences were included if these were significant in previous model, they were excluded if non-significant in previous models.
[c]The model is compared to the psychometric model with sex differences if sex differences were significant and to the psychometric model without sex differences if sex differences were not significant.

in the absence of additive genetic influences is biologically implausible (Falconer and Mackay 1996). The influences of A, D, and E were similar in boys and girls (χ^2 (9) = 15.33, $p = 0.08$). The fit of the best-fitting model is rather poor ($p < 0.05$). However, it should be remembered that the χ^2 fit index is a function of sample size, which in the present case is large (N = 9689 pairs).

The variance in withdrawn behavior is explained by an ACE model (χ^2 (42) = 57.61, $p = 0.06$). Both common and unique factors are present, but the common factors were slightly more important than the unique factors. The effects of these factors were significantly different in boys and girls (χ^2 (9) = 30.65, $p = 0.00$). The genetic influences were higher in boys than in girls while the shared

Table VIII. Model Fitting Results, Internalizing Disorders

	N par	With model	Δ df	Withdrawn (ACE) −2 LL	Withdrawn (ACE) $\Delta\chi^2$	Withdrawn (ACE) p	Anxiety (ACE) −2 LL	Anxiety (ACE) $\Delta\chi^2$	Anxiety (ACE) p
1. Fully saturated	84	–	–	102032.5	–	–	143968.7	–	–
2. Equal variances[a]	76	1	8	102041.5	9.0	.34	143983.1	14.5	.07
3. Rater bias, unequal Bias MZ/DZ	44	1	40	102214.1	181.6	.00	144110.3	141.7	.00
4. Rater bias, equal bias MZ/DZ	40	3	4	102221.5	7.4	.12	144120.7	10.4	.03
5. Psychometric + sex Differences	42	1	42	**102090.1**	**57.6**	**.06**	144025.9	57.2	.06
6. Psychometric − sex Differences	33	5	9	102120.7	30.7	.00	144037.1	11.2	.26
7. Psychometric Unique A excluded[b]	38/31[b]	5/6[c]	42	102242.3	152.2	.00	144183.6	146.5	.00
8. Psychometric Common A excluded[b]	40/32[b]	5/6[c]	2/1	102344.3	254.2	.00	144558.6	521.5	.00
9. Psychometric Unique C/D excluded[b]	38/31[b]	5/6[c]	4/2	102146.7	56.6	.00	144089.5	52.4	.00
10. Psychometric Common C/D excluded[b]	40/32[b]	5/6[c]	2/1	102137.9	47.8	.00	**144037.1**	**.0**	–

[a]The variances of MZ and DZ twins were fixed on similar values, to test if social interaction is plausible.
[b]The number of parameters varies because sex differences were included if these were significant in previous model, they were excluded if non-significant in previous models.
[c]The model is compared to the psychometric model with sex differences if sex differences were significant and to the psychometric model without sex differences if sex differences were not significant.

Table IX. The Standardized and Unstandardized Estimates of the Genetic and Environmental Influences

		Common part			Unique part paternal		Unique part maternal	
		Stand. paternal	Stand. maternal	Non-stand. (95% conf. interval)	Stand.	Non-stand. (95% conf. interval)	Stand.	Non-stand. (95% conf. interval)
Aggression Girls	A	50	44	2.98 (2.67–3.24)	11	.66 (.66–.88)	20	1.32 (1.31–1.62)
	C	10	9	.62 (.38–.70)	11	.67 (.60–.95)	9	.61 (.33–.61)
	E	8	7	.46 (.45–.53)	10	.59 (.58–.59)	11	.72 (.64–.80)
Aggression Boys	A	43	38	2.97 (2.97–3.39)	7	.51 (.51–.82)	19	1.52 (1.51–1.86)
	C	18	16	1.26 (1.26–1.69)	11	.77 (.49–.77)	10	.75 (.64–.76)
	E	11	10	.76 (.76–.86)	10	.68 (.64–.77)	8	.61 (.57–.70)
Oppositional Boys/Girls	A	19	18	18.16 (16.73–19.62)	4	.51 (.50–1.49)	15	5.60 (5.60–6.54)
	C	38	35	5.70 (5.68–7.11)	16	6.80 (5.93–7.75)	8	3.92 (3.92–5.01)
	E	16	15	4.90 (4.89–5.29)	17	2.97 (2.78–2.97)	9	4.06 (3.71–4.42)
Overactive Boys/Girls	A	0	0	.00 (.00–.08)	17	.80 (.80–.80)	18	.862 (.57–1.09)
	D	49	48	2.28 (2.16–2.39)	0	.00 (.00–.11)	4	.21 (.00–.48)
	E	19	19	.90 (.88–.97)	14	.67 (.67–.73)	11	.54 (.49–.54)
Withdrawn Girls	A	27	24	.53 (.39–.68)	13	.25 (.25–.38)	25	.56 (.56–.67)
	C	20	18	.40 (.26–.48)	14	.27 (.16–.40)	6	.13 (.13–.13)
	E	9	8	.18 (.15–.19)	18	.35 (.33–.39)	19	.41 (.41–.41)
Withdrawn Boys	A	40	36	.85 (.85–.95)	15	.32 (.18–.32)	24	.56 (.56–.56)
	C	8	8	.18 (.10–.28)	12	.26 (.16–.39)	7	.17 (.04–.32)
	E	10	9	.21 (.21–.24)	15	.33 (.33–.38)	18	.42 (.42–.47)
Anxiety Boys/Girls	A	54	49	4.64 (4.41–4.82)	5	.42 (.42–.62)	20	1.93 (1.86–2.10)
	C	0	0	.00 (.00–.13)	11	.95 (.94–1.22)	1	.06 (.00–.36)
	E	18	16	1.54 (1.42– 1.65)	13	1.10 (.99–1.22)	14	1.29 (1.18–1.41)

Note: Stand.,= standardized estimate of the variance. Nonstand.= non-standardized estimate of the variance.

environmental influences were higher in girls than in boys.

Anxious/depressed behavior in children is influenced by A (common and unique), C (unique only), and E (common and unique) (χ^2 (52) = 68.46, $p = 0.06$). These effects were similar for boys and girls (χ^2 (9) = 11.23, $p = 0.26$).

Table IX reports the standardized and unstandardized estimates of the genetic and environmental influences. For example, aggressive behavior is explained by factors that influence both parental ratings (common view), and by factors that influence only the paternal or only the maternal rating (unique view). The paternal ratings of girls are explained by these common factors for 50 (A) + 10 (C) + 8 (E) = 68%. The remaining variance is unique to the paternal rating:11 (A) + 11 (C) + 10 (E) = 32%. The heritability of the paternal ratings of aggression in girls can be calculated by summing the standardized variances that are explained by the genetic factor that influences both parental ratings (50%) and the genetic factor that influences the paternal rating only (11%). The heritability of the paternal ratings is therefore 61%. Likewise, the heritability of the maternal ratings of aggression in girls is 64%.

The lack of significant sex differences in oppositional, overactive, and anxiety implies that the absolute variances are also equal in boys and girls. The sex differences in aggression and withdrawn may be caused by differences in the relative influences of A, C, and E but also by differences in absolute variances. Additional tests revealed that the absolute variances in aggression were slightly higher in boys than in girls. The absolute variances in withdrawn behavior were not significantly different between boys and girls.

A graphical representation of the genetic and environmental influences on behavioral problems is given in Figure 3. The genetic influences are the sum of common and unique additive and dominant genetic influences and is therefore a representation of the total heritability. The shared environmental influences are the sum of the common and unique shared environmental influences. Likewise, the nonshared environmental influences are the sum of the

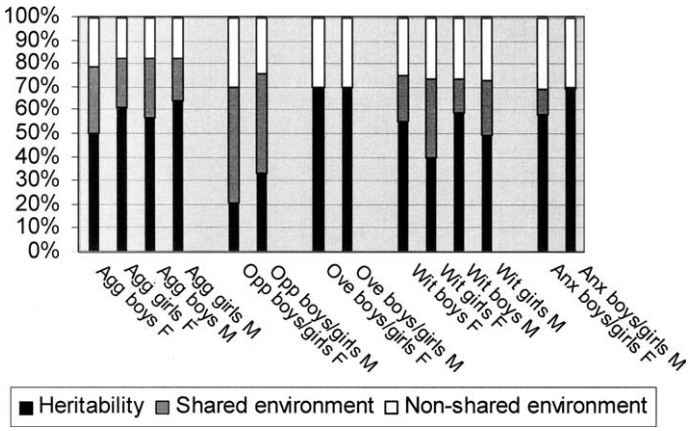

Fig. 3. Graphical representation of the influences of genes, and shared and non-shared environment (common + unique influences). *Note*: Agg, aggression; Opp, oppositional; Ove, overactive; Wit, withdrawn; Anx, anxious/depressed; F, father; M, mother.

common and unique non-shared environmental influences.

DISCUSSION

The goal of this study was to examine the relative influences of genes and environment on variation in problem behavior in 3-year-old boys and girls. We choose the CBCL because it is a widely used, quantitative, highly reliable instrument.

The data show that mothers and fathers agree to a large extent about the degree of problem behavior in 3-year-old children. The correlations we report are slightly higher than those reported for preschool children by Achenbach (1992) and Koot (1993), but similar to the correlations found in older children (Achenbach, 1991).

For all scales, the Psychometric Model provided a better fit to the data than the Rater Bias Model. This implies that differences between parental reports are not only influenced by rater bias, but by aspects of the child's behavior that are perceived uniquely by each parent. This is in agreement with the findings of Hewitt *et al.* (1992) and Van der Valk *et al.* (2001). Although the parental ratings were influenced by unique perceptions of the child's behavior, the major part of the variance in problem behavior was explained by aspects of the child's behavior that were perceived commonly by the parents. These common perceptions explained 50–73% of the variance in the problem behavior scales.

Individual differences in problem behavior in 3-year-old children are mainly due to genetic differences. The large sample size allowed us to test whether shared environment contributes to problem behavior. To date it has been difficult to determine whether the often reported absence of shared environmental influences is due to the actual absence of these influences or to inadequate power to detect them in the classical twin design (Rutter *et al.*, 1999). Confidence intervals are usually wide, even with a sample size as large as 2682 twin pairs (Slutske *et al.*, 1997). With the current sample size of 9689 Dutch 3-year-old twin pairs, we detected significant shared environmental influences on four of the five scales:aggressive, oppositional, withdrawn and anxious/depressed. Because multiple rater data have been used, these shared environmental influences are not confounded with rater bias. However, as is to be expected in view of previous failures to detect these effects, the percentage of variance explained by shared environment was low compared to the percentage of variance explained by genes. The low DZ correlations in overactive behavior suggest that the presence of shared environmental influences on this problem scale is unlikely, although its influence could not be tested formally, due to inclusion of dominant genetic effects in the model. Thus, genetic effects are the most important etiological factor in problem behavior in young children, although shared and non-shared environmental influences are also present.

The present finding of large genetic influences on behavior in 3-year-olds suggests that the results of other studies, which do not take genetic effects into account, may be misinterpreted. For example, Carter *et al.* (2001), found that children of mothers, who experience a depressive disorder in addition to anxious behavior, substance abuse or an eating

disorder, are at high risk for attachment insecurity. According to Carter *et al.* (2001) this attachment insecurity is caused by a less optimal interaction pattern of the depressed mothers. The results of the present study show that it is likely that the children of depressed mothers show similar symptoms because of the genes they received from their mothers or because of an interaction between these environmental and genetic factors.

An extensive literature exists on the presence of sex differences in psychopathology (for a review, see Rutter *et al.*, 2003). However, sex comparisons are often based on specialized clinic-groups rather than on representative general population samples. In the present study, sex differences were examined in a large general population (twin) sample. Sex differences were found on aggressive, overactive, and withdrawn behavior. On these three scales, the scores of the boys were higher than those of the girls. The findings on aggressive and overactive behavior are consistent with the perception that boys show more of these behaviors than girls. Indeed, similar differences in scores on aggressive behavior have been reported at ages 7, 10, and 12 (Van Beijsterveldt *et al.*, 2003). The finding of higher maternal scores in boys than girls on withdrawn behavior is unexpected, but the size of the effect does not seem to be of clinical significance.

Sex differences in relative importance of genetic and environmental influences on individual differences were found in aggressive and withdrawn behavior, but not in oppositional, overactive, and anxious/depressed behavior. Compared to boys, individual differences in aggressive behavior in girls were influenced more by genes, and less by shared environment. In contrast, compared to boys, individual differences in withdrawn behavior in girls were more influenced by shared environment and less by genes.

The results of the present analyses of parental data on 9689 3-year-old twin-pairs show that behavioral syndromes of early childhood are primarily influenced by genetic factors. Additive genetic factors account for the majority of these influences in all syndromes except for the parental ratings of overactive behavior, where dominant genetic factors were found to be more important. Non-shared and shared environmental effects also contribute to the expression of the common syndromes of early childhood problem behavior. The contribution of shared environment at this early age is plausible and expected. It will be interesting to determine whether shared environmental influences increase or decrease as the children age.

Parental reports were found to be influenced mainly by aspects of the child's behavior that are perceived commonly by the parents. However, parents also report on aspects of the child's behavior that are experienced uniquely by each parent. These unique aspects may arise because the child behaves differently towards both parents, or because both parents observe the child in different situations.

The finding of relatively large genetic contributions to early child psychopathology may facilitate gene finding expeditions. Specifically the finding that individual differences in behavioral problems are largely attributable to genetic influences increase the likelihood that chromosomal areas will be found to contribute to the phenotypic variance in linkage analyses. These results also have implications for diagnostics. The presence of heritable influences this early in life implies that the diagnosis of behavioral problems in young children should take into account a possible (early) history of behavioral problems in the parents. Needless to say, environmental factors cannot not be discarded, even in the presence of established familial history of behavioral problems.

ACKNOWLEDGMENTS

This work was supported by NWO Grant numbers 575-25-006, 575-25-012, and 904-57-94 (Boomsma, P.I.), and by NIMH Grant number MH58799 (Hudziak, P.I.).

REFERENCES

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4–18 and 1991 Profile*. Burlington, Vermont: Department of Psychiatry, University of Vermont.

Achenbach, T. M. (1992). *Manual for the Child Behavior Checklist/2–3 and 1992 Profile*. Burlington, Vermont: Department of Psychiatry, University of Vermont.

Achenbach, T. M., and Edelbrock, C. S. (1984). Psychopathology of childhood. *Annu. Rev. Psychol.* **35**:227–256.

Achenbach, T. M., Howell, C. T., McConaughy, S. H., and Stanger, C. (1995). Six-year predictors of problems in a national sample of children and youth: II. Signs of disturbance. *J. Am. Acad. Child Adolesc. Psychiatry* **34**(4):488–498.

Achenbach, T. M., and Rescorla, L. A. (2000). *Manual for ASEBA Preschool Forms & Profiles*. Burlington, Vermont: Department of Psychiatry, University of Vermont.

Boomsma, D. I. (1998). Twin registers in Europe: An overview. *Twin Res.* **1**(1):34–51.

Boomsma, D. I., Vink, J. M., Van Beijsterveldt, C. E. M., Geus, E. J. C., Beem, A. L., Mulder, E. J. C. M., Derks, E. M., Riese, H., Willemsen, A. H. M., Bartels, M., Van den Berg, M., Kupper, H. M., Polderman, J. C., Posthuma, D., Rietveld,

M. J. H., Stubbe, J. H., Knol, L. I., Stroet, T., and Van Baal, G. C. M. (2002). Netherlands Twin Register: a focus on longitudinal research. *Twin Res.* **5**(5):401–406.

Carey, G. (1986). Sibling imitation and contrast effects. *Behav. Genet.* **16**(3):319–341.

Carter, A. S., Garrity-Rokous, F. E. J. D., Chazan-Cohen, R., Little, C., and Briggs-Gowan, M. J. (2001). Maternal depression and comorbidity: Predicting early parenting, attachment security, and toddler social-emotional problems and competencies. *J. Am. Acad. Child Adolesc. Psychiatry* **40**(1):18–26.

Eaves, L. J. (1976). A model for sibling effects in man. *Heredity* **36**(2):205–214.

Falconer, D. S., and Mackay, T. F. C. (1996). *Introduction to Quantitative Genetics (*4th ed.) Harlow: Pearson Education Limited.

Faraone, S. V., and Tsuang, M. T. (1994). Measuring diagnostic accuracy in the absence of a ''gold standard''. *Am. J. Psychiatry* **151**:650–657.

Hewitt, J. K., Silberg, J. L., Neale, M. C., Eaves, L. J., and Erickson, M. (1992). The analysis of parental ratings of children's behavior using LISREL. *Behav. Genet.* **22**(3):293–317.

Hewitt, J. K., Silberg, J. L., Rutter, M., Simonoff, E., Meyer, J. M., Maes, H., Pickles, A., Neale, M. C., Loeber, R., Erickson, M. T., Kendler, K. S., Heath, A. C., Truett, K. R., Reynolds, C. A., and Eaves, L. J. (1997). Genetics and developmental psychopathology: 1. Phenotypic assessment in the Virginia Twin Study of Adolescent Behavioral Development. *J. Child Psychol. Psychiatry* **38**(8):943–963.

Hofstra, M. B., End e van der, J., and Verhulst, F. C. (2000). Continuity and change of psychopathology from childhood into adulthood: a 14-year follow-up study. *J. Am. Acad. Child Adolesc. Psychiatry* **39**(7):850–858.

Hudziak, J. J. (2001). The role of phenotypes (diagnoses) in genetic studies of attention-deficit/hyperactivity disorder and related child psychopathology. *Child Adolesc. Psychiatr. Clin. N. Am.* **10**(2):279–297.

Hudziak, J. J. (1997). Identification of phenotypes for molecular genetic studies of common childhood psychopathology. In K. Blum and E. Noble (Eds.). *Handbook of Psychiatric Genetics*. Boca Raton, Florida: CRC Press, pp. 201–217.

Jöreskog, K. G., and Sörbom, D. (1993). *New features in PRELIS 2*. Chicago: Scientific Software International, Inc.

Koot, H. M. (1993). *Problem behaviour in Dutch preschoolers*. Rotterdam: Doctoral dissertation, Erasmus University.

Koot, H. M., Oord van den, E. J. C. G., Verhulst, F. C., and Boomsma, D. I. (1997). Behavioral and emotional problems in young preschoolers: Cross-cultural testing of the validity of the Child Behavior Checklist/2-3. *J. Abnorm. Child Psychol.* **25**(3):183–196.

Neale, M. C. (1997). *Mx:Statistical modeling*. Richmond, Virginia:Department of psychiatry, Medical College of Virginia.

Neale, M. C., and Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. North Atlantic Treaty Organization. Scientific Affairs Division. Dordrecht: Kluwer Academic Publisher.

Neale, M. C., and Miller, M. B. (1997). The use of likelihood-based confidence intervals in genetic models. *Behav. Genet.* **27**(2):113–120.

Neale, M. C., and Stevenson, J. (1989). Rater Bias in the EASI temperament scales: a twin study. *J. Pers. Soc. Psychol.* **56**:446–455.

Plomin, R., DeFries, J. C., McClearn, G. E., and McGuffin, P. (2001). *Behavior Genetics* (4th ed.). New York: W. H. Freeman.

Rietveld, M. J. H., Hudziak, J. J., Bartels, M., Van Beijsterveldt, C. E. M., and Boomsma, D. I. (2004). Heritability of attention problems in children: Longitudinal results from a study of twins, age 3 to 12. *J. Child Psychol. Psychiatry* **45**(3):577–588.

Rietveld, M. J. H., Valk van der, J. C., Bongers, I. L., Stroet, T. M., Slagboom, P. E., and Boomsma, D. I. (2000). Zygosity diagnosis in young twins by parental report. *Twin Res.* **3**(3):134–141.

Rutter, M., Caspi, A., and Moffitt, T. E. (2003). Using sex differences in psychopathology to study causal mechanisms:unifying issues and research strategies. *J. Child Psychol. Psychiatry* **44**(8):1092–1115.

Rutter, M., Silberg, J., O'Connor, T., and Simonoff, E. (1999). Genetics and child psychiatry: I Advances in quantitative and molecular genetics. *J. Child Psychol. Psychiatry* **40**(1):3–18.

Shaw, D. S., Owens, E. B., Giovanelli, J., and Winslow, E. B. (2001). Infant and toddler pathways leading to early externalizing disorders. *J. Am. Acad. Child Adolesc. Psychiatry* **40**(1):36–43.

Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P. A. F., Bucholz, K. K., Dunne, M. P., Statham, D. J., and Marti, N. G. (1997). Modeling genetic and environmental influences in the etiology of conduct disorder: A study of 2,682 adult twin pairs. *J. Abnorm. Psychol.* **106**:266–279.

Simonoff, E., Pickles, A., Hervas, A., Silberg, J. L., Rutter, M., and Eaves, L. (1998). Genetic influences on childhood hyperactivity:contrast effects imply parental rating bias, not sibling interaction. *Psychol. Med.* **28**:825–837.

SPSS (2001). *SPSS for Windows Rel 11.0.1*. Chicago: SPSS.

Thomas, J. M., and Guskin, K. A. (2001). Disruptive behavior in children: what does it mean? *J. Am. Acad. Child Adolesc. Psychiatry* **40**(1):44–51.

Van Beijsterveldt, C. E. M., Bartels, M., Hudziak, J. J., and Boomsma, D. I. (2003). Causes of stability of aggression from early childhood to adolescence: A longitudinal genetic analysis in Dutch twins. *Behav. Genet.* **33**(5):591–605.

Van den Oord, E. J. C. G., Verhulst, F. C., and Boomsma, D. I. (1996). A genetic study of maternal and paternal ratings of problem behaviors in 3-year-old twins. *J. Abnorm. Psychol.* **105**(3):349–357.

Van der Valk, J. C., Oor dvan den, E. J. C. G., Verhulst, F. C., and Boomsma, D. I. (2001). Using parental ratings to study the etiology of 3-year-old twins' problem behaviors: Different views or rater bias? *J. Child Psychol. Psychiatry* **42**(7):921–931.

Van der Valk, J. C., Verhulst, F. C., Stroet, T. M., and Boomsma, D. I. (1998). Quantitative genetic analysis of internalizing and externalizing problems in a large sample of 3-year-old twins. *Twin Res.* **1**(1):25–33.

Edited by Irwin Waldman