The Association Between Internalizing and Externalizing Behavior in Childhood and Early Adolescence: Genetic or Environmental Common Influences?

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This study analyzed the genetic and environmental influences on internalizing and external-izing behavior problems and the nature of their cooccurrence in a national Norwegian twin sample. The sample comprised 526 identical and 389 fraternal same-sexed twin pairs from five birth cohorts, aged 5-6, 8-9, 12-13, 13-14, and 14-15 years. Behavior problems were assessed by parental ratings on the Child Behavior Checklist. A model of additive genetic, shared, and nonshared environmental influences was fitted to both internalizing and exter-nalizing behavior in four sex and age groups. The considerable covariance, $r = .51$ to $.58$, between these traits is accounted for mainly by common environmental components; this effect was most marked in the 5 to 9-year olds. Concordance rates for children scoring above 1 standard deviation from the total sample mean on the internalizing scale only, externalizing scale only, or both indicated stronger genetic influences for internalizing or externalizing problems only than for combined problems.

KEY WORDS: Comorbidity; internalizing behavior; externalizing behavior; twins.

The cooccurrence of emotional or internalizing problems and conduct or externalizing problems is substantial in epidemiological samples (Gould, Bird, & Jaramillo, 1993; Rose, Rose, & Feldman, 1989; Verhulst & van der Ende, 1993; Weiss & Catron, 1994). Corresponding findings of comorbidity be-tween conduct disorder and depression and between conduct and anxiety disorders are consistently re-ported from both clinical (Harrington, Fudge, Rutter, Pickles, & Hill, 1991; Puig-Antich, 1982) and epidemiological samples (see Zoccolillo, 1992, for re-view).

Several factors could contribute to comorbidity or cooccurrence between distinct conditions at rates exeeding chance expectations in the same individual (Caron & Rutter, 1991). This could be due to shared risk factors, to an overlap in risk factors acting via different patterns, to the comorbidity pattern being a single diagnostic entity, or to one disorder increas-ing the risk for the other. The above chance cooc-currence could also be an artifact due to referral bias in clinical samples, overlapping criteria for assess-ment of the conditions considered to be distinct, or subdivision of syndromes which are alternative mani-festations of the same underlying cause.

Utilizing twin data, several aspects of shared risk factors between comorbid traits can be investigated (Kendler, Heath, Martin, & Eaves, 1987). The twin de-sign allows the separation of the factors influencing cooc-currence into possible shared familial environmental influences, genetic influences, or individual life experi-ences, all of which may influence both conditions.

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Studies have indicated that children with comorbid externalizing and internalizing problems are relatively similar to pure externalizing children in terms of antisocial outcome (Graham & Rutter, 1973; Harrington et al., 1991) and social impairment (Cole & Carpentieri, 1990). Puigh-Antich et al. (1989) demonstrated less familial loading in depressive cases with comorbid conduct problems compared to pure major depressive cases, suggesting that that associated conduct disorder might be a clinical indicator of the nongenetic nature of major depression in some children. Weiss and Catron (1994) have assessed the comorbidity of depression and aggression and concluded that this may well be due to the same factors as the associated internalizing-externalizing cooccurrence. Taken together, the above studies indicate that the internalizing-externalizing cooccurrence is influenced by factors similar to those underlying pure externalizing conditions.

The present study addressed the genetic and environmental influences on internalizing and externalizing behavior problems and the nature of their covariance in a national sample of five birth cohorts of young twins. Age and gender differences in genetic and environmental influences were investigated. The main analysis concerned internalizing and externalizing scores as continuous trait measures. In the final analyses, concordance and heritability of group membership for internalizing and externalizing problems were investigated in groups with pure internalizing or externalizing or comorbid problems.

METHODS

Sample

Five national cohorts of same-sex twins, born in 1977-1979, 1983, and 1986, were drawn from the Norwegian Medical Birth Register. All pairs with both twins alive were selected (1,529 pairs). Parents of 915 pairs (59.8% of the total) gave information on behavior problems. The respondents were mothers only for 77.3% of the children and fathers only for 6,6%. For 16.1% a joint response was given from mothers and fathers (in a few cases mother combined with mothers' new spouses or other close relatives).

Representativeness of Sample

Respondents and nonrespondents could be compared on data available from the Norwegian Birth Register. This revealed a similar sex distribution (47.5% males, 52.5% females). The Central Population Register has classified place of living on a 1 to 7-point scale based on three criteria: the industrial structure of the area, population density, and availability of public services. No significant difference in place of living was found when respondents and nonrespondents were compared by this classification. The duration of pregnancy was significantly lower in the nonresponder group, although the difference was small (263.9 vs. 265.3 days, $p < .05$). There was no significant birth weight difference. No information on socioeconomic status (SES) was available for the nonrespondents. Fathers in responder families showed a significantly higher SES when compared to the general Norwegian male population in the Norwegian standard classification of SES ($\chi^2 = 61.9$, $df = 4, p < .001$). Comparing twin mothers with the general Norwegian female population showed a similar difference in SES ($\chi^2 = 32.2$, $df = 4, p < .001$). Comparison of the present twin sample and a general population sample showed similar levels of externalizing problems and a small age-dependent difference in level of internalizing problems with slightly higher levels in younger and lower levels in older twins compared to the general population sample (Gjone & Nøvik, 1995).

Procedure and Measures

Behavioral asessment. The families were contacted by mail and one parent was asked to reply to the Child Behavior Checklist (CBCL; Achenbach, 1991; Achenbach & Edelbrock, 1983) for both twins. Further details on data collection procedure are provided elsewhere (Gjone & Nøvik, 1995). The CBCL contains a social competence section and a behavioral problem section with 120 items. These items are scored on a 3-point scale. The 1991 version of the CBCL generates eight syndrome scale scores across sex and age from 4 to 18 years. The syndrome scales designated as Withdrawn, Somatic Complaints and Anxious/Depressed are grouped under the name Internalizing. The Internalizing score is the sum of the scores on the three Internalizing scales. The scales designated as Delinquent Behavior and Aggressive Behavior are grouped under the name Externalizing.

The Externalizing score is the sum of the scores on the two Externalizing scales. A satisfactory reliability and validity of the CBCL has been demonstrated in both clinical and nonclinical populations and across cultures (Verhulst & Koot, 1992). Studies have shown significant relationships between CBCL scales and one or more *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.) (DSM-III; American Psychiatric Association, 1980) diagnoses but with no one-to-one relationship with separate subscales (Edelbrock & Costello, 1988; Gould et al, 1993). Therefore the two second-order Internalizing and Externalizing scales are used to provide measures of emotional/neurotic/depressive problems and antisocial/conduct problems, rather than the separate subscales.

Zygosity. The twins were identified as identical (MZ) or fraternal (DZ) by a zygosity questionnaire (Cohen, Dibble, Grawe, & Pollin, 1973). Several studies have demonstrated 90% to 98% agreement between questionnaire measures and blood typing for zygosity determination (Bønnelykke, Hauge, Holm, Kristoffersen, & Gurtler, 1989; Cohen et al, 1973; Nichols & Bilbro, 1966). This questionnaire has a range of scores from 0 to 20. The cut-off between 10 and 11 was chosen from the frequency distribution, but with two modifications: If hair or eye color was different, the twins were classified as DZ even if scores were above or equal to 11. By this procedure 57.5% were assigned to the MZ group and 42.5% to the DZ group. All twins who were reported to be DZ by blood testing (not further specified in the questionnaire) scored equal to or less than 11 (26 pairs) and all those who were reported to be MZ scored equal to or above 12 (37 pairs). This was in good agreement with the cut-off based on the frequency distribution.

Data Analysis

The twins were separated by age into a group of younger twins aged 5 to 9 and a group of older twins aged 12 to 15. Maternal, paternal and combined parental, responses were pooled together for these analyses in order to provide a maximum sample size.

Estimates of Heritability, Shared Environment and Nonshared Environment. These were obtained for younger and older twins, boys and girls separately, using structural equation modeling with Lisrel 7 (Jöreskog & Sörbom, 1989). Univariate three- and two-parameter models were fitted to the data, consisting of (1) additive genetic influence (A), nonshared environmental influences (E), and shared environmental influences (C) (ACE model); and (2) A and E (AE model) (Neale & Cardon, 1992). Models were rejected when the observed variance/covariance structure differed significantly from that expected from the model, and goodness of fit (GFI) was assessed by the maximum-likelihood chi square with $p < .05$.

The *additive genetic* component denotes the influence of alleles at several gene loci acting in an additive manner. The *nonshared environmental* component denotes the impact of all environmental factors influencing only one twin of a pair, such as individual experiences at school, with peers, and in interactions with parents, or various life events affecting only one of the twins. The nonshared environment factor also includes measurement error. The *shared environmental* component denotes life experiences affecting both twins similarly; these could include socioeconomic level and parenting. The shared environment factor also includes rater bias. Thus nonshared and shared environment were identified by the way they influenced MZ and DZ intrapair differences. The factors included in these constructs could overlap, i.e., parenting can influence twins in a similar manner (acting as a shared environmental influence) or elicit different interaction patterns with each twin of a pair (acting as a nonshared environmental influence).

A bivariate independent pathway model (Kendler et al., 1987; Neale & Cardon, 1992) was then applied to the internalizing/externalizing variance/covariance matrix separately for each sex and age group. This model identified the genetic and shared and nonshared environmental components of variance in common between internalizing and externalizing behavior, and the additional genetic and nonshared environmental components of variance specific to each trait. Standardizing these maximum likelihood parameter estimates for internalizing and for externalizing behavior yielded the relative impact of the genetic and environmental variance component in the model for each trait. The full model is shown in Fig. 1.

To address the issue of differences in genetic influences on internalizing and externalizing behavior contingent upon comorbidity, probandwise concordance rates were computed. As there are no Norwegian norms for the CBCL available, a statistical
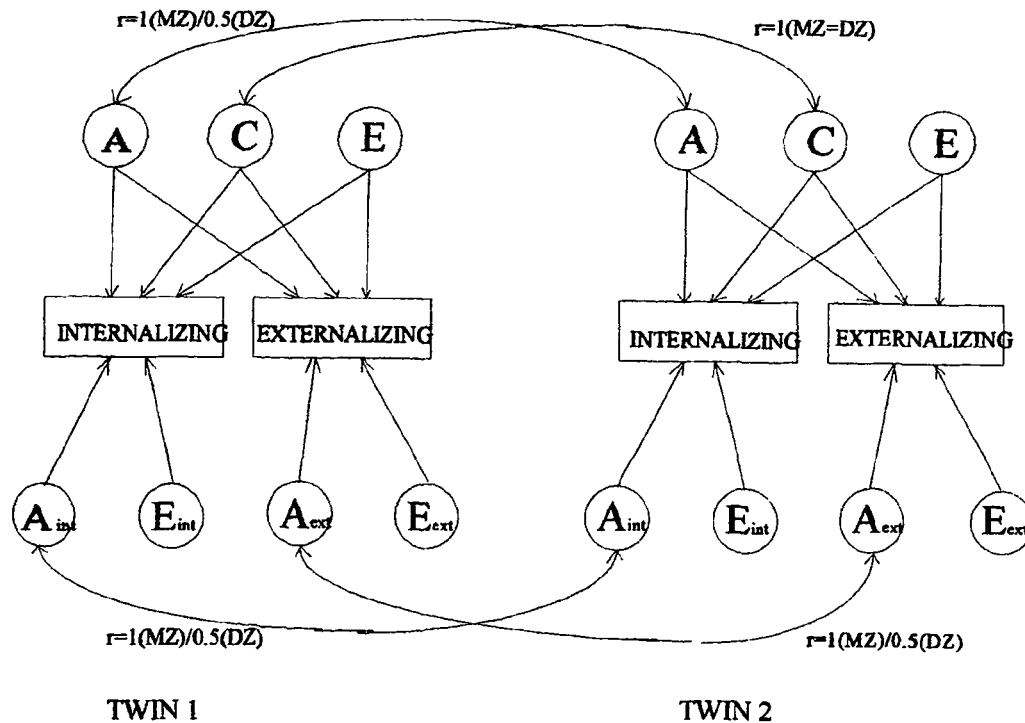
Fig. 1. Independent pathway model for internalizing (int) and externalizing (ext) behavior. A = additive genetic influence; C = nonshared environmental influences; E = shared environmental influences; MZ = identical twins; DZ = fraternal twins.

cut-off equal to or above 1 standard deviation from the total sample mean was chosen to provide a sufficient group of twins for these analyses. Pairs could be classed as one or both twins showing internalizing behavior only ("pure internalizers"), externalizing behavior only ("pure externalizers"), or both behaviors ("comorbid cases"). Probandwise concordance is the number of affected twins in concordant pairs divided by the total number of affected twins and expresses the morbidity risk for cotwins of affected probands (Plomin, DeFries, & McLearn, 1990). Extreme group heritability was estimated using the De-Fries and Fulker regression method (DeFries & Fulker, 1985, 1988). The relative genetic influence or heritability was estimated by the following regression equation: $C = B_1P + B_2R + A$, where C is the cotwin score, P is the proband score, and R is the coefficient of relationship (1 for MZ and 0.5 for DZ twins). With appropriate transformations, the partial regression of the cotwin score on the coeficcient of relationship was a direct estimate of heritability of group membership.

Data Transformation. The distribution of scores on the Internalizing and Externalizing behavior prob-

lem scales were considerably skewed and the structural equation models carried strong normal distribution assumptions. This skewness could reflect underlying genetic or environmental influences on the distribution as well as scalar artifacts (Falconer, 1989). Log transformation of data brought the distribution closer to normality and hence reduced these effects. The transformation was carried out after adding 1 to the variable value. The log transformed data were used in all model fitting analyses in order to satisfy the statistical assumptions.

RESULTS

Table I shows the level of internalizing and externalizing behavior for younger (5 to 9 years) and older (12 to 15 years) boys and girls. For externalizing behavior, a 2×2 analysis of variance (ANOVA) demonstrated significantly higher scores in younger children ($F = 80.344$, $p < .001$) and in boys ($F = 13.005$, $p < .001$). The level of internalizing behavior was higher in girls ($F = 12.59$, $p < .001$), with no significant age influence.

Table I. Internalizing and Externalizing Behavior by Sex and Age Group

	Internalizing behavior mean (SD)	Externalizing behavior mean (SD)
Boys, ages 5 to 9, $n = 380$	3.8 (3.8)	8.1 (6.9)
Girls, ages 5 to 9, $n = 400$	4.2 (4.3)	7.0 (6.5)
Boys, ages 12 to 15, $n = 488$	3.5 (3.8)	5.4 (6.6)
Girls, ages 12 to 15, $n = 562$	4.4 (4.9)	4.4 (5.3)

The overall correlations between log-internalizing and log-externalizing behavior were .52, .54, .51, and .58 for younger boys, younger girls, older boys, and older girls, respectively. Variances/covariances and correlations for log-internalizing and log-externalizing behavior by sex, age, and zygosity are presented in Table II.

Within each Sex × Age group, ACE models were fitted. Table III presents the estimates of the variance accounted for by these three parameters in the full ACE model. Again, for each Sex × Age group, the A, C, and E terms were tested in turn and the best fit (i.e., most parsimonious) model was identified. It can be seen that the full model was the best model for all groups on both log-externalizing and log-internalizing, except for log-internalizing in younger boys for whom the C term could be dropped without a significant worsening of the fit.

To test whether the values for the A, C, and E parameters differed significantly by sex and by age group, a series of models was tested constraining the parameter estimates to be the same for the younger and older groups within each sex separately and for the parameters to be the same for boys and girls within each age group. These analyses were conducted for both the log-externalizing and log-internalizing scores. The only significant effects were found when the log-externalizing scores of the younger boys were included. For the log-externalizing scores, both the effect of sex within the younger group ($\chi^2 = 7.837$, $df = 3$, $p < .05$) and age within the boys ($\chi^2 = 15.039$, $df = 3$, $p < .01$) were significant. To examine which of the A, C, and E terms was producing these significant effects for the log-externalizing scores, additional analyses were conducted constraining just one term at a time to be equal across sex or age groups. This showed that both for the sex effects in the younger children ($\chi^2 = 7.099$, $df = 1$, $p < .01$) and for the age effects within the boys ($\chi^2 = 10.09$, $df = 1$, $p < .001$) it was only when the E term was constrained to be equal across groups that a significant worsening of model

Table II. Internalizing and Externalizing Within Pair Variance, Covariance, and Correlation Matrix by Sex, Age, and Zygosity[a]

	lInt	lExt	lInt2	lExt2
MZ boys, 5 to 9 years old, $n = 109$ pairs				
lInt	.129	**.554**	**.704**	**.586**
lExt	.084	.179	**.545**	**.941**
lInt2	.092	.084	.132	**.588**
lExt2	.088	.165	.089	.173
DZ boys, 5 to 9 years old, $n = 81$ pairs				
lInt	.111	**.488**	**.452**	**.435**
lExt	.055	.113	**.389**	**.665**
lInt2	.053	.046	.123	**.397**
lExt2	.056	.087	.054	.150
MZ girls, 5 to 9 years old, $n = 120$ pairs				
lInt	.123	**.566**	**.720**	**.505**
lExt	.074	.138	**.488**	**.881**
lInt2	.088	.063	.121	**.569**
lExt2	.068	.126	.076	.148
DZ girls, 5 to 9 years old, $n = 80$ pairs				
lInt	.140	**.412**	**.527**	**.461**
lExt	.058	.142	**.427**	**.684**
lInt2	.059	.048	.091	**.591**
lExt2	.063	.094	.064	.132
MZ boys, 12 to 15 years old, $n = 139$ pairs				
lInt	.127	**.570**	**.758**	**.493**
lExt	.084	.173	**.427**	**.891**
lInt2	.098	.065	.132	**.416**
lExt2	.072	.152	.062	.169
DZ boys, 12 to 15 years old, $n = 105$ pairs				
lInt	.136	**.489**	**.638**	**.498**
lExt	.078	.187	**.530**	**.630**
lInt2	.087	.085	.135	**.621**
lExt2	.083	.123	.103	.203
MZ girls, 12 to 15 years old, $n = 158$ pairs				
lInt	.130	**.540**	**.792**	**.540**
lExt	.074	.146	**.523**	**.869**
lInt2	.103	.072	.131	**.534**
lExt2	.074	.126	.073	.145
MZ girls, 12 to 15 years old, $n = 123$ pairs				
lInt	.124	**.604**	**.623**	**.446**
lExt	.088	.171	**.493**	**.567**
lInt2	.080	.074	.133	**.595**
lExt2	.062	.093	.086	.157

[a]Variances on diagonal, covariances below diagonal, correlations above diagonal (boldface) for each cell. lInt = log-internalizing behavior, twin 1; lExt = log-externalizing behavior, twin 1; lInt2: log-internalizing behavior, twin 2; lExt2 = log externalizing behavior, twin 2; MZ = identical twins; DZ = fraternal twins.

fit was obtained. The distinctive feature of the ACE model parameters for the younger boys was the low value of the E estimate (.067) (see Table III).

These sex and age effects analyses indicate that, with the exception of small effects of E on the log-

Table III. Proportions of Variance in Internalizing and Externalizing Scores Attributed to Genetic (A), Shared (C), and Nonshared (E) Environmental Factors by Sex and Age[a]

| | Full model | | | | | | Best model | χ^2 | df | p | GFI |
	A	C	E	χ^2	df	p					
lInt											
Boys, 5 to 9 years old	.444	.247	.308	0.731	3	n.s.	AE	2.450	4	n.s.	1.000
Girls, 5 to 9 years old	.377	.338	.286	5.149	3	n.s.	ACE	5.149	3	n.s.	.981
Boys, 12 to 15 years old	.262	.500	.236	0.236	3	n.s.	ACE	0.236	3	n.s.	1.000
Girls, 12 to 15 years old	.331	.449	.209	0.222	3	n.s.	ACE	0.222	3	n.s.	1.000
lExt											
Boys, 5 to 9 years old	.465	.468	.067	5.569	3	n.s.	ACE	5.569	3	n.s.	.991
Girls, 5 to 9 years old	.376	.503	.122	0.904	3	n.s.	ACE	0.904	3	n.s.	1.000
Boys, 12 to 15 years old	.573	.325	.102	1.073	3	n.s.	ACE	1.073	3	n.s.	1.000
Girls, 12 to 15 years old	.651	.225	.123	0.988	3	n.s.	ACE	0.988	3	n.s.	1.000

[a]GFI = goodness-of-fit index; lInt = log-internalizing behavior; lExt = log-externalizing behavior.

externalizing scores of the younger boys, the estimates of A, C, and E were not significantly different across age groups or for boys and girls. For log-externalizing behavior, a model with A = .563, C = .323 and E = .115 for girls and older boys and A = .465, C = .469 and E = .067 for younger boys, was the most parsimonious (difference in χ^2 from unconstrained model = 10.784, df = 6, n.s.). For log-internalizing behavior, a model with A = .338, C = .408 and E = .254 provides a good fit for all groups (difference in χ^2 from the unconstrained model = 9.108, df = 9, n.s.).

Table IV. Bivariate Independent Pathway Model; Proportions of Variance Attributed to Genetic (A), and Shared (C) and Nonshared (E) Environmental Influences; Specific and Common Factors by Sex and Age[a]

	A	C	E	χ^2, 10df	p	GFI
Boys 5 to 9 years old						
Specific lInt	.284	–	.319	10.76	n.s.	.962
Specific lExt	.213	–	.062			
Common lInt	.037	.360	.0001			
Common lExt	.217	.498	.0009			
Girls, 5 to 9 years old						
Specific lInt	.167	–	.189	13.66	n.s.	.928
Specific lExt	.350	–	.069			
Common lInt	.134	.410	.100			
Common lExt	.0003	.529	.051			
Boys, 12 to 15 years old						
Specific lInt	.224	–	.183	13.05	n.s.	.979
Speciifc lExt	.261	–	.091			
Common lInt	.010	.526	.057			
Common lExt	.263	.371	.014			
Girls, 12 to 15 years old						
Specific lInt	.224	–	.203	2.86	n.s.	.992
Specific lExt	.138	–	.123			
Common lInt	.116	.450	.006			
Common lExt	.511	.225	.003			

[a]GFI = goodness-of-fit index; lInt = log-internalizing behavior; lExt = log-externalizing behavior.

A bivariate independent pathway model (Fig. 1) was then applied separately to younger boys, younger girls, older boys, and older girls (Table IV). The model estimated genetic, shared and non shared environmental influences common to the two traits, and additional genetic and nonshared environmental influences specific to each trait. Standardized parameter estimates and values of A, C, and E for specific and common components of the variance in log-internalizing and log-externalizing behavior are shown in Table IV. Shared environmental factors for log-externalizing and log-internalizing behavior appeared to be the most influencial common factor. The common genetic component was small for both log-internalizing and log-externalizing behavior in younger children, while both shared environmental and genetic common factors were found for older children, girls especially. The model demonstrated an adequate fit in all sex/age groups.

The influence of sex and age on the common and specific ACE estimates was examined. Models constraining the parameter estimates to be equal for younger and older children within each sex separately were then compared to models allowing parameter estimates to vary within sexes, and similarly for sexes within age groups. These analyses showed that the effect of age was significant for both sexes (difference in χ^2 from the unconstrained model = 20.21, df = 10, $p < .05$ for girls, and for boys = 19.73, df = 10, $p < .05$). The effect of sex was nonsignificant in both age groups (Table V). Thus for younger children, a model with both sexes combined yielded A_{common} = .311, C_{common} = .384, E_{common} = .050, $A_{specific}$ = .0001, $E_{specific}$ = .254 for internalizing behavior and A_{common} = .005, C_{common} = .514, E_{common} = .027, $A_{specific}$ = .386, $E_{specific}$ = .068 for externalizing behavior. For

Table V. Fit Statistics for Constrained and Unconstrained Bivariate Independent Pathway Model

Model	Fit statistics		
	χ^2	df	p
Unconstrained			
1. Boys; 5 to 9 ≠ 12 to 15	23.81	20	n.s.
2. Girls; 5 to 9 ≠ 12 to 15	16.52	20	n.s.
3. 5 to 9; Boys ≠ girls	24.42	20	n.s.
4. 12 to 15: Boys ≠ girls	15.91	20	n.s.
Constrained			
5. Boys; 5 to 9 = 12 to 15	43.54	30	n.s.
6. Girls; 5 to 9 = 12 to 15	36.73	30	n.s.
7. 5 to 9; Boys = girls	39.47	30	n.s.
8. 12 to 15: Boys = girls	25.56	30	n.s.
Differences between models			
Model 5-Model 1	19.73	10	< .05
Model 6-Model 2	20.21	10	< .05
Model 7-Model 3	15.05	10	n.s.
Model 8-Model 4	9.65	10	n.s.

older children, both sexes combined, the estimates were A_{common} = .050, C_{common} = .482, E_{common} = .022, $A_{specific}$ = .244, $E_{specific}$ = .203 for internalizing behavior and A_{common} = .441, C_{common} = .299, E_{common} = .012, $A_{specific}$ = .144, $E_{specific}$ = .104 for externalizing behavior. Figure 2 shows the genetic and environmental

mediation of the total phenotypic correlation in younger and older children, respectively.

The above results were based on the total twin sample and log-transformed data. To provide more information about the comorbidity of these behaviors MZ and DZ probandwise concordance rates for internalizing and externalizing behaviour were computed in pure internalizers, pure externalizers, and comorbid cases. Twins were assigned to the deviant group when scoring equal to or above 1 standard deviation from the total sample mean. These results are shown in table VI. There were highly significant MZ-DZ differences in concordance for both pure internalizers and pure externalizers, while the concordance rates for internalizing and externalizing problems in comorbid cases were more similar. The MZ-DZ difference for internalizing behavior was nonsignificant and for externalizing behavior just reached the significance level of < .05 in comorbid cases. These results indicate less genetic and more shared environmental influence in the comorbid cases, which is illustrated further by the extreme group heritability estimates for the four groups. This estimation yielded heritability estimates with large standard errors, and the differences between pure and comorbid estimates were not significant for either internalizing or externalizing behavior.
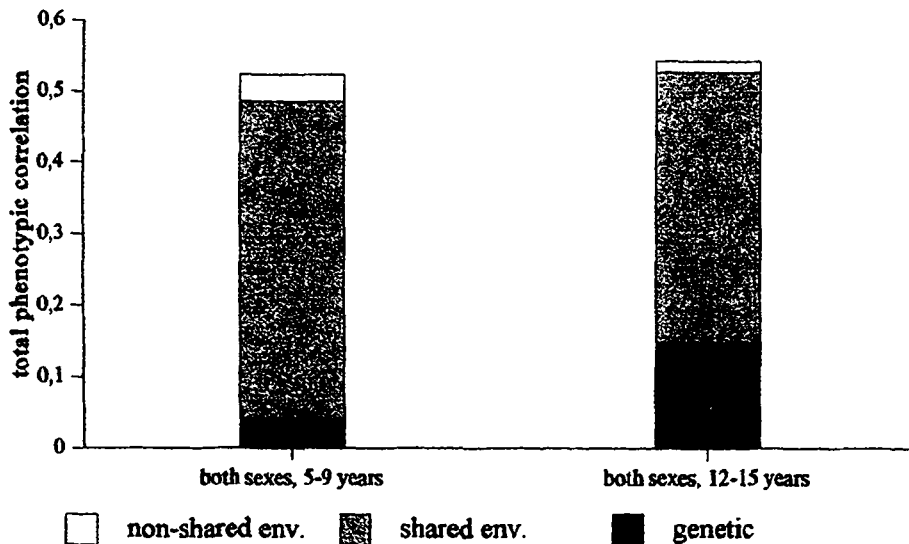

Fig. 2. Genetic and environmental mediation of phenotypic correlation. Genetic mediation = √(A_{common} int.) × √(A_{common} ext.); shared environmental mediation = √(C_{common} int.) × √(C_{common} ext.); nonshared environmental mediation = √(E_{common} ext.) × √(E_{common} ext.). int. = internalizing; ext. = externalizing.

Table VI. Twin Concordance Rates and Extreme Group Heritability for Internalizing and Externalizing Behavior Above 1 Standard Deviation from Total Population Mean, in Pairs Where One or Both Twins Have Internalizing (Int.) Problems Only, Externalizing (Ext.) Problems Only or Combined Problems[a]

	Ext. only	Int. only	Ext. in combination	Int. in combination
Number of twins	110	100	122	122
cc MZ twins	48	30	48	47
dc MZ twins	19	23	22	23
%cc MZ	71.6	56.6	68.6	67.1
cc DZ twins	8	10	27	24
dc DZ twins	35	37	25	28
%cc DZ	18.6	21.3	51.9	46.2
χ^2, 1 df	31.23[c]	13.40[c]	3.49	5.41[b]
Extreme group heritability	1.22	.74	.58	.34
(Standard error)	(.21)	(.26)	(.22)	(.23)

[a]cc = number of twins in concordant pairs, both twins scoring above 1 SD; dc = number of twins in discordant pairs; %cc = probandwise concordance = number of twins in concordant pairs/all twins with scores above 1 SD; MZ = identical twins; DZ = fraternal twins.
[b]$p < .05$.
[c]$p < .001$.

DISCUSSION

Variations in internalizing and externalizing behavior can be explained by models consisting of additive genetic, shared environmental, and nonshared environmental influences, with few sex or age differences. The findings indicate that the covariance between internalizing and externalizing behavior was explained by both genetic and shared environmental common factors, with shared environmental factors beeing the most influential especially in the young children. This is in accordance with the results reported for 2 to 3-year-old twins (Schmitz, Cherny, Fulker & Mrazek, 1994).

The concordance rates above a statistically defined cut-off supported these findings for deviant children. MZ and DZ cotwins of comorbid probands had a considerable risk of showing extreme internalizing or externalizing behavior, though the risk was somewhat higher for MZ cotwins. The risk of developing internalizing behavior problems was less for DZ than for MZ cotwins of pure internalizers, and the pattern was quite similar for externalizing behavior in pure externalizers. This indicates that a pure internalizing or externalizing behavior problem was

more genetically influenced than a comorbid condition.

These results suggest that genetic factors are more influential for separate internalizing or externalizing behavior, while shared environmental factors are more influential for the comorbid condition, although here genetic factors still are of some importance. Other studies have indicated similarities between comorbid and pure conduct disordered groups (Cole & Carpentieri, 1990; Graham & Rutter, 1973; Harrington at al., 1991; Puig-Antich et al., 1989). Harrington et al. discussed two possible models in accordance with their findings: Comorbid depression and conduct disorder could be classified as a separate diagnostic group or comorbid problems and pure conduct disorder could be parts of one diagnostic group. The present findings support the first suggested model.

There may be differences in genetic and environmental influences on conduct disorder and a continous measure of externalizing problems. Thus there may be a similarity between comorbid depression and conduct problems and pure conduct problems not apparent with these measures of internalizing and externalizing behavior. The large MZ-DZ difference in concordance for externalizing behavior only is suggestive of a strong genetic influence. The heritability of externalizing behavior in this study was higher than that reported in most twin studies of these behaviors (Edelbrock, Rende, Plomin, & Thompson, 1995; Hewitt, Silberg, Neale, Eaves, & Erickson, 1992; Silberg et al., 1994; Stevenson & Graham, 1988). Cultural differences may be influential and were shown in this study by less environmental variance possibly as a result of the social and cultural homogeneity of the Norwegian society. The mean scores were lower and the variances smaller for the internalizing and externalizing behavior problem scores than in the Achenbach (1991) normative sample. Thus it may be that the heritability estimates for externalizing behavior were increased due to a possibly smaller environmental variance.

The present findings to some extent support the findings from Puig-Antich et al. (1989) of less familial loading for comorbid depressive and conduct problems compared to pure depressive problems. There appears to be less genetic impact on comorbid problems. However, with the strong shared environmental influence on covariance, one would still expect a strong familial loading for combined internalizing and externalizing problems.

This study benefited from an epidemiological sampling, thus avoiding referral bias (Caron & Rutter, 1991). However, the response rate of 59.8% could indicate that a proportion of the most severly disturbed children were lost (Cox, Rutter, Yule, & Quinton, 1977). Consequently, the findings reported here may not be representative of the factors influencing cooccurrence across the entire range of problems in the general population. It could be that more severe problems were more influenced by environmental strains, increasing the relative environmental influence also on pure internalizing and externalizing behavior with increasing severity (Scarr, 1992). This would make the difference between comorbid and pure internalizing or externalizing behavior less marked. However, investigation of differential heritability with increasing severity of internalizing and externalizing behavior did not show such trends within the present sample (Gjone, Stevenson, Sundet, & Eilertsen, 1996). Rather, there was a trend toward increasing heritability with increasing severity of externalizing behavior, and of internalizing behavior in younger children only. These trends were apparent also when a cut-off point of 1 standard deviation above the mean was applied (unpublished results); thus it is unlikely that factors influencing comorbidity at a higher cut-off point would have differed significantly, though a participation bias could have left a possible influence as hypothesized by Scarr (1992), with increasing environmental influence for the more severe cases being undetected. There are no established norms for the CBCL in the Scandinavian countries, although norms for the Swedish child and adolescent population are being produced. These are not yet available and this prevents us from addressing the issue of genetic and environmental influences on problem behavior in the clinical range further at this stage. However, it is of interest to note that the mean level of behavior problems in a Swedish sample where the response rate was 81.7% was somewhat lower than in the present sample (Larsson & Frisk, 1993), and this suggests that more deviant cases have not been lost in the present sample.

The increased variance of externalizing behavior in twins when compared to a general population sample (Gjone & Nøvik, 1995) must be kept in mind as this could create a twin-specific environmental influence on externalizing behavior only, thus underestimating the genetic influence on externalizing behavior. A slightly lower level of internalizing problems in all but the youngest twins was found while there was no difference in level of externalizing behavior, indicating that the influence of twin-specific factors was small.

There was no item overlap spuriously influencing the cooccurrence of the CBCL Internalizing and Externalizing scales. Gould et al. (1993) found significant correlations for the CBCL Internalizing and Externalizing scales with DSM-III diagnoses of anxiety, depression, conduct disorder, oppositional disorder, and attention deficit disorder (ADD) in an epidemiological sample. The correlations were larger for internalizing with anxiety and depression and for externalizing with conduct, oppositional, disorder, and ADD. A similar though more specific correspondence was found by Edelbrock and Costello (1988) in a clinical sample. According to Gould et al., the internalizing and externalizing correlations with DSM-III diagnoses may reflect the significant correlations between internalizing and externalizing behavior indicative of a "multidimensional deviance."

Internalizing and Externalizing behavior scores were obtained from one rater only, and a shared rater bias could have inflated the covariance rates (Cole & Carpentieri,1990; Fergusson & Horwood, 1993). How the possible shared bias would influence the results would depend on the extent to which the bias for internalizing and externalizing behavior was correlated, and whether the rater bias differed between identical and fraternal twins. Correcting for these shared error terms would require information from different raters and through different methods of assessment, which was not available in the present study.

In summary, a model of additive genetic and shared and nonshared environmental influences fit internalizing and externalizing behavior in all the sex and age groups of this study. The considerable covariance between these traits appears to be accounted for mainly by shared environmental components, though the genetic factors were also of importance. The pattern of concordance rates indicates that mechanisms operating in the comorbid group differed from those influencing pure internalizing and pure externaling problems.

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