Quantitative genetic analysis of Internalising and Externalising Problems in a large sample of 3-year-old twins

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For a quantitative genetic study of pre-school problem behaviours, we have collected data with the Child Behavior Checklist for 2 and 3-year-old children (CBCL 2/3). Questionnaires were completed by mothers of 3620 twin pairs: 633 monozygotic males, 581 dizygotic males, 695 monozygotic females, 519 dizygotic females and 1192 dizygotic opposite sex twin pairs. The genetic and environmental influences on the Externalising and Internalising Problem scales were estimated, simultaneously with sex differences and sibling interaction effects. Genetic factors explained most of the observed variance for both Externalising and Internalising Problems. Cooperative sibling interactions were found for Externalising Problems, indicating that twins reinforce each other's behaviour. Sex differences in genetic architecture were found for Externalising Problems. Genetic factors explained 75% of the variance in girls and 50% in boys. Shared environmental influences were only of importance in boys. For both problem scales, non-shared environmental factors accounted for 25 to 32% of the variance. The observed variances of Internalising Problems could be adequately explained by genetic and nonshared environmental factors, with genetic factors accounting for 68% of the variance.

Keywords: pre-school children, problem behaviour, Child Behavior Checklist, twins, behaviour genetics

Introduction

A number of studies indicate that roughly 10 to 15% of pre-school children show problem behaviours.[1,2] Despite the fact that problem behaviours in pre-school children may cause suffering for both the child and its family as well as put the child at risk for later malfunctioning, relatively few studies have looked at the aetiology of problem behaviours in pre-school children. Most problem behaviours in young children generally involve quantitative variations in behaviour that most children display to some degree. These continuous variations in behavioural problems are hypothesised to be caused by multiple genes and environmental influences. A better understanding of the aetiology of individual differences in pre-school problem behaviours is important, for it may guide clinical interventions and provide ideas for future research.

By carrying out quantitative genetic studies, the relative influences of genetic and environmental factors on the continuous variations in problem behaviours can be estimated. In order to determine what the genetic and environmental effects on variation in behaviour are, genetically informative subjects (such as twins) are needed. Their observed, ie phenotypic, variance can be partitioned into a genetic part, an environmental part that is shared between children growing up in the same family and an environmental part that is not shared with other family members (idiosyncratic experiences). A way to quantify pre-school children's problem behaviours is by asking their parents to score their children's behavioural and emotional problems on the Child Behavior Checklist for 2 and 3-year-old children (CBCL 2/3).[3] The CBCL 2/3 is a standardised questionnaire consisting of 99 problem items which are scored by the parents on a 3-point scale, based on the occurrence of the behaviour during the preceding two months: 0 if the problem item was not true of the child, 1 if the item was somewhat or sometimes true, and 2 if it was very true or often true. Using factor analysis different problem scales have been derived, which can be computed by summing the items belonging to that scale. For instance, the scale Aggressive Behaviour is composed of items like: demands must be met, disobedient, easily frustrated, jealous, fights, hits others, screams, moody, etc. Different scales can be

combined to form two broad band scales: Internalising Problems and Externalising Problems. The broad band scale Internalising Problems reflects anxious, depressed and withdrawn behaviours, while the broad band scale Externalising Problems is characterised by 'acting out'-oppositional and aggressive behaviours. Finally, a Total Problem Score can be computed by summing all 99 items.

Studies disentangling the influence of nature and nurture on the aetiology of differences among preschool children in problem behaviours are rare. We know of only two quantitative genetic studies of preschool children's problem behaviours, each using 3-year-old twins. Both studies employed the CBCL 2/3. Schmitz[4] studied 260 twin pairs from Colorado, and Van den Oord[5] used 1358 Dutch twin pairs. Overall, genetic influences appeared to be most important for explaining the observed phenotypic variance, while shared environmental influences had only a minor influence. For most scales, sex differences in the magnitude of the genetic and environmental influences were not found. A limitation however, especially of the first study, is the sample size used. To evaluate genetic models, which test do not only for genetic and environmental influences but also for possible sex differences, large sample sizes are needed.

Social interactions between siblings may also influence problem behaviours. Especially for behaviours which are easily observable for the other sibling, like aggressive behaviours, one can expect siblings to influence each other. Interactions can either be in a cooperative manner, through imitation or mutual reinforcement, or in a competitive manner, when the behaviour of one sibling evokes the opposite reaction in the other sibling[6]. The incorporation of sibling interaction into a model can dramatically change estimates of genetic factors and especially of shared environmental factors. For a sample of juvenile twins, aged between 8 and 16,[7] mothers' ratings for Externalising Behaviour were obtained. Because the pooled individual phenotypic variances of the monozygotic twins were greater than those of the dizygotic twins, a model with sibling interactions was tried as a way of illustrating typical sibling interaction.[8] Incorporating sibling interaction into the model caused the shared environmental factor to decrease from a large influence to zero. This indicated that the obtained shared environmental effect could totally be explained by sibling interactions. Boys proved to stimulate each other in showing Externalising Behaviour.

To enable a quantitative genetic study of preschool problem behaviours with a reasonable power to detect sex differences and social interactions between the twins, we supplemented the original Dutch sample of 1358 3-year-old twin pairs,[5] with an additional sample of 2658 3-year-old twin pairs. For all these twins, we collected the CBCL 2/3,[3] a standardised questionnaire, when the twins just reached their third birthday. With this sample of twin pairs, we estimated the genetic and environmental influences on the two broad band groupings of the CBCL 2/3 – Internalising Problems and Externalising Problems – while at the same time testing for possible sex differences and sibling interactions.

Methods

Subjects

This study is part of a project which examines the genetic and environmental influences on the development of problem behaviours in 3 to 7-year-old children. All participants were members of the Netherlands Twin Registry (NTR), kept by the Department of Psychonomics at the Free University in Amsterdam. Of all multiple births in The Netherlands, 40–50% are registered by the NTR.[9] For this study, all twins from the birth cohorts 1987 to 1991 were used. Questionnaires were mailed to 5103 families within three months of the twins' third birthday. After two to three months reminders were sent and four months after the initial mailing persistent non-responders were contacted by phone. A response rate of 78.7% was obtained, giving data on a total of 4016 families of twins; 60 twin pairs were excluded from the analyses because either one or both of the children had a disease or handicap that interfered severely with daily functioning. Another 183 twin pairs were excluded because the questionnaires of either one or both of the children were not filled in by the mother. Zygosity was determined for 686 twin pairs by either blood group polymorphisms or DNA analyses. For all other twin pairs, zygosity was determined by discriminant analysis, using questionnaire items which the parents had completed when the children were about 5 years of age. Parents were asked how much the twins resembled each other in hair colour, eye colour, facial structure, and whether they were ever mistaken for each other by family, friends or the parents themselves. The discriminant analysis resulted in a 92.71% correct classification, suggesting that at most 4% of the twins' zygosity was wrongly classified $((7.29\% \times (4016 - 686 - 1122 \text{ (dizygotic opposite sex twins not included in group with blood/DNA data)}))/4016)$. For 153 twin pairs zygosity could not be determined because the questionnaire on zygosity information was missing. These twin pairs were excluded from the study. This procedure left a sample of 633 monozygotic males (MZM), 581 dizygotic males (DZM), 695 monozygotic females

(MZF), 519 dizygotic females (DZF) and 1192 dizygotic opposite sex (DOS). Children were rated by both parents in 45% of cases. In this paper we report maternal ratings.

Measures

The CBCL 2/3 is a standardised questionnaire, developed for parents to score the behavioural and emotional problems of their 2 and 3-year-old children.[3] It was modelled after a similar questionnaire for children of 4–18 years of age. Dutch syndrome scales for the CBCL 2/3 were derived by exploratory, followed by confirmatory, factor analyses across three independent samples: 426 children referred to the mental health services, 420 children from the general population and 1306 twin pairs from the present study.[10] Koot *et al* showed that the Dutch syndrome scales are comparable to those developed by Achenbach.[3] The Dutch scale Oppositional has a high correlation with the American scale Aggressive Behaviour (0.94), while the Dutch scale Aggressive has a high correlation with the American scales Destructive Behaviour (0.82) and Aggressive Behaviour (0.80). All other scales obtained similar names: correlation between Dutch Withdrawn/Depressed and American Withdrawn was 0.88, Dutch Anxious and American Anxious/Depressed was 0.84, Dutch

Internalising and American Internalising was 0.90 and Dutch Externalising and American Externalising was 0.97. All these correlations were significantly higher than those between any other combinations of Dutch and American syndrome scales (except Internalising and Externalising). In contrast to the Dutch version, there is no Overactive scale in the American version.

The syndrome scales used in this study were composed according to the Dutch version. The broad band scale Internalising Problems was composed of the items of the Anxious and Withdrawn/Depressed subscales. (In contrast to the composition of the Anxious scale reported by Koot *et al*,[10] item 32 was not included because it lowered Cronbach's α.[11]) The broad band scale Externalising Problems was composed of the items of the Aggressive, Oppositional and Overactive subscales.

The data were subjected to square-root transformation before the analyses were performed, because most children showed no or just little problem behaviours, causing a skewed distribution. The distribution of Externalising Problems and Internalising Problems after transformation is shown in Figure 1 and Figure 2, respectively.

For the scale Internalising Problems, the kurtosis of the total twin sample was −0.415 (range of all different zygosity by sex groups −0.568–0.021) and the skewness was −0.101 (range of all different
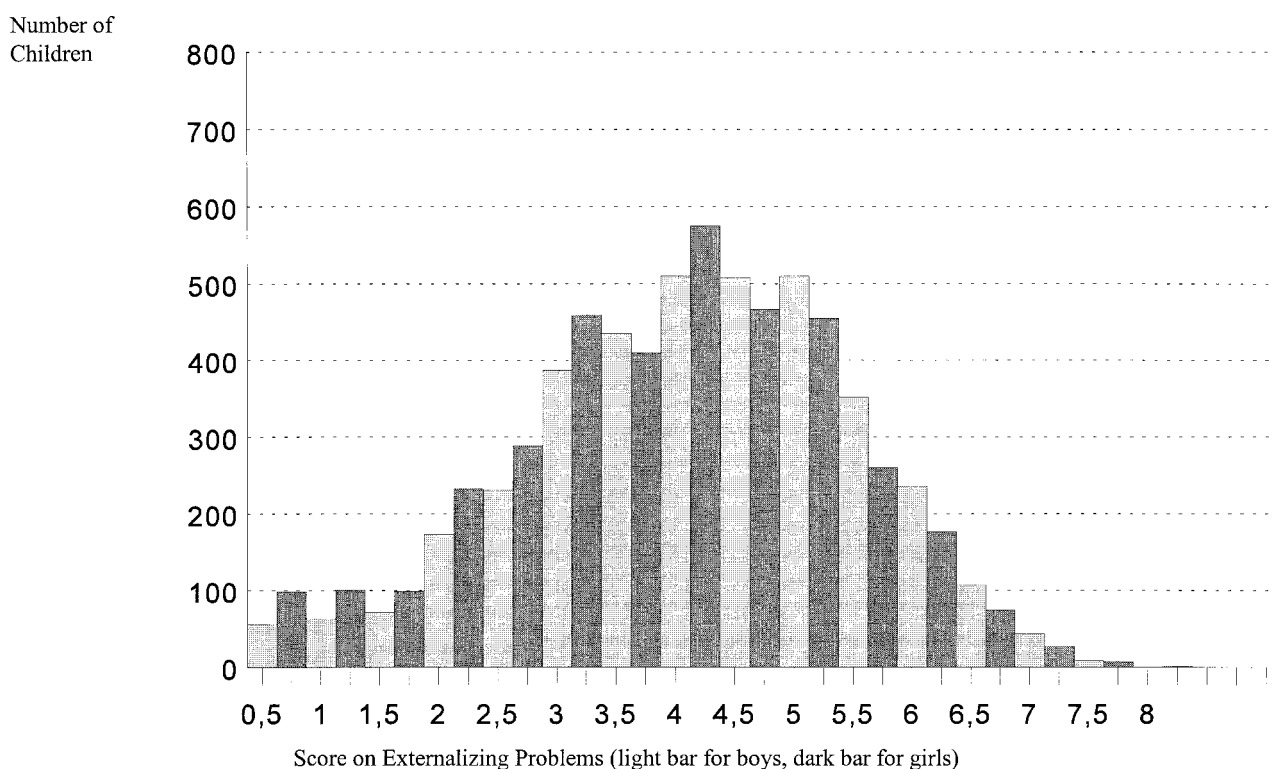
Figure 1 Distribution of the broad band scale Externalising Problems after square-root transformation

Number of
Children
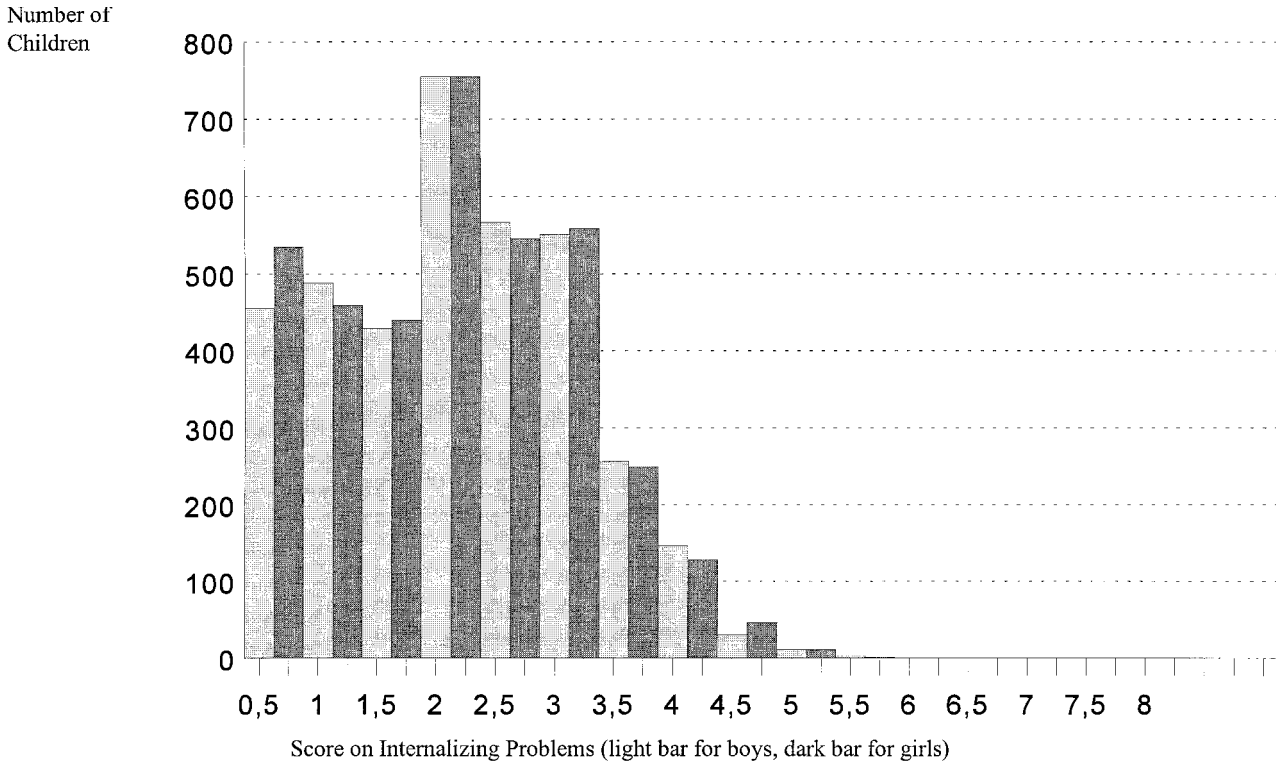
Figure 2 Distribution of the broad band scale Internalising Problems after square-root transformation

zygosity by sex groups −0.324–0.047). The scale Externalising Problems showed a smaller kurtosis for the total twin sample of −0.038 (range of all different zygosity by sex groups −0.404–0.007) and a slightly larger skewness of −0.326 (range of all different zygosity by sex groups −0.404–0.197). All absolute values of kurtosis and skewness were smaller than 0.6, suggesting that after transformation the distribution of both scales approached normality.
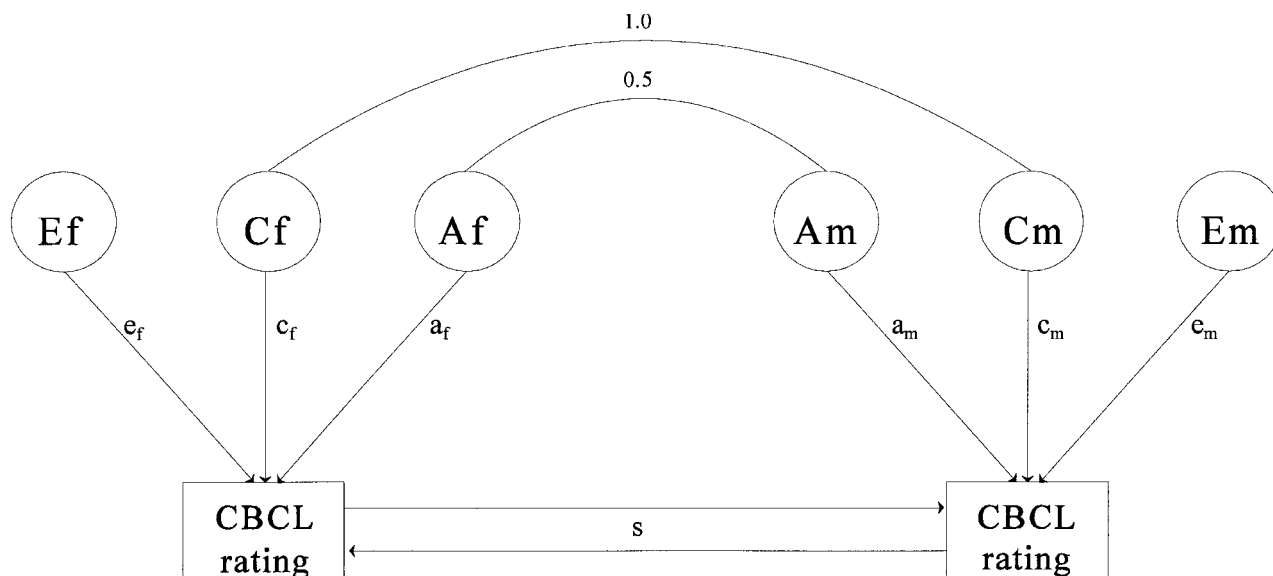
Models

A twin model, composed to test for genetic and environmental influences on the CBCL 2/3 broad band scales, was fitted to the data. Monozygotic twins, who are genetically identical, were compared with dizygotic twins, who share on average 50% of their segregating genes. Both types of twins grow up in a family; they are assumed to share the same kind of familial environment. By comparing the similarity between the monozygotic twins with the similarity between the dizygotic twins, identification of the model to estimate the contributions of genotype (A), shared environment (C), and nonshared environment (E) is achieved (ACE model). If the monozygotic twins resemble each other to the same degree as the dizygotic twins, only environmental influ-

ences can be of importance. However, when the monozygotic twins resemble each other more than the dizygotic twins, genetic factors are supposed to be of importance, since the only difference between the two groups is in genetic relatedness.

In order to estimate the genetic and environmental influences on pre-school problem behaviours, while testing for possible sex differences and sibling interactions, the model shown in Figure 3 was fitted to the observed variance–covariance matrices of the five different twin groups (MZM, DZM, MZF, DZF, DOS). Monozygotic twin covariances and dizygotic twin covariances are compared, assuming a correlation between the twins' shared environmental influences of 1.0, regardless of twin type, and a genotypic correlation of 1.0 for monozygotic twins and 0.5 for dizygotic twins. The model decomposes the observed variance of the maternal ratings into three latent factors that may have a different influence on females (ie A_f, C_f, E_f) and on males (ie A_m, C_m, E_m). Sibling interaction is incorporated in the model by allowing the behaviour of the twins to influence each other(s).

Model fitting

Structural equation modelling was used, in which the observed variance–covariance matrices of the

Figure 3 ACE model allowing for sex differences and sibling interactions

five different twin groups are compared with the expected variance–covariance matrices of the theoretical model. A good model describes the observed variance–covariance matrices to such an extent that the residual variance–covariance matrices are trivially small. In this case one can say that the theoretical model describes the observed data adequately, which is also indicated by the χ^2 test statistic. So the χ^2 provides a test of whether the residual differences between the observed and the expected variance–covariance matrices converge in probability to zero as the sample size approaches infinity.[12] However, because theoretical models are never able to describe the real world perfectly, any model can be rejected if the sample size is large enough. Because of this effect of sample size, a poor fit based on a small sample size may result in a model being accepted, whereas a good fit based on a large sample size may result in a model being rejected.[13] Using a large sample of twins to test the fit of the model to the observed variance–covariance matrices, we have not only taken the χ^2 test statistic as a measure of how well the model described the observed data, but also looked at the differences between the observed and predicted variance–covariance matrices.

Using Mx, a structural equation modelling program,[14] we first fitted an ACE model to the observed data, that allowed for sex differences and sibling interactions. Next we tested whether a model without either sibling interactions or without sex differences or without both interactions and sex differences fitted the observed data as well as the full ACE model. This test was accomplished by subtracting the model's χ^2 from the χ^2 of a less constrained

model. The degrees of freedom for this test statistic are the number of parameters in the model, subtracted from the number of parameters in the less constrained model. The most simplified model was then retained to analyse the causes of variation in pre-school problem behaviours.

Results

Table 1 gives the untransformed mean problem scores and standard deviations of the twin sample and those of a Dutch community sample[11] of 420 singleton children. For all CBCL 2/3 broad band and subscales, the two samples showed comparable means and standard deviations.

The sample sizes of the different zygosity-by-sex groups and their means and standard deviations for oldest and youngest twins (male and female twins in the opposite sex group) are given in Table 2. The scales were subjected to square-root transformation. There were no mean differences between the sexes

Table 1 Means and standard deviations of community sample and twin sample for Dutch CBCL/2–3 broad band and subscales

CBCL/2–3 profiles	community sample	twins
sample size	420	3773×2
Externalising scale	17.0 (9.2)	16.0 (10.1)
Aggressive	3.2 (2.6)	3.3 (2.8)
Oppositional	10.7 (6.0)	10.0 (6.6)
Overactive	3.1 (2.4)	2.7 (2.2)
Internalising scale	4.4 (4.0)	4.6 (4.1)
Anxious	3.3 (2.9)	3.5 (3.1)
Withdrawn/Depressed	1.1 (1.8)	1.1 (1.6)

Table 2 Sample size, means and standard deviations (oldest and youngest twin) for each zygosity-by-sex group for CBCL/2–3 and broad band scales. Order of DOS twins: male, female

CBCL/2–3 profiles	MZM	DZM	MZF	DZF	DOS
twin pairs	633	581	695	519	1192
broad band scales:					
Externalising Problems	4.03 (1.37)	3.97 (1.24)	3.70 (1.43)	3.73 (1.35)	3.76 (1.36)
	3.92 (1.38)	3.83 (1.33)	3.61 (1.45)	3.54 (1.36)	3.47 (1.38)
Internalising Problems	1.93 (1.00)	1.94 (0.99)	1.96 (1.06)	1.96 (1.04)	1.86 (1.04)
	1.84 (1.04)	1.77 (1.10)	1.86 (1.12)	1.88 (1.01)	1.68 (1.06)

MZM/F = monozygotic males/females, DZM/F = dizygotic males/females, DOS = dizygotic opposite sex.
Note. Scales have been subjected to square-root transformation.

for the broad band scale Internalising Problems, but for the scale Externalising Problems females obtained lower mean scores than males. For the Externalising scale, the standard deviations shown by the monozygotic twins were larger than the standard deviations shown by the dizygotic twins, both for males and females.

Table 3 shows this result in more detail by giving the variance–covariance matrices of the observed data, for both broad band scales per zygosity-by-sex group. For Externalising Problems, monozygotic twins showed larger variances and covariances than dizygotic twins, both for males and females. A larger variance of monozygotic twins than for dizygotic twins indicates the possibility of sibling interaction. Cooperative interactions between siblings causes the variances of the monozygotic twins, who are genetically identical, to be larger than the variances of the dizygotic twins, who share on average half of their segregating genes.[8]

The Internalising scale did not show these systematic differences in variances between monozygotic and dizygotic twins, so the siblings probably do not influence each other with respect to internalising behaviours.

The correlations between the twins, given per zygosity-by-sex group and for each broad band scale, are shown in Table 4. For the Externalising scale, the

correlation between the monozygotic males was higher than the correlation between the dizygotic males. However, it did not approach twice the size of the correlation between the dizygotic males. This suggests that apart from genetic influences, shared and non-shared environmental influences are also important for explaining the males' externalising behaviours. The correlation between the female twins showed the same pattern, suggesting that also for female twins genetic influences, shared environmental influences and non-shared environmental influences will be necessary to explain their externalising behaviours.

For the Internalising scale, the correlations between the monozygotic males were almost twice the size of the correlations between the dizygotic males. In order to explain internalising behaviors of the males, we expect genetic and non-shared environmental influences to be important, but not shared environmental influences. Again, female twin correlations showed comparable results, suggesting that also for the female twins genetic and non-shared environmental influences will be important.

For both scales, correlations of dizygotic opposite-sex twins were of the same size as those of same-sex twins. This suggests that the same genes are expressed in males as in females.

We fitted a twin model with genetic, shared environmental and non-shared environmental factors to the observed data. The model allowed for possible sex differences and sibling interactions. The fit of the full model and its submodels are given in Table 5.

Table 3 Observed variance–covariance matrix for Externalising Problems and Internalising Problems per zygosity-by-sex group

zygosity	Externalising Problems observed (co)variance		Internalising Problems observed (co)variance	
MZM	1.8748		1.0000	
	1.4746	1.8914	0.6652	1.0839
DZM	1.5396		0.9730	
	0.9542	1.7731	0.4021	1.2103
MZF	2.0419		1.1256	
	1.6715	2.0960	0.8433	1.2517
DZF	1.8260		1.0816	
	0.9502	1.8457	0.3911	1.0245
DOS	1.8533		1.0886	
	0.9506	1.9115	0.3948	1.1278

MZM/F = monozygotic males/females, DZM/F = dizygotic males/females, DOS = dizygotic opposite sex.

Table 4 Twin correlations per zygosity by sex group for CBCL/2–3 broad band scales

CBCL/2–3 profiles	MZM	DZM	MZF	DZF	DOS
broad band scales:					
Externalising Problems	0.78	0.58	0.81	0.52	0.51
Internalising Problems	0.64	0.37	0.71	0.37	0.36

MZM/F = monozygotic males/females, DZM/F = dizygotic males/females, DOS = dizygotic opposite sex.

Table 5 Fit of the different models for CBCL 2/3 Externalising and Internalising Problems

	Externalising Problems			Internalising Problems		
Model	χ^2	df	P	χ^2	df	P
ACE+sex diffs.+sibl.int.	**9.716**	**8**	**0.286**	17.938	8	0.022
ACE+sex diffs.	14.313	9	0.112	18.549	9	0.029
ACE+sibl. int.	23.970	11	0.013	22.182	11	0.023
ACE	28.094	12	0.005	22.491	12	0.032
AE+sex diffs.+sibl. int.	18.507	10	0.047	18.262	10	0.051
AE+sex diffs.	96.063	11	0.000	21.028	11	0.033
AE+sibl. int.	23.970	12	0.021	22.182	12	0.036
AE	101.321	13	0.000	**24.735**	**13**	**0.025**

Note: Sex diffs. = sex differences; sibl. int. = sibling interactions.

For the Externalising scale, the full model described the observed variance–covariance matrices adequately and better than the more parsimonious models. The χ^2 of the full model proved to have a good fit with a P-value of 0.29. All residual variance–covariance matrices were trivially small, indicating that almost all of the observed variances and covariances were explained by the theoretical model.

The different model fits of the Internalising scale showed that the submodel with only genetic influences and non-shared environmental influences described the observed data adequately and not significantly worse than a more complex model. The residual variance–covariance matrices were trivially small, indicating that although the model's obtained P-value was low (0.03), it described the observed data satisfactorily.

The percentage of variance explained by the genetic, shared and non-shared environmental factors is given in Table 6. Because the model of Externalising Problems contained sex differences and sibling interactions, the estimates for monozygotic males and females and dizygotic males and females differed.[8] The path allowing for sibling interactions was constrained to be equal for male and female twins (which did not lead to a worse fit than the model in which it differed for males and females). For males, genetic factors explained half of the percentage of variance. Shared and non-shared environmental factors had almost equal influences,

explaining 22–29% of the variance. For females, shared environmental factors were nonexistent. Most of their variance 74–75%, was explained by genetic factors, while the non-shared environmental factors explained the rest of the variance.

The best fitting model for Internalising Problems only allowed for genetic and non-shared environmental factors, without sex differences or sibling interactions. The genetic factors explained 68% of the variance, while the non-shared environmental factors explained 32% of the variance. Genetic factors were, for males and females, more important in explaining the observed data.

Discussion

In the present study, the CBCL 2/3 questionnaire was used to assess the genetic and environmental influences on two broad band scales Externalising Problems and Internalising Problems, scored for 3620 twin pairs. For both scales, genetic factors explained most of the observed variances. Non-shared environmental factors accounted for 25–32%. These results are consistent with the estimates Van den Oord[5] found for the previously collected smaller sample of 1358 Dutch twin pairs. However, in contrast to the former study,[5] using an effective sample size of 3620 twins we now also found sibling interactions and sex differences in the estimates of the scale Externalising Problems. Genetic factors accounted for 74–75% of

Table 6 Percentage of variance explained by genetic, shared and non-shared environmental factors for best-fitting models and path estimate for sibling interaction

		environment		path estimate
CBCL 2/3 scales	genetic %	shared %	non-shared %	sibl. interac.
Externalising Problems				
monozygotic males	51	22	27	0.102
dizygotic males	49	22	29	0.102
monozygotic females	75	–	25	0.102
dizygotic females	74	–	26	0.102
Internalising Problems	68	–	32	–

the variance for females, versus 49–51% for males. Shared environmental influences were present only in males, explaining 22% of the variance. Overall, these results indicate that differences in externalising problems in pre-school children are caused predominantly by genetic differences. Although genetic influences are stronger for females than for males, the same genes seem to be responsible for this influence in both sexes, as was shown by the similarity between the correlations of same sex and opposite sex dizygotic twin pairs. The finding that shared environmental influences are present only in males but not in females is difficult to interpret without the help of further studies. It could be an indication that boys, even as young as 3 years of age, are more sensitive to the morals and values the family attaches to externalising behaviours, or it could indicate that families are more directive and controlling over externalising behaviours in young boys.

For the broad band scale Internalising Problems we did not find any evidence of sex differences or the effects of shared environment. All the observed variances of this scale could be explained by genetic and non-shared environmental factors, with genetic factors accounting for 68% of the variance. Finding this simple model in such a large sample of twins is strong evidence that Internalising Problems in 3-year-old children, regardless of sex, are largely influenced by genes and, to a lesser degree, by idiosyncratic experiences that are not shared by other children in the family. This result is in contrast with the estimates reported by Schmitz.[4] In a small sample of 3-year-old twins from Colorado, Schmitz[4] found that the scale Internalising Problems was more strongly influenced by shared and non-shared environmental factors than by genetic factors.

Using the large effective sample size of 3620 twins, we now also found evidence of sibling interactions for the scale Externalising Problems. The interactions proved to be cooperative, with twins reinforcing each other's behaviour. We are not aware of any other study investigating sibling interactions in pre-school children. However, the results are consistent with the interactions Hewitt *et al* found for a sample of 8 to 16-year-old twins. These school-aged and adolescent children also reinforced each other's externalising behaviours.[8] For the scale Internalising Problems no sibling interactions were found. It appears that pre-school children, who exhibit internalising problems such as anxiety and depression, do not influence their twin in showing either the same or opposite behaviours.

Non-shared environmental influences, apart from genetic influences, were the only other factor of importance for females, accounting in both broad band scales for 25–32% of the observed variance.

For males, the non-shared environmental factor was just as important as it was for females. This result indicates that, for both scales and for both sexes, idiosyncratic experiences are of importance in the rate of problem behaviours shown by pre-school children. However, errors of measurement also come into the estimate of the non-shared environmental factor. Perhaps by including the ratings of other raters, such as fathers and carers other than parents, possible errors of measurement can be reduced, thereby decreasing the estimates of the non-shared environmental factor. Rater bias – another possible error of measurement – caused by raters consistently scoring their children as having either more or fewer problems, was probably not very large in this data set. If it had occurred, estimates of the shared environmental factors would have been increased. Considering that we found evidence of shared environmental factors for the scale Externalising Problems only in males, and not for females or for the scale Internalising Problems, rater bias probably did not play an important role. Van den Oord *et al*[5] addressed this issue in the sample of 1358 Dutch twin pairs and found that rater bias did not affect the estimates of genetic and environmental factors. Rater bias therefore does not seem to be a large problem in this sample.

Fitting the most simplified model for the scale Internalising Problems, the obtained *P*-value of the χ^2 was low. Nevertheless, the residual variance–covariance matrices were trivially small. So probably the poor fit of this model was caused by the large sample size of twin pairs used.[13].

The model used assumed there were no interactions between genes and the environment. However, one cannot be certain this is true in real life. It could be that the kind of environmental influences the child experiences depends on the genotype of the child itself. As Campbell[1] suggests in her review article of recent studies 'it seems likely that biological propensities in the child interact with salient aspects of the care-giving environment to produce either adaptive or maladaptive outcomes…' (p. 141). If this interaction occurs with non-shared environmental influences, the estimate of the non-shared environmental factor increases. Probably it was not so in this study, because the estimate of the non-shared environmental factor was quite small between 25–32%. If the kind of shared environmental influences the child experiences depends on its genotype, the estimate of the genetic factor will increase. Because we found estimates of quite large genetic influences, interactions between shared environment and genotype could have inflated the genetic estimate. However, in case the kind of shared environmental influences that the child experiences depends on its genotype, then the influence of this

interaction actually also belongs to the estimate of the genetic factor.

The estimates found are not applicable to the individual. Quantitative genetic studies estimate average differences between individuals in a certain population. For other populations or for specific individuals different estimates might be applicable. This study used a nonclinical sample of twin pairs, showing problem behaviours in the normal range. Whether the results also apply to a clinical population, showing problem behaviours in an extreme range, will have to be tested by further studies.

Acknowledgements

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