Genetic and Environmental Mechanisms Underlying Stability and Change in Problem Behaviors at Ages 3, 7, 10, and 12

M. Bartels
Vrije Universiteit

E. J. C. G. van den Oord
Medical College of Virginia /
Virginia Commonwealth University

J. J. Hudziak
University of Vermont College of Medicine

M. J. H. Rietveld, C. E. M. van Beijsterveldt,
and D. I. Boomsma
Vrije Universiteit

Maternal ratings on internalizing (INT) and externalizing (EXT) behaviors were collected in a large, population-based longitudinal sample. The numbers of participating twin pairs at ages 3, 7, 10, and 12 were 5,602, 5,115, 2,956, and 1,481, respectively. Stability in both behaviors was accounted for by genetic and shared environmental influences. The genetic contribution to stability (INT: 43%; EXT: 60%) resulted from the fact that a subset of genes expressed at an earlier age was still active at the next time point. A common set of shared environmental factors operated at all ages (INT: 47%; EXT: 34%). The modest contribution of nonshared environmental factors (INT: 10%; EXT: 6%) could not be captured by a simple model. Significant age-specific influences were found for all components, indicating that genetic and environmental factors also contributed to changes in problem behavior.

Understanding the origins, nature, and course of psychopathology across childhood is important for clinical purposes as well as for scientific purposes. Of particular clinical importance are the mechanisms underlying continuity. The longer an individual continues along a maladaptive pathway, the more difficult it is to reclaim a normal developmental trajectory (Sroufe, 1990). Further, research into child development suggests that early recognition of problems and causes of stability will lead to early intervention in families and successful prevention programs (Hermanns & Leu, 1998).

Problem behavior during childhood shows considerable continuity. For example, Richman, Stevenson, and Graham (1982) found that 61% of the problematic children at age 3 still showed considerable difficulties on a clinical rating scale 5 years later.

Graham and Rutter (1973) showed that 75% of the 10–11-year-old children who received a diagnosis of conduct disorder and 46% of the children who received a diagnosis of emotional disorder remained deviant at the follow-up 4 years later. Caspi, Moffitt, Newman, and Silva (1996) tested whether behavioral observations at age 3 were predictive of psychiatric disorders at age 21. Undercontrolled and inhibited children were at risk of later psychiatric problems. The Christchurch study also reported strong continuities of behavioral problems (Fergusson, Horwood, & Lynskey, 1995). Wright (1988) reported that three out of four antisocial adolescents grow up to be extremely maladjusted adults.

Stability is not confined to clinical groups and has also been found in general population samples. Verhulst and Van der Ende (1992b; the Zuid Holland study, the Netherlands) reported a correlation of .56 for problem behaviors across a 6-year period in a population sample of Dutch children originally studied at ages 4–11 years. Ghodsian, Fogelman, Lambert, and Tibbenham (1980) studied a national sample of British children assessed at ages 7, 11, and 16. Correlations for parental ratings of problem behavior were .48 between ages 7 and 11, .38 between 7 and 16 years, and .46 between 11 and 16 years. Despite several large studies on psychopathology throughout development, the causes of individual variation in longitudinal pathways throughout development are underexposed.

In order to study continuity and change, two broad classes of developmental mechanisms can be distinguished (e.g., see Rowe & Britt, 1991). The first class assumes that successive levels of functioning are causally linked so that each new event or change builds upon earlier experiences. In addition to the effects of past behavior, new influences may enter the picture at each phase to account for change. This class could be labeled *transmission models,* because parts of earlier influences and experiences are

transmitted to later points in time. For instance, Patterson, Dishion, and Yoerger (2000), argued that antisocial behavior among boys may be a process that builds and expands, a cascading of (negative) effects in which there is a significant path from early involvement with deviant peers to new forms of antisocial behavior in adolescence. The impact of earlier effects may not necessarily be confined to the same phenotype. Thus in the Great Smoky Mountains study (Costello, Farmer, Angold, Burns, & Erkanli, 1997) of White and American Indian youths living in rural southern Appalachia, Federman, Costello, Angold, Farmer, and Erkanli (1997) found that alcohol use without permission predicted subsequent use of illicit drugs and regular tobacco use.

A second class of developmental mechanisms can be referred to as *liability* or *common factor models* (e.g., see Van den Oord & Rowe, 1997). In contrast to transmission models, these mechanisms do not assume causal relations between subsequent time points. A stable underlying liability may be the "third variable" that explains the relations between subsequent levels of problem behavior. Liability mechanisms can also account for change, because at each point in time there may be new effects of other variables. However, continuity is completely explained by stable underlying liability and is not affected by these time-specific effects.

Transmission and liability models both imply a certain degree of continuity. However, in a longitudinal study, these mechanisms result in a different pattern of correlations between successive assessments. In a transmission model, subsequent levels of problem behavior are influenced by prior levels. The implication is that effects of prior events or experiences will be larger to the extent that they happened closer in time (Guttman, 1954; Jöreskog, 1970). The transmission model therefore predicts higher correlations among adjoining assessments than among those occurring more distantly in time. In contrast, the liability model assumes that the same stable factors exert their effects at each assessment and does not imply that correlations between assessments vary as a function of the length of the time lag. Thus, transmission and liability models predict different patterns of longitudinal correlations, and tests can be performed to derive the underlying mechanism by comparing observed and predicted correlations.

In a genetically informative longitudinal study, it is possible to go one step further and explore the transmission and liability models at the genetic and environmental level (Neale & Cardon, 1992). In such a study, one can account for the fact that genetic and environmental influences on the psychopathology during childhood display different patterns of development. So, there may be a mix of developmental processes. For example, a genetic contribution to continuity and change could operate through a transmission mechanism, and environmental influences, through a liability mechanism. However, by studying phenotypic data it is assumed that all influences exert the same developmental mechanism, and thus no insight into the mixture of different developmental processes can be obtained. A mixture of different developmental patterns is distinguishable at a multivariate genetic level, so using phenotypic data only could lead to false conclusions.

A powerful tool for unraveling the genetic and environmental architecture of individual differences in the development of behavioral and emotional problems is the study of genetically related individuals. Family studies might give a first impression of familial aggregation, but they cannot distinguish between genetic and environmental effects. Similarities between family members may be created either by genetic relatedness or by sharing the same family environment. A method that solves this problem is the classical twin design. Monozygotic (MZ) twins derive from a single zygote, and therefore the two individuals of an MZ twin pair are genetically identical. Dizygotic (DZ) twins develop from two distinct zygotes and share on average 50% of their genes, like ordinary brothers and sisters. Hence, the only possible way to explain the variation in problem behavior between two members of an MZ twin pair is environmental effects that are not shared by those two: the so-called nonshared environmental influences. Conversely, the variation in problem behavior between two members of a DZ twin pair could result from different genes and/or nonshared environmental influences. Accordingly, the difference in relatedness between MZ and DZ twin pairs (mostly expressed as correlation coefficients: r_{MZ} and r_{DZ}) gives information about the strength of the genetic and environmental influences on the trait under investigation. It further allows the separation of environmental influences into those of the environment shared by members of a family and those unique for each individual.

Only a few studies used this method to disentangle the genetic and environmental influences on continuity and change in the development of problem behaviors or problem-behavior-related disorders. In three studies, the Child Behavior Checklist (CBCL; Achenbach, 1991, 1992) was used to rate problem behavior in children. First, Van der Valk, van den Oord, Verhulst, & Boomsma (2003a) used a two-wave behavior genetic model to estimate genetic, shared environmental, and nonshared environmental contributions to stability and change of internalizing and externalizing problems from age 3 to age 7 in a Dutch sample that overlapped with our sample. For externalizing problems, the estimated influences of additive genetic, shared, and nonshared environmental factors remained relatively constant over the years. The phenotypic stability ($r = .54$) was explained for 55% by genetic factors. The same shared environmental influences were significant at ages 3 and 7, whereas nonshared environmental influences were mostly age specific. For internalizing problems, additive genetic influences decreased, whereas nonshared environmental influences increased, over the years. The phenotypic stability ($r = .38$) was explained for 66% by genetic factors. Second, Schmitz, Fulker, and Mrazek (1995) conducted a study in which they examined a small longitudinal sample of 95 twin pairs and assessed them at ages 2 and 7 years. Results indicated that for internalizing problems, continuing shared environmental factors had an effect both in early and middle childhood, whereas genetic influences had mostly age-specific effects. For externalizing problems, the opposite effect was found: continuing genetic and age-specific shared environmental effects. However, as suggested by the authors, these results need to be replicated with larger samples of genetically related subjects. Third, a study with biologically related and unrelated adoptees suggested that stability in externalizing problem behavior over a 3-year interval was mainly accounted for by additive genetic influences, whereas nonshared environmental influences mainly accounted for stability in internalizing problem behavior (Van der Valk, Verhulst, Neale, & Boomsma, 1998).

A developmental study of siblings, half siblings, and cousins, by Van den Oord and Rowe (1997), looked at maternal ratings of the Behavior Problems Index (Peterson & Zill, 1986). There were 436 pairs of full siblings, 119 pairs of half siblings, and 122 pairs of

cousins assessed at ages 4–6, 6–8, and 8–10. In this study, the continuity of problem behaviors was entirely explained by genetic and shared environmental factors. Nonshared environmental factors showed only age-specific effects, influencing changes in children's problem behaviors. O'Connor, Neiderhiser, Reiss, Hetherington, and Plomin (1998) followed 405 families over a 3-year interval. Subjects consisted of MZ and DZ twins and full, half, and step siblings (all of the same sex) between 10 and 18 years of age at the first assessment. Results showed that the phenotypic stability of antisocial symptoms ($r = .63$) was explained for 54% by continuing genetic influences and for 30% by continuing shared environmental influences. For depressive symptoms, the phenotypic stability ($r = .59$) was explained for 64% by continuing genetic influences and for 36% by continuing nonshared environmental influences. In short, even though each study investigated subjects at a different age interval and with different methods for measuring problem behavior, most studies showed large influences of genetic factors on the stability of problem behaviors. Effects of shared and nonshared environmental factors were less clear, with continuing influences shown for some studies and only age-specific effects shown for others.

In conclusion, a great deal of research has been conducted on the development of problem behavior during childhood. Most studies have pointed out that problem behavior is fairly stable throughout childhood. Children who exert problem behavior in a clinical range at a younger age are more likely to be deviant at an older age as well. This stability is accounted for by genetic as well as shared environmental influences. What is clearly lacking in the field of developmental psychopathology are studies on the developmental patterns of genetic and environmental influences on this stability. In order to answer unsolved questions on the possible mixture of genetic and environmental developmental mechanisms influencing problem behavior, we chose a behavior genetics perspective.

We had multiple aims in the present study. Our first aim was to estimate the importance of heritability and environmental influences at each age and to estimate the influence of genetic and environmental influences on the covariance (stability) between certain ages (which factors are responsible for continuity). The unique nature of this study, with CBCL data from large samples at four measurement occasions, provides the power to gain reliable insight into the underlying causes and developmental patterns of stability and change in problem behavior throughout development. Structural equation modeling techniques were used to examine the influences of genetic and environmental factors on the development of internalizing and externalizing behavior in a large sample of Dutch twin pairs at 3, 7, 10, and 12 years of age. A transmission model and a liability model were used to study the underlying pattern of development by taking into account continuity and change in genetic and environmental influences over time. In all analyses, estimates were tested for the presence or absence of sex differences. Clinical implications of the outcome of this study are discussed.

Method

Participants

All participants were registered by the Netherlands Twin Registry (NTR), which is kept by the Department of Biological Psychology at the Vrije Universiteit in Amsterdam. Of all multiple births in the Netherlands, 40%–50% are registered by the NTR (Boomsma, 1998; Boomsma, Orlebeke, & Van Baal, 1992). For this study, data from twins from the 1986–1993 birth cohorts were used. Behavioral questionnaires have been collected longitudinally at ages 3, 7, 10, and 12. CBCLs were mailed to families within 3 months of the twins' 3rd, 7th, 10th, and 12th birthdays. Two to three months after this mailing, reminders were sent, and 4 months after the initial mailing, persistent nonresponders were contacted by phone. Families whose address was no longer available were included in the nonresponse group. From the original sample, 253 twin pairs were excluded because either one or both of the children had a disease or handicap that interfered severely with daily functioning at age 12 or at a younger age. Thus, maternal ratings were available for 5,602 twin pairs at age 3, for 5,115 twin pairs at age 7, for 2,956 twin pairs at age 10, and for 1,481 twin pairs at age 12.

Zygosity was determined for 787 same-sex twin pairs by DNA analyses or blood group polymorphisms. For all other same-sex twin pairs, zygosity was determined by discriminant analysis, using questionnaire items at each age separately. Parents were asked how much the twins resembled each other in facial structure, hair color, facial color, and eye color and whether they were ever mistaken for each other by the parents themselves, by family, or by strangers. Parents were also asked if the twins were as much alike as two peas in a pod, whether it was difficult for the parents to tell the twins apart in a recent picture, and how similar the twins' hair structure was. Agreement between zygosity assignment by the replies to the questions and zygosity determined by DNA markers/blood typing was around 93% (for details, see Rietveld et al., 2000).

Measures

Mother ratings were collected by making use of the CBCL (Achenbach, 1991, 1992). The checklist for 2–3-year-old children (CBCL 2/3) shows age-adjusted differences from the checklist for 4–18-year-old children (CBCL 4–18).

The CBCL 2/3 was developed for parents to score the behavioral and emotional problems of their 2- and 3-year-old children. It consists of 100 items that are scored by the parents on a 3-point scale based on the occurrence of the behavior during the preceding 2 months: 0 if the problem item was *not true,* 1 if the item was *somewhat or sometimes true,* and 2 if it was *very true or often true.* Dutch syndrome scales and their comparability with the syndrome scales developed by Achenbach (1992) were reported by Koot, van den Oord, Verhulst, and Boomsma (1997). In the present study, the two broadband Internalizing and Externalizing scales are analyzed. The Internalizing scale consists of the Anxious and Withdrawn/Depressed subscales. The Externalizing scale consists of the Aggressive, Oppositional, and Overactive subscales. For the Internalizing scale, subjects were included only if not more than one item was missing for the Anxious subscale and not more than two items were missing for the Withdrawn/Depressed subscale. For the Externalizing scale, the inclusion criterion was not more than one item missing for the Aggressive and the Overactive subscales and not more than three items missing for the Oppositional scale. These criteria ensured that the two syndrome scales were always composed of all problem behaviors loading on that scale.

The CBCL 4–18 was developed for parents to score the behavioral and emotional problems of their 4- to 18-year-old children. It consists of 120 problem items that are scored by the parents on a 3-point scale based on the occurrence of the behavior during the preceding 6 months: 0 if the problem item was *not true,* 1 if the item was *somewhat or sometimes true,* and 2 if it was *very true or often true.* The syndrome scales were composed according to the 1991 profile (Achenbach, 1991). Dutch syndrome scales and comparability with the syndrome scales developed by Achenbach are reported in Verhulst, Van der Ende, and Koot (1996). In this manual, the two broadband Internalizing and Externalizing scales are analyzed. The Internalizing scale consists of the Anxious/Depressed, Somatic Com-

plaints, and Withdrawn subscales. The Externalizing scale consists of the Aggressive and Rule Breaking Behavior subscales. For the Internalizing scale, subjects were included only if not more than three items were missing for the Anxious/Depressed scale and not more than two items were missing for the Somatic Complaints and Withdrawn scales. For the Externalizing scale, the inclusion criterion was not more than three items missing for the Aggressive and Rule Breaking Behavior scales. These criteria ensured that the two syndrome scales were always composed of all problem behaviors loading on that scale.

Data Analyses

Descriptive statistics for internalizing and externalizing behavior at the distinct ages were calculated using SPSS 11 for Windows. To assess stability of problem behavior throughout development, we calculated phenotypic correlations over time for boys and girls separately. Cross-twin/cross-age correlations for the five zygosity groups have been calculated. These correlations give a first impression of the genetic and environmental contributions to stability and change in internalizing and externalizing problem behavior over time.

Genetic Modeling

In general, genetic model fitting of twin data allows for separation of the observed phenotypic variance into its genetic and environmental components. Additive genetic variance (A) is the variance that results from the additive effects of alleles at each contributing genetic locus. Shared environmental variance (C) is the variance that results from environmental events that make two children of a twin pair more alike (to the extent that they do not influence twins differently, possible examples include style of parenting, socioeconomic level, and religion). Nonshared environmental variance (E) is the variance that results from environmental effects that are not shared by members of a twin pair and that can cause differences between two members of a twin pair (such as an illness, relationships with peers, or measurement errors). Estimates of the nonshared environmental effects also include measurement error.

The different degree of genetic relatedness between MZ and DZ twin pairs can be used to estimate the contribution of genetic and environmental factors to the trait under study. Similarities for MZ twins are assumed to be due to additive genetic influences plus environmental influences that are shared by both members of a twin pair and that make them more alike. Experiences that make MZ twins different from one another are nonshared environmental influences. Because DZ twins share 50% of their genetic material on average, like other siblings, genetic factors contribute only half to their resemblance. As for MZ twins, the shared environment contributes fully. Model fitting to twin data is based on the comparison of the variance–covariance matrices in MZ and DZ twins. For a summary of the twin method, the various assumptions, and the plausibility of these assumptions, see, for example, Neale and Cardon (1992).

If data are collected repeatedly from the same subject at different ages, insight into developmental patterns of genetic and environmental influences can be gained. Multivariate genetic model fitting techniques were used to obtain insight into the developmental patterns of internalizing and externalizing problem behavior and to obtain estimates of the genetic and environmental influences on variances and covariances in problem behavior. In the present study, three types of models were used (see Figure 1): saturated models (I), transmission models (II), and common factor models (III). For each of the models, the total variances and covariances were decomposed into additive genetic (A), shared environmental (C), and nonshared environmental (E) parts.
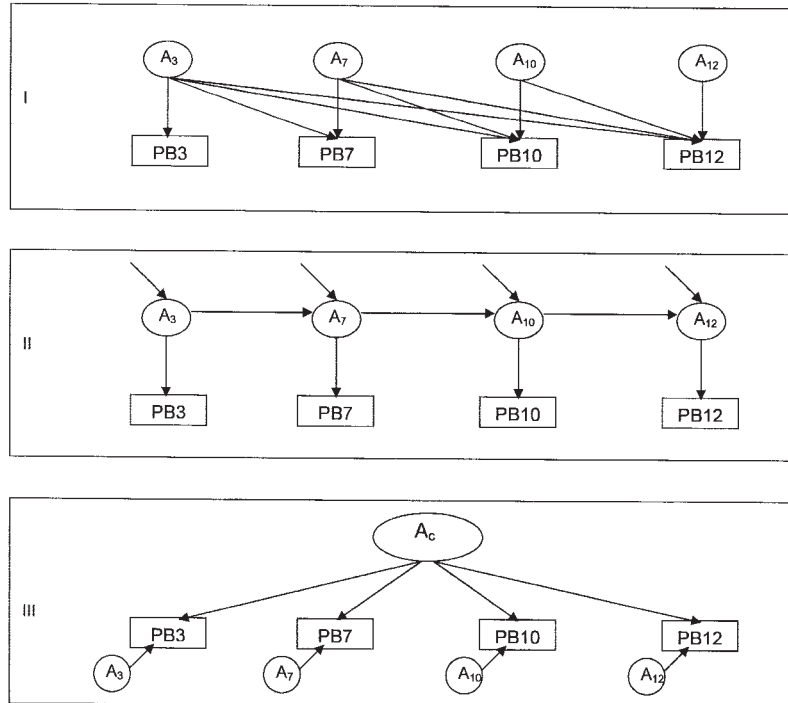
Figure 1. The three models used to investigate the underlying process of the development of problem behavior (PB3, PB7, PB10, and PB12 = problem behavior at ages 3, 7, 10, and 12, respectively): I, the saturated model; II, the transmission model; and III, the common factor model, with age-specific influences. Obviously, all three variance components (A, C, and E) can be expressed in either way. A = additive genetic variance, C = shared environmental variance, E = nonshared environmental variance.

The saturated model, also know as a Cholesky decomposition or triangular decomposition, decomposes the phenotypic statistics into genetic, shared environmental, and nonshared environmental contributions. In other words, the pattern of the factor loadings on the latent genetic and environmental factors (see II in Figure 1) reveals a first insight into the etiology of covariances between problem behaviors over time. Because the saturated model is fully parameterized, it yields the best possible fit to the input matrices. The model is descriptive and not driven by a specific developmental hypothesis, like transmission models and common factor models are. However, it is a useful model to gain a first insight into what to expect developmentally for genetic and environmental influences. It can be used further in reference to the fit of more specific developmental models and to evaluate the fits of these more restricted models. This evaluation consists of performing tests that compare the genetic and environmental contributions as predicted by a developmental model (transmission or common factor) with the unconstrained genetic and environmental contributions from the saturated model. If a developmental model fits the data significantly more poorly than the saturated model, the predicted contributions are inconsistent with the data, and the developmental model should be rejected.

The transmission model (II in Figure 1) represents a first-order autoregressive process. In the transmission model, covariances among the four ages of measurement are specified by genetic and environmental factors specific to each age and by "carry-over effects" or transmission of these factors to subsequent ages. The model specifies the variance unique to each measurement occasion by an innovation term that comes into play at each time point. The total variance is the sum of the age-specific effects and the age-to-age transmission effect. If this model fits the data well for the longitudinal genetic variance component, it implies that genetic influences account for both stability (by the transmission parameters) and change (by the innovation terms) in problem behavior throughout childhood. The same is true for shared and nonshared environmental influences. From a clinical perspective, knowledge of the strength of the innovation terms and the strength of transmission of genetic and environmental influences is essential for intervention.

In a common factor model (III in Figure 1), one underlying factor with age-specific factor loadings is specified, which implies a continuous influence from time of onset. In order to account for some age-specific variance, age-specific influences are added to the model. If this model fits the data well for the longitudinal genetic variance component, it implies that genetic influences mainly account for stability (by the underlying common factor). Little room is left for change due to genetic influences, specified by the age-specific influences. The same is true for shared and nonshared environmental influences. If an underlying genetic factor is the main cause of stability in problem behavior, implying a possible inborn vulnerability with long-lasting effects, it does not mean that those behaviors are unchangeable. An underlying factor will never explain 100% of the variance and/or covariance, which leaves room for intervention.

The order of model reduction and the possibilities of model specification influence the results of the parameter estimates and the goodness-of-fit procedure. To take this into account, we analyzed all three variance components (A, C, and E) separately, leaving the other two expressed in a saturated model. Finally, a "best" model was fitted to the data to obtain estimates of additive genetic, shared environmental, and nonshared environmental influences on the variances and covariances of internalizing and externalizing behavior.

Because in a longitudinal design data from one or more measurement occasions or from one twin may be missing from the data set, multivariate structural models were fitted to the transformed raw data by the method of maximum-likelihood pedigree analysis (Lange, Westlake, & Spence, 1976) using the Mx statistical software package (Neale, Boker, Xie, & Maes, 1999). Parameter estimates, including those for means of each variable for the first and second twin of each zygosity group, were produced that maximized the joint likelihood of the raw data under a given structural

model (Neale & Cardon, 1992; Neale et al., 1999). In order to use this method, the data were square-root transformed to approximate the normal distributions that are required for maximum-likelihood estimation. After transformation, all skewness and kurtosis indices were between −1.0 and 1.0, implying that not much distortion is to be expected (Muthén & Kaplan, 1985).

Submodels were compared by hierarchical chi-square tests. The chi-square statistic is computed by subtracting −2(log-likelihood) for the full model from that for a reduced model: $\chi^2 = 2(LL_1 - LL_0)$. In addition to the chi-square test statistic, Akaike's information criterion (AIC = $\chi^2 - 2 \times$ degrees of freedom) was computed. The lower the AIC, the better the fit of the model to the observed data. Although the transmission model and the factor model do not form a nested pair, they may be compared in terms of parsimony and goodness of fit because they represent alternative sets of constraints on the saturated model (Neale & Cardon, 1992). Fit statistics for the reduced developmental models are compared with those for the saturated models. This results in a chi-square and an AIC that are comparable for the different reduced models.

Results

The untransformed mean problem scores and standard deviations of the twin sample and those of comparable community samples (Koot et al., 1997, and Verhulst et al., 1996) are given in Table 1. For both the Internalizing and the Externalizing scale, the ratings given to the twins were quite similar to the ratings given to the Dutch community sample. Table 2 shows the phenotypic correlations for boys and girls separately. Twin correlations and the twin cross-correlations (off-diagonal) for internalizing and externalizing problem behavior at ages 3, 7, 10, and 12 are presented in Table 3.

The phenotypic correlations give a first impression of the underlying developmental pattern (transmission or common factor) of internalizing and externalizing problem behavior. For both internalizing and externalizing behavior, the phenotypic cross-age correlations are lower for longer intervals. This structure suggests, as explained in the introduction, a transmission pattern. This transmission structure, though, cannot be the sole mechanism because the stability in problem behavior between the distinct ages, represented by the cross-age correlation coefficients, is higher than would be expected on the basis of a transmission structure. For instance, if a transmission structure is describing the developmental process of internalizing problem behavior in boys, the product of the cross-correlations between ages 7 and 10 and ages 10 and 12 is about .40 (.61 × .66). However, the real cross-correlation between ages 7 and 12 is .54. This higher-than-expected cross-correlation implies, besides transmission, the influence of an underlying common factor on the development of childhood psychopathology. It is this mixture of underlying developmental mechanisms that can be investigated by using genetically related subjects.

Table 3 presents twin correlations (diagonal) and cross-correlations (off-diagonal). The twin correlations give a first indication of the genetic and environmental influences on the individual differences in problem behavior at the distinct ages. For both the MZM vs. DZM and the MZF vs. DZF comparisons (M = male; F = female), influences of additive genetic and shared environmental factors are expected for internalizing and externalizing problem behavior. Cross-age correlations represent, for instance, the correlation between internalizing behavior in the oldest twin at age 3 and internalizing behavior in the youngest twin at age

7. In other words, this cross-age correlation is a cross-twin/cross-age correlation, which gives information on the development of genetic and environmental influences on problem behavior over

Table 1

Means, Standard Deviations, and Sample Sizes for Maternal Ratings on the CBCL Internalizing and Externalizing Scales in 3-, 7-, 10-, and 12-Year-Old Twins (by Zygosity) Compared With a 2- and 3-Year-Old Dutch Community Sample and a 4–11-Year-Old Dutch Community Sample

	Internalizing			Externalizing		
Sample	*M*	*SD*	*N*	*M*	*SD*	*N*
	3-year-olds					
Boys						
MZ	4.64	4.00	1,740	17.94	10.49	1,738
DZ	4.59	4.05	1,752	16.92	9.90	1,752
DOS	4.52	3.82	1,771	16.04	9.83	1,769
COM	4.5	4.4	215	17.5	9.5	215
Girls						
MZ	4.78	4.08	2,038	15.64	10.01	2,035
DZ	4.90	4.18	1,637	15.30	9.71	1,641
DOS	3.98	3.77	1,770	14.26	9.43	1,772
COM	4.3	3.6	205	16.5	8.8	205
	7-year-olds					
Boys						
MZ	4.31	4.11	1,719	9.68	7.24	1,735
DZ	4.78	4.74	1,616	8.96	7.22	1,650
DOS	4.24	4.44	1,560	8.45	7.02	1,595
COM	4.52	4.27	579	8.26	6.36	579
Girls						
MZ	5.05	4.77	1,965	6.90	6.30	1,985
DZ	5.21	4.91	1,577	6.77	6.02	1,611
DOS	4.52	4.31	1,570	6.25	6.09	1,596
COM	5.16	5.02	593	6.04	5.57	593
	10-year-olds					
Boys						
MZ	4.59	4.60	1,008	8.67	7.43	1,029
DZ	5.18	5.44	933	8.03	7.34	937
DOS	4.66	4.97	877	7.69	7.10	885
COM	4.52	4.27	579	8.26	6.36	579
Girls						
MZ	5.12	5.00	1,216	5.91	5.53	1,223
DZ	5.35	5.35	893	5.90	5.86	905
DOS	4.76	4.91	878	5.34	5.37	885
COM	5.16	5.02	593	6.04	5.57	593
	12-year-olds					
Boys						
MZ	4.02	4.53	552	7.20	6.90	557
DZ	4.05	4.61	446	6.67	6.47	460
DOS	3.79	5.68	410	6.70	6.76	416
COM	5.36	5.36	440	6.35	6.13	440
Girls						
MZ	4.72	4.70	612	5.17	5.27	624
DZ	4.56	4.42	440	4.86	5.06	451
DOS	4.17	4.31	405	4.75	5.31	418
COM	6.32	5.93	456	5.21	5.43	456

Note. CBCL = Child Behavior Checklist; MZ = monozygotic; DZ = dizygotic; DOS = dizygotic opposite sex; COM = Dutch community samples.

the years. We calculated cross-correlations for MZ and DZ twins separately in order to explore the genetic and environmental influences on the observed stability. As can be seen in Table 3, the MZ cross-correlations are higher than the DZ cross-correlations, but certainly not twice as high, which suggests that stability in internalizing and externalizing problem behavior over time is due to additive genetic factors as well as shared environmental factors. The correlations' patterns over time are less clear, so no conclusion about the underlying developmental mechanisms can be drawn for these cross-correlations solely. No heterogeneity (different genes influencing problem behavior in boys or in girls) is expected because of the fact that the correlations in DZ twins of opposite sex (DOS) are not lower than the correlations in same-sex DZ twins. Model-fitting procedures are used to test for sex differences in the strength of genetic and environmental influences.

Model-fitting procedures for internalizing and externalizing problem behavior yielded the results presented in Table 4. As described in the Method section of this article, distinct models were fitted to the longitudinal data. The saturated, Cholesky decomposition, model without restrictions (Model 1; I in Figure 1) was taken as a reference for evaluating changes in chi-square and associated degrees of freedom of more parsimonious models. Model reduction consisted of a transmission model on the one hand and a common factor model (one underlying factor) on the other hand. Because of the fact that the order of the model reductions could influence the results of the goodness-of-fit procedure, all three variance components (A, C, and E) were analyzed separately. The mechanism of one variance component at a time was investigated, leaving the other two components expressed in a saturated model (Models 2 through 7). Model 8 and Model 9 represent the best-fitting simplified models with and without sex differences. Sex differences in the strength of the genetic and environmental effects were tested by constraining the influences for boys and girls to be equal.

For internalizing problem behavior, a best-fitting simplified model to describe the processes of development is a model with a transmission structure for additive genetic factors and a factor structure with age-specific factor loadings for shared environmental influences (Model 9). As can be seen in Table 4, for additive

Table 2

Phenotypic Correlations (With Their 95% Confidence Intervals) for Internalizing (Above Diagonals) and Externalizing (Below Diagonals) Problem Behavior Separately for Boys and Girls

Age	3 years	7 years	10 years	12 years
	Boys			
3 years	—	.35 (.32–.37)	.32 (.32–.32)	.29 (.29–.33)
7 years	.54 (.52–.54)	—	.61 (.61–.62)	.54 (.51–.58)
10 years	.50 (.50–.51)	.73 (.72–.75)	—	.66 (.63–.68)
12 years	.49 (.46–.52)	.69 (.69–.71)	.76 (.75–.78)	—
	Girls			
3 years	—	.41 (.40–.44)	.35 (.32–.39)	.33 (.28–.38)
7 years	.57 (.57–.60)	—	.62 (.59–.65)	.59 (.55–.63)
10 years	.48 (.45–.51)	.70 (.68–.72)	—	.69 (.66–.72)
12 years	.46 (.41–.50)	.66 (.66–.69)	.72 (.71–.75)	—

Table 3

Twin Correlations and Cross-Correlations for Internalizing (Above Diagonals) and Externalizing (Below Diagonals) Problem Behavior for the Five Zygosity Groups

Age	3 years	7 years	10 years	12 years
		MZM		
3 years	.712[a] (859)[b]\.840[c] (859)[b]	.283 (694)	.266 (408)	.240 (227)
7 years	.498 (701)	.693 (853)\.847 (865)	.529 (432)	.420 (237)
10 years	.463 (417)	.665 (447)	.659 (498)\.857 (514)	.487 (253)
12 years	.461 (228)	.616 (239)	.695 (254)	.734 (273)\.875 (276)
		DZM		
3 years	.408 (868)\.567 (867)	.250 (684)	.271 (397)	.248 (.192)
7 years	.327 (697)	.460 (799)\.544 (822)	.370 (396)	.239 (182)
10 years	.287 (397)	.400 (404)	.481 (465)\.535 (467)	.405 (195)
12 years	.266 (198)	.314 (190)	.399 (202)	.451 (217)\.531 (405)
		MZF		
3 years	.734 (1006)\.832 (1003)	.399 (821)	.316 (502)	.397 (250)
7 years	.541 (829)	.734 (975)\.861 (989)	.513 (521)	.473 (260)
10 years	.437 (509)	.634 (531)	.680 (602)\.811 (609)	.540 (276)
12 years	.410 (257)	.600 (270)	.612 (283)	.719 (301)\.839 (310)
		DZF		
3 years	.428 (810)\.564 (813)	.275 (646)	.284 (359)	.230 (176)
7 years	.369 (663)	.533 (778)\.553 (802)	.387 (383)	.354 (188)
10 years	.337 (364)	.396 (395)	.507 (442)\.582 (450)	.329 (197)
12 years	.250 (180)	.286 (196)	.389 (202)	.535 (214)\.553 (223)
		DOS		
3 years	.388 (1752)\.542 (1752)	.243 (1324)	.286 (745)	.263 (341)
7 years	.340 (1342)	.509 (1550)\.562 (1591)	.351 (718)	.342 (324)
10 years	.301 (748)	.377 (740)	.532 (871)\.502 (883)	.384 (345)
12 years	.273 (352)	.408 (344)	.454 (361)	.592 (398)\.605 (413)

Note. MZM = monozygotic males; DZM = dizygotic males; MZF = monozygotic females; DZF = dizygotic females; DOS = dizygotic opposite sex.
[a] Internalizing behavior. [b] Number of complete twin pairs. [c] Externalizing behavior.

genetic influences, the factor model (Model 3) and the transmission model (Model 2) gave almost identical fits to the data. The transmission model, however, is a more parsimonious model, and we therefore prefer the transmission model over a factor model. The parsimony is reflected in a lower AIC for the transmission model than for the factor model, suggesting a better fit of the transmission model. It should be noted that the difference between the models is small and that our preference for the transmission model is based solely on parsimony. A transmission model for the genetic influences on internalizing behavior, though, implies that genetic influences are transmitted from one age to the previous age. Further new genetic influences seem to be significant at each age. A result of this model is that stability and change are caused by transmitted and innovative genetic influences, respectively. If the common factor model was preferred for the additive genetic influences on internalizing behavior, A also would account for stability and change. However, the autoregressive properties of the transmission model imply that new genetic influences *build on* earlier influences, whereas this transmission process cannot be captured in the liability model. A common factor model for shared environmental influences describes the process of one underlying

shared environmental factor (with age-specific factor loadings) that influences internalizing behavior at all ages. So, shared environment is mainly important for stability in internalizing behavior over age. No sex differences in the magnitude of genetic and environmental influences are found for internalizing problem behavior.

For externalizing problem behavior (see Table 4), a model with a transmission structure for additive genetic influences and the saturated model for shared and nonshared environmental influences (Model 2) did not give a significantly worse fit than the full saturated model (Model 1), $\chi^2 = 9.344$, $\Delta df = 6$, $p = .16$. Further, shared environmental influences display a factor structure (Model 5). This implies that the developmental pattern of genetic and shared environmental influences on externalizing behavior is similar to the patterns found for internalizing problem behavior, with genetic influences accounting for stability and change and shared environmental influences mainly accounting for stability. For nonshared environmental influences on both internalizing and externalizing problem behavior, neither the transmission model nor the factor model gave a satisfactory fit. The implication is that the processes that account for the nonshared environmental contribu-

Table 4
Multivariate Model Fitting for Internalizing and Externalizing Problem Behavior at Ages 3, 7, 10, and 12

Model	Internalizing							Externalizing						
	−2LL	df	Compared to model	Δχ²	Δdf	p	AIC	−2LL	df	Compared to model	Δχ²	Δdf	p	AIC
1. Saturated model	77,086.981	29,285						82,410.846	29,569					
2. A: Transmission structure	77,095.034	29,291	1	8.053	6	.23	−3.95	82,420.190	29,575	1	9.344	6	.16	−2.66
3. A: Factor structure	77,094.903	29,286	1	7.922	4	.09	−.08	82,424.485	29,573	1	13.639	4	.00	5.64
4. C: Transmission structure	77,102.743	29,291	1	15.762	6	.02	3.76	82,427.391	29,575	1	16.545	6	.01	.455
5. C: Factor structure	77,092.403	29,289	1	5.422	4	.25	−2.58	82,415.336	29,573	1	4.49	4	.34	−.351
6. E: Transmission structure	77,131.185	29,291	1	44.204	6	.00	32.20	82,455.500	29,575	1	34.654	6	.00	22.65
7. E: Factor structure	77,100.577	29,289	1	13.596	4	.01	5.60	82,433.770	29,573	1	22.924	4	.00	14.92
8. Simplified model	77,098.961	29,295	1	11.980	10	.29	−8.02	82,429.340	29,579	1	18.494	10	.05	−1.51
A: Transmission structure														
C: Factor structure														
E: Saturated														
9. Simplified model—no sex differences	77,130.102	19,320	8	31.141	25	.18	−18.86	82,506.974	29,604	8	77.634	25	.00	27.63
A: Transmission structure														
C: Factor structure														
E: Saturated														

Note. A = additive genetic variance; C = shared environmental variance; E = nonshared environmental variance; −2LL = −2 log likelihood; AIC = Akaike's information criterion.

tion were complex and could not be captured by one of our relatively simple models. For externalizing problem behavior, sex differences seem to be significant (Model 8). In other words, the parameters estimated for genetic and environmental influences are of different magnitude for boys and girls.

The parameter estimates based on the best-fitting model for internalizing and externalizing problem behavior are represented in Figures 2 and 3. These figures clearly represent the transmission model for the genetic influences and the common factor model for shared environmental influences. The percentages of the total age-specific variance and the total between-age covariances explained by additive genetic, shared environmental, and nonshared environmental factors based on the best-fitting reduced models are presented in Table 5. In Table 5, the total genetic variance at each specific age is decomposed into variance due to innovation and variance due to transmission from a previous age. In this way, the part of the additive genetic influences that accounts for change and the part that accounts for stability in childhood psychopathology are quantified. The total shared environmental variance is decomposed into variance due to the common underlying factor and variance due to age-specific shared environmental influences. The total nonshared environmental variance is decomposed into variance specific to a certain age and variance as a result of nonshared environmental factors from a previous age. A decrease in additive genetic effects and an increase in shared environmental effects are observed for internalizing problem behaviors (e.g., A for internalizing at age 3 = 59%; A for internalizing at age 12 = 37%). These influences seem to stabilize from age 7 onward. For externalizing behavior, less change in additive genetic and shared environmental influences is observed, although an increase in additive genetic influences for boys between ages 3 and 10 can be seen in Table 5.

In Table 5 (off-diagonal), the influences of additive genetic, shared environmental, and nonshared environmental influences on the covariances are important. Stability, represented by these covariances, can be explained both by additive genetic and shared environmental influences. However, as mentioned before, genetic influences operate by a transmission process, whereas shared environmental influences are best described by one underlying factor. Nonshared environmental influences seem to be of no significance for stability in problem behavior, which is represented by very low influences on the covariances. The only exception is the covariance between ages 10 and 12 for internalizing behavior, 22% of which is accounted for by nonshared environmental influences. On average, 47% of the stability of internalizing behavior over the years ([52% + 43% + 32% +51% + 40% + 38%] / 6) can be explained by additive genetic factors, 43% of this stability can be explained by shared environmental factors, and the remaining 10% can be explained by nonshared environmental influences. For externalizing behavior in both boys and girls, though, additive genetic factors seem to be the main source of stability. For externalizing behavior in boys, 67% of the stability is explained by additive genetic factors. Twenty-seven percent and 6% of the stability are explained by shared and nonshared environmental influences, respectively. For externalizing problem behavior in girls, 53% of the stability over the years is explained by additive genetic factors. Shared environmental influences account for 40% of the stability, and 7% is explained by nonshared environmental influences.
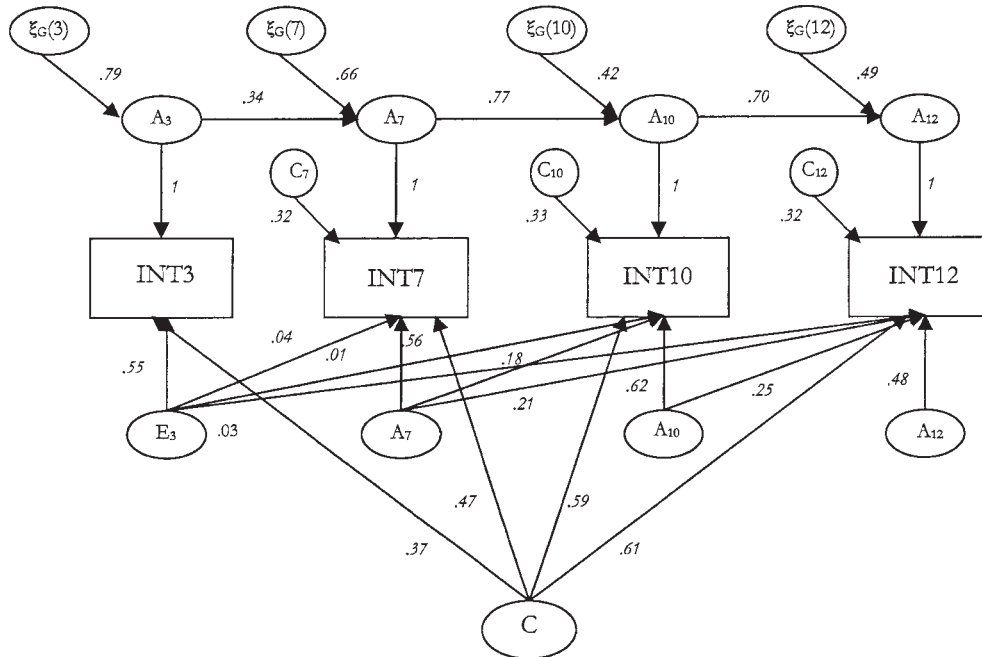
Figure 2. The best-fitting model for internalizing problem behavior (INT3, INT7, INT10, and INT12 = internalizing problem behavior at ages 3, 7, 10, and 12, respectively), with a transmission structure for additive genetic (A) factors, a common factor with age-specific influences for shared environmental (C) factors, and a Cholesky decomposition for nonshared environmental (E) factors. (Note that the variance of all latent factors is fixed to unity.)

Discussion

To understand the development of internalizing and externalizing problem behavior, we used structural equation modeling techniques in a large longitudinal sample of Dutch twins. This longitudinal study with large sample sizes and four measurement occasions gave us the unique opportunity to distinguish between a transmission process and a common factor process underlying the genetic and environmental influences on internalizing and externalizing problem behavior throughout childhood. The transmission model assumes that successive levels of functioning were causally linked and that earlier experiences and/or genetic effects affected later maladjustment. The factor model related continuity in problem behavior to stable underlying environmental and/or genetic factors.

Phenotypic Stability

Our phenotypic correlation structure is consistent with phenotypic stability coefficients reported in large-scale longitudinal studies. Verhulst and Van der Ende (1992a, 1992b) studied stabilities in problem behavior in a sample of 936 Dutch 4- to 11-year-old children. Their study is especially interesting because the children were almost in the same age range and from the same Dutch population as the children described in the present article. The average observed stability coefficients for the 2-, 4-, and 6-year time intervals were, respectively, .53, .48, and .42. The stability coefficients in our study and in this previous comparable study suggest a transmission pattern as the underlying develop-

mental process. However, the stability between the distinct ages is higher than would be expected solely on the basis of a transmission structure. This same pattern of stability coefficients was also found in a national sample of 16,000 British children at ages 7, 11, and 16 years (Ghodsian et al., 1980). In comparison with the present study, though, those children were somewhat older, and the interval covered the onset of puberty and the transition to high school. The previous studies could not distinguish between genetic and environmental influences on the developmental process. These different sources of variances may display a distinct developmental pattern. An important feature of the present longitudinal twin studies is that they offer an opportunity to investigate the developmental pattern of each source of variance independently.

Developmental Patterns and Stability

Stability in the development of internalizing problem behavior can be explained by additive genetic transmission factors accounting for 43% of the stability on average. Another factor explaining stability in the development of internalizing problem behavior is an underlying common factor for shared environmental influences accounting for 47% of the total stability over the years. Ten percent of the stability of internalizing problem behavior over the years is accounted for by nonshared environmental factors. Change is mainly accounted for by nonshared environmental influences. Genetic innovation factors and small but significant age-specific influences of shared environment account for some change as well.

A comparable pattern for stability is found for externalizing problem behavior. Stability is explained by additive genetic trans-
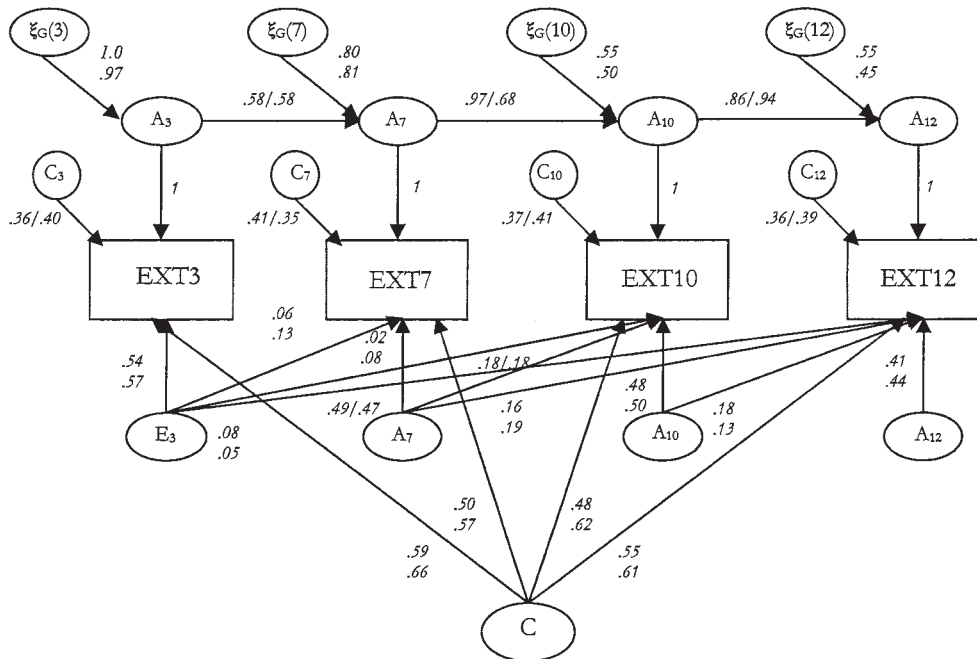
Figure 3. The best-fitting model for externalizing problem behavior (EXT3, EXT7, EXT10, and EXT12 = externalizing problem behavior at ages 3, 7, 10, and 12, respectively) in boys (first number) and girls (second number), with a transmission structure for additive genetic (A) factors, a common factor with age-specific influences for shared environmental (C) factors, and a Cholesky decomposition for nonshared environmental (E) factors. (Note that the variance of all latent factors is fixed to unity.)

mission factors accounting for 67% of the stability over the years, on average, for boys and 53% of the stability over the years, on average, for girls. Stability is further accounted for by a common shared environmental factor explaining 27% and 40% of the total stability for boys and girls, respectively. Change in externalizing behavior in both boys and girls can be mainly explained by nonshared environmental influences. Genetic innovations and age-specific shared environmental influence account for some change in externalizing problem behavior over the years. It is generally acknowledged that for univariate analyses, at one age only, most environmental influences are of the nonshared type and that shared environmental influences are rather small (Plomin & Daniels, 1987; Rowe, Woulbroun, & Gullwy, 1994). In the present study, this was found for shared and nonshared environmental influences at age 3. An increase in shared environmental influences, though, was observed at older ages. Further, the results suggest that for continuity in problem behavior, shared environmental influences are more important than nonshared environmental influences.

The finding of a transmission structure for additive genetic influences in this study explains the finding of the developmental pattern in the phenotypic correlation. This transmission pattern for additive genetic factor accounts for the lower cross-correlations for longer intervals. The higher-than-expected stability between the distinct ages that is based solely on a transmission structure, though, can be explained by the common underlying factor for shared environmental influences. It should be noted that the distinction between a transmission model and a liability model for additive genetic factors on internalizing behavior is based solely on parsimony, which indicates that further research is necessary to

investigate the developmental mechanism for genetic influences on internalizing behavior.

Several authors have pointed out that although all genes are present from conception onward, this does not necessarily imply that genetically influenced traits are stable over time (Plomin, 1986; Wilson, 1986). This is because not all genes are important all the time, and the effects of specific subsets of genes may be age dependent. Further, the idea of transmission is quite common and is present in many developmental theories such as psychoanalytic theory or attachment theory (Lamb & Nash, 1989). In addition, developmental concepts such as *critical periods* or *developmental tasks* also refer to a process in which outcomes of certain phases affect future function. No previous study found this transmission to be accounted for by genetic factors rather than environmental influences.

A common factor for shared environmental influences on internalizing and externalizing problem behavior was found. Our study indicates, though, that besides a continuing influence of shared environmental factors, age-specific influences are present. These age-specific effects were significant, but the proportion of variance explained was much smaller than the proportion explained by the shared environmental factor common to all ages. This common factor could be accounted for by stable familial factors such as socioeconomic status, as this important shared environmental aspect is not sensitive to large changes over a time span of 9 years. Aspects outside the family environment, such as friends or being a member of a sports club or school, might also cause similarities between two children of a twin pair during childhood but could be age specific rather than continuous throughout development (Har-

Table 5

Percentages of the Total Variances (Diagonal) and Covariances (Off-Diagonal) (With Their 95% Confidence Intervals) for Internalizing and Externalizing Problem Behavior Explained by Additive Genetic (A), Shared Environmental (C), and Nonshared Environmental (E) Components Based on the Best-Fitting Models

Age	A				C				E			
	3 years	7 years	10 years	12 years	3 years	7 years	10 years	12 years	3 years	7 years	10 years	12 years
Internalizing												
3 years	.59 (.54–.63)				.13 (.09–.17) *.00/.13*				.28 (.26–.30)			
7 years	.52 (.45–.62)	.44 (.38–.50) *.38/.06*			.43 (.34–.51)	.28 (.23–.33) *.09/.19*			.05 (.02–.09)	.28 (.26–.30) *.27/.01*		
10 years	.43 (.32–.55)	.51 (.43–.58)	.36 (.28–.43) *.14/.22*		.56 (.45–.66)	.36 (.29–.43)	.34 (.27–.40) *.09/.25*		.01 (.00–.06)	.13 (.11–.16)	.31 (.28–.33) *.28/.03*	
12 years	.32 (.23–.45)	.40 (.30–.50)	.38 (.28–.47)	.37 (.27–.46) *.19/.18*	.63 (.52–.74)	.42 (.33–.53)	.40 (.32–.48)	.37 (.28–.45) *.08/.29*	.04 (.00–.09)	.17 (.14–.21)	.22 (.19–.25)	.26 (.24–.30) *.18/.08*
Externalizing boys												
3 years	.57 (.48–.64)				.27 (.25–.35) *.07/.20*				.16 (.16–.18)			
7 years	.64 (.54–.73)	.59 (.53–.66) *.39/.20*			.33 (.24–.41)	.26 (.19–.32) *.11/.15*			.03 (.01–.06)	.15 (.14–.17) *.14/.01*		
10 years	.66 (.56–.76)	.74 (.67–.81)	.65 (.59–.71) *.16/.49*		.33 (.25–.45)	.19 (.12–.26)	.20 (.15–.27) *.07/.13*		.01 (.00–.04)	.07 (.05–.09)	.15 (.14–.15) *.13/.01*	
12 years	.57 (.45–.68)	.69 (.62–.78)	.73 (.64–.73)	.64 (.54–.73) *.16/.48*	.38 (.31–.49)	.23 (.22–.33)	.18 (.11–.27)	.23 (.16–.23) *.07/.16*	.05 (.02–.08)	.07 (.05–.10)	.08 (.06–.11)	.13 (.11–.15) *.09/.04*
Externalizing girls												
3 years	.50 (.42–.58)				.32 (.24–.40) *.08/.24*				.18 (.16–.20)			
7 years	.54 (.44–.64)	.59 (.52–.66) *.40/.19*			.38 (.28–.43)	.27 (.27–.29) *.07/.20*			.07 (.06–.09)	.14 (.13–.16) *.13/.01*		
10 years	.45 (.43–.57)	.60 (.50–.70)	.45 (.36–.53) *.16/.29*		.50 (.38–.59)	.32 (.31–.41)	.36 (.31–.45) *.11/.25*		.06 (.03–.09)	.09 (.07–.11)	.19 (.17–.21) *.16/.03*	
12 years	.44 (.34–.57)	.58 (.48–.66)	.58 (.46–.66)	.51 (.43–.61) *.13/.38*	.52 (.39–.63)	.33 (.23–.43)	.33 (.33–.44)	.33 (.33–.44) *.09/.24*	.04 (.00–.07)	.09 (.06–.11)	.09 (.07–.12)	.16 (.13–.17) *.12/.04*

Note. In the pairs of italic numbers on the diagonals, the first number represents variance explained by age-specific influences, and the second number represents common influences specific to the underlying developmental mechanism.

ris, 1995). An explanation for age-specific shared environmental influences at ages 7, 10, and 12 could be the change of teacher and classroom at every level in Dutch elementary schools. Information on whether both children of a twin pair had the same teacher or different teachers in a large sample of 7-, 10-, and 12-year-old twin pairs (based on unpublished data from the Netherlands Twin Register) indicated that in 63% of the cases, on average, both children of a twin pair were taught by the same teacher, whereas 37% went to separate classes. This ratio makes teacher or classroom environment a shared environmental influence for the majority of the children. Because in the Dutch school system, children move to a different teacher each school year, this results in a lack of continuity in this particular aspect of the shared environment. So these shared but age-specific experiences within the classroom may be represented by the age-specific factors specified as significant in the best-fitting model. Indications for considering the classroom and teacher as a shared environment in general are given by preliminary results (based on unpublished data from the Netherlands Twin Register) for a different phenotype (scholastic achievement) measured in a sample that overlapped with the sample used in the present study. The twin correlations for scholastic achievement measured with the CITO test (*Eintoets Basisonderwijs 2002,* 2002) in twins taught by the same teacher (MZ = .85; DZ = .63) indicated higher influences of shared environment than did the twin correlations for CITO in twins taught by different teachers (MZ = .78; DZ = .29). It should be noted that because only a minority of the twins went to separate classes, the zygosity groups used to calculate these twin correlations are small.

The finding in the present study that shared environmental influences are represented by a common factor and time-specific influences replicates the results of developmental studies in other areas (Bartels, Rietveld, Van Baal, & Boomsma, 2002). It indicates that there could be a very stable set of shared environmental influences that causes problems to persist over the years. Similar results seem to be suggested by a number of epidemiological studies showing that problems tend to continue in families with ongoing family adversities such as marital stress, negative maternal control, and maternal depression (Campbell, March, Pierce, Ewing, & Szumowski, 1991; Egeland, Kalkoske, Gottesman, & Erickson, 1990; Richman et al., 1982). Thus, not family adversity as such but its persistence predicts chronic problems (Campbell, 1994). It should also be mentioned that although ongoing family adversity may indeed represent shared environmental influences, parts of its relation with continuity in problem behavior might be explained by genetic influences. This could be due to genetic factors that are shared by parents and children and influence both the family environment and children's behavior (Braungart-Rieker, Rende, Plomin, DeFries, & Fulker, 1995; Plomin, 1995; Plomin, Reiss, Hetherington, & Howe, 1994; Rowe, 1981, 1983).

Nonshared environmental influences were substantial at each age and contributed mainly to change in children's problem behavior. For the covariance between ages 7, 10, and 12 in internalizing behavior, however, nonshared environmental factors seem to be of significant influence. Possible examples of nonshared environmental influences include illness, trauma, fluctuations in mood and state, and peer group influences (Plomin & Daniels, 1987; Rowe et al., 1994). Findings from this study imply that these adverse experiences are important, and although they are mostly of a transient nature and children recover from them, sometimes they also exert long-lasting effects.

Genetic and Environmental Influences

In addition to investigating the underlying developmental patterns of genetic and environmental influences, this large longitudinal study gives reliable estimates of genetic, shared, and nonshared environmental influences on internalizing and externalizing problem behavior throughout childhood.

For internalizing behavior, no sex differences were found over the years. The relative importance of the additive genetic effects decreased from age 3 (59%) to age 7 (44%) but remained about the same from age 7 to age 10 (36%) and age 12 (37%). An increase in shared environmental influences was found. At age 3, shared environmental influences were small (13%), whereas at ages 7, 10, and 12, shared environmental influences were significant (age 7: 28%; age 10: 34%; age 12: 37%). An explanation for the presence of change between ages 3 and 7 and the relatively small changes between ages 10 and 12 could be that the 3–7-year age interval includes children's transition to school. During this transition, children must cope with many new demands such as meeting academic challenges, learning school and teacher expectations, and adjusting to the daily routine of a school class (Barth & Parke, 1993; Cowan, Cowan, Schultz, & Heming, 1994; Ladd & Price, 1987). An important aspect of this transition is the development of social relations with other children (Asher, 1990; Schneider, 1993). Although multiple pathways can be involved, poor relations with peers have been shown to be a powerful predictor of behavior and emotional problems later in life.

One explanation for the increase in the influence of shared environment is that if parents are only able to guide the child's behavior when he or she is able to understand other people's values and can direct his or her behavior accordingly, then shared environmental influences are more likely to be found in older children. However, it may be important to realize that shared environment is not necessarily confined to the home environment. For instance, there are indications that these environmental effects are shared not only by siblings but also by cousins (Van den Oord & Rowe, 1998, 1999). This suggests that shared environment reflects the wider community in which families are embedded as well (Bronfenbrenner, 1979; Parke & Kellam, 1994, p. 3). This point has also been stressed by Harris (1995), who argued that we should think about environmental effects in terms of group processes in which peers play an important role. That is, phenomena such as within-group assimilation and between-groups contrast, which increase the homogeneity of behaviors within groups and widen differences between social groups, could show up as shared environment in behavior genetic analysis.

For externalizing behavior, sex differences were found. For boys, a small increase in additive genetic effects was found from age 3 (57%) to age 12 (64%). A complementary small decrease in shared environmental influences was observed, explaining 27% of the variance at age 3 and 23% of the variance at age 12. For girls, the influences of genetic and environmental factors remained relatively stable over the years. For both boys and girls, nonshared environmental influences were found at ages 3, 7, and 10 years. Thus idiosyncratic experiences seem to be important in explaining both preschool and school-age children's problem behaviors.

Limitations of the Study and Clinical Implications

Aspects of contrast effects and rater bias need to be considered in studying the etiology of childhood psychopathology using twin pairs and parental ratings. A previous cross-sectional study on contrast effects for attention problems in a comparable sample of Dutch twins detected a rater contrast effect at age 3. The authors hypothesized that the contrast effect represented a maternal rater bias effect that was dependent on the age of the twins (Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2003). Further, a study in an overlapping sample of 3-year-old Dutch twin pairs showed evidence of sibling interaction for externalizing behavior. The interaction proved to be in a cooperative manner, with twins reinforcing each other's behavior (Van der Valk, Verhulst, Stroet, & Boomsma, 1998). To gain insight into the influences of contrast effects on internalizing and externalizing problem behavior, we fitted cross-sectional models taking this interaction parameter into account for internalizing and externalizing problem behavior at ages 3, 7, 10, and 12. For internalizing behavior in girls at age 7 ($b = .07$) and for externalizing behavior in both girls and boys at age 3 ($b = .11$) only, significant influences of a contrast effect were found. Because these were the only significant finding, contrast effects were not considered in the longitudinal model-fitting procedures.

An explanation for the significance of shared environmental influences, or at least part of it, could be rater bias. Sources of rater bias include stereotyping, using different normative standards, or having certain response styles, that is, judging problem behaviors more or less severely. We expect that rater bias in this sense will be a continuous process that influences the ratings at all ages. Less obvious, but not erasable, is the fact that these types of bias may change over time; for instance, mothers change their opinions on certain kinds of behavior, leading to a change in rating style. This change of rating style could show up as age-specific shared environmental influences at the distinct ages. In order to solve this uncertainty about continuous or age-specific influences of shared environmental influences and to distinguish "real" shared environmental influences from rater bias, longitudinal psychometric models, making use of mother and father ratings, should be used. These models assume that in addition to assessing similar aspects of the child's behavior, each parent assesses a unique aspect of his or her child's behavior. This results in aspects of the child's behavior being similarly assessed by both parents, representing "real" unbiased behavior, as well as factors of rater bias and the unique views of each parent. If the shared environmental influences are a result of maternal rating style, it should show up in the unique shared environmental influences instead of in the shared environmental influences from behavior similarly assessed by both parents. Previous cross-sectional studies on the magnitude of rater bias on individual differences in internalizing and externalizing behavior in 3-, 7-, 10-, and 12-year-old twins indicate that influences of rater bias are small but significant, explaining about 10% of the total variance (Bartels et al., 2003, 2004; Van der Valk, van den Oord, Verhulst, & Boomsma, 2001; Van der Valk, van den Oord, Verhulst, & Boomsma, 2003b). It should be noted that the rater bias components of mothers and fathers could be correlated because of the combination of two mechanisms. First, parents tend to have similar levels of psychopathology. Significant spousal correlations are found for internalizing behaviors such as depres-

sion and anxiety as well as for externalizing behaviors such as antisocial behavior (Dufouil & Alperovitch, 2000; Krueger, Moffitt, Caspi, Bleske, & Silva, 1998; Mathews & Reus, 2001; Stallings et al., 1997). These correlations could result either from assortative mating or contagion/interaction effects. Second, levels of parents' psychopathology affect ratings of problem behavior in their children. Several studies suggest that depression in mothers may lead to their overestimating their children's symptomatology (Fergusson & Horwood, 1987). In one study (Breslau, Davis, & Prabucki, 1988), mothers who were depressed rated their children as showing a greater number of symptoms of all psychiatric syndromes. Like mothers' reports, fathers' reports of their children's behavioral problems are influenced by their own level of psychological symptoms (Jensen, Traylor, Xenakis, & Davis, 1988; Phares, Compas, & Howell, 1989). Because this shared rater bias component will affect MZ and DZ twin correlations in the same way, it will show up as shared environmental effects on the common part of the parental ratings. The inclusion of measures of parental psychopathology or the use of different types of raters, such as teachers, will be helpful in accounting for these correlated rater bias effects. Teachers observe the behaviors of the child in a different setting, resulting in a more complete picture of the child's behavior when both parental and teacher ratings are used. However, it should be mentioned that although the impact of contrast effects, rater bias, and assortative mating correlations could be significant, it is most often low, so we do not expect this phenomenon to significantly overshadow our results.

Further, our analyses were performed on a nonclinical sample. If one assumes that psychopathology is caused by environmental hazards or pathogenic genes that are qualitatively distinct from those that cause variation in the normal range (Rutter et al., 1990), our results would have little clinical importance. However, there is evidence that clearly suggests links between normal and abnormal behavior. First, several CBCL studies have shown correlations between behavior problem syndromes and *Diagnostic and Statistical Manual of Mental Disorders* diagnoses (Costello, Edelbrock, & Costello, 1985; Edelbrock & Costello, 1988; Ferdinand, Stijnen, Verhulst, & Van der Reijden, 1999; Kasius, Ferdinand, van den Berg, & Verhulst, 1997). This convergence indicates that behavior problem syndromes as studied in this article must be relevant for psychiatric conditions. Second, several studies supported the view that the sources of normal variation may also affect psychopathology in children and adolescents. So latent class analyses have been used to identify subgroups of individuals with normal or pathological behavior (Eaves et al., 1993; Hudziak et al., 1998; Neuman et al., 1999). Results tend to suggest that these groups differ in degree rather than in kind. Furthermore, using methods from item response theory, Van den Oord, Pickles, and Waldman (2003) found that liability distributions for behavior and emotional problems showed very little or no evidence of nonnormality. This also seems to suggest that psychopathology may often be an extreme on the same continuum that describes variation in the normal range. Thus, although we used a nonclinical sample, it can be argued that our longitudinal analyses are also important for understanding psychopathology.

Longitudinal behavior genetic analyses provide knowledge about the mechanisms underlying stability and change in problem behavior. Our finding of different developmental patterns for the distinct sources of variance has important implications for the

prevention of later maladjustment. The shared environmental influences, for instance, exert a continuous influence from their time of onset. So the children who continue to experience adverse shared environment are at risk for later maladjustment. For additive genetic influences, parts of previous effects are transmitted to later ages. However, the genetic influence is less static owing to new genetic influences that come into play at each age. Nonshared environmental influences seem to be important for age-specific behavior problems and have almost no developmental significance. This implies that influences of nonshared environment are important but that they are mostly of a transient nature and specific to a moment in time.

Further, this study emphasizes the use of results of behavior genetic studies instead of focusing solely on phenotypic studies. Multivariate behavior genetic analyses of patterns of problem behavior make it possible to divide disordered children into groups that have mainly a genetic, a shared environmental, or a nonshared environmental etiology to make the crucial differential diagnosis (see, e.g., Boomsma, Molenaar, & Orlebeke, 1990; Van den Oord et al., 2000). In combination with the knowledge about mechanisms that underlie stability and change and in combination with results of actual experimental intervention studies with random assignment and appropriate control groups, this division into groups can be useful. For instance, for both internalizing and externalizing problem behavior, continuing genetic and shared environmental effects were important for stability. When these results are generalized, it implies that children with high genetic liability or children who continue to experience an adverse shared environment are especially at risk for later maladjustment. Future research could investigate whether for these children, a wait-and-see policy would be inappropriate and an active intervention would be required.

References

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

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

Asher, S. R. (1990). Recent advances in the study of peer rejection. In S. R. Asher & J. D. Coie (Eds.), *Peer rejection in childhood* (pp. 3–14). Cambridge, England: Cambridge University Press.

Bartels, M., Boomsma, D. I., Hudziak, J. J., Rietveld, M. J. H., van Beijsterveldt, C. E. M., & Van den Oord, E. J. C. G. (2004). Disentangling genetic, environmental, and rater effects on internalizing and externalizing problem behavior in 10-year-old twins. *Twin Research, 7,* 162–175.

Bartels, M., Hudziak, J. J., Boomsma, D. I., Rietveld, M. J. H., van Beijsterveldt, C. E. M., & van den Oord, E. J. C. G. (2003). A study of parent ratings of internalizing and externalizing behavior in 12-year-old twins. *Journal of the American Academy of Child and Adolescent Psychiatry, 42,* 1351–1359.

Bartels, M., Rietveld, M. J. H., Van Baal, G. C. M., & Boomsma, D. I. (2002). Genetic and environmental influences on the development of intelligence. *Behavior Genetics, 32,* 237–249.

Barth, J. M., & Parke, R. D. (1993). Parent–child relationship influences on children's transition to school. *Merrill-Palmer Quarterly, 39,* 173–195.

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

Boomsma, D. I., Molenaar, P. C. M., & Orlebeke, J. F. (1990). Estimation of individual genetic and environmental factor scores. *Genetic Epidemiology, 7,* 83–91.

Boomsma, D. I., Orlebeke, J. F., & Van Baal, G. C. M. (1992). The Dutch twin register: Growth data on weight and height. *Behavior Genetics, 22,* 247–251.

Braungart-Rieker, J., Rende, R., Plomin, R., DeFries, J. C., & Fulker, D. W. (1995). Genetic mediation of longitudinal associations between family environment and childhood behavior problems. *Development and Psychopathology, 36,* 233–245.

Breslau, N., Davis, G. C., & Prabucki, K. (1988). Depressed mothers as informants in family history research. *Psychiatry Research, 24,* 345–349.

Bronfenbrenner, U. (1979). *The ecology of human development.* Cambridge, MA: Harvard University Press.

Campbell, S. B. (1994). Behavior problems in preschool children: A review of recent research. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 36,* 113–149.

Campbell, S. B., March, C. L., Pierce, E., Ewing, I. J., & Szumowski, E. K. (1991). Hard-to-manage preschool boys: Family context and the stability of externalizing behavior. *Journal of Abnormal Child Psychology, 22,* 147–166.

Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of General Psychiatry, 53,* 1033–1039.

Costello, E. J., Edelbrock, C. S., & Costello, A. J. (1985). Validity of the NIMH Diagnostic Interview Schedule for Children: A comparison between psychiatric and pediatric referrals. *Journal of Abnormal Child Psychology, 13,* 579–595.

Costello, E. J., Farmer, E. M. Z., Angold, A., Burns, B. J., & Erkanli, A. (1997). Psychiatric disorders among American Indian and White youth in Appalachia: The Great Smoky Mountains Study. *American Journal of Public Health, 87,* 827–832.

Cowan, P. A., Cowan, C. P., Schultz, M. S., & Heming, G. (1994). Prebirth to preschool family factors in children's adaption to kindergarten. In R. D. Parke & S. G. Kellam (Eds.), *Exploring family relationships with other social contexts* (pp. 75–114). Hillsdale, NJ: Erlbaum.

Dufouil, C., & Alperovitch, A. (2000). Couple similarities for cognitive functioning and psychological health. *Journal of Clinical Epidemiology, 53,* 589–593.

Eaves, L. J., Silberg, J. L., Hewitt, J. K., Rutter, M., Meyer, J. M., Neale, M. C., & Pickles, A. (1993). Analyzing twin resemblance in multisymptom data: Genetic application of a latent class model for symptoms of conduct disorder in juvenile boys. *Behavior Genetics, 23,* 5–19.

Edelbrock, C., & Costello, A. J. (1988). Convergence between statistically derived behavior problem syndromes and child psychiatric diagnoses. *Journal of Abnormal Child Psychology, 16,* 219–231.

Egeland, B., Kalkoske, M., Gottesman, N., & Erickson, M. F. (1990). Preschool behavior problems: Stability and factors accounting for change. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 31,* 891–909.

Eindtoets Basisonderwijs 2002 [Final Test of Primary School Education]. (2002). Arnhem, The Netherlands: Citogroep.

Federman, E. B., Costello, E. J., Angold, A., Farmer, E. M., & Erkanli, A. (1997). Development of substance use and psychiatric comorbidity in an epidemiologic study of White American Indian young adolescents: The Great Smoky Mountains Study. *Drug and Alcohol Dependence, 44*(2–3), 69–78.

Ferdinand, R. F., Stijnen, T., Verhulst, F. C., & Van der Reijden, M. (1999). Associations between behavioural and emotional problems in adolescence and maladjustment in young adulthood. *Journal of Adolescence, 22,* 123–136.

Fergusson, D. M., & Horwood, L. J. (1987). The trait and method com-

ponents of ratings of conduct disorder. Part II: Factors related to the trait component of conduct disorder scores. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 28,* 261–272.

Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1995). The stability of disruptive childhood behaviors. *Journal of Abnormal Child Psychology, 23,* 379–396.

Ghodsian, M., Fogelman, K., Lambert, L., & Tibbenham, A. (1980). Changes in behavior ratings of a national sample of children. *British Journal of Social and Clinical Psychology, 19,* 247–256.

Graham, P., & Rutter, M. (1973). Psychiatric disorder in the young adolescent. *Proceedings of the Royal Society of Medicine, 66,* 1226–1229.

Guttman, L. (1954). A new approach to factor analysis: The Radax. In P. F. Lazarsfeld (Ed.), *Mathematical thinking in the social sciences* (pp. 258–349). Glencoe, IL: Free Press.

Harris, J. R. (1995). Where's the child's environment? A group socialization theory of development. *Psychological Review, 102,* 458–489.

Hermanns, J., & Leu, H. R. (1998). *Family risk and family support: Theory, research, and practice in Germany and in the Netherlands.* Delft, the Netherlands: Eburon.

Hudziak, J. J., Heath, A. C., Madden, P. F., Reich, W., Bucholz, K. K., Slutske, W., et al. (1998). Latent class and factor analysis of DSM-IV ADHD: A twin study of female adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 848–857.

Jensen, P. S., Traylor, J., Xenakis, S. N., & Davis, H. (1988). Child psychopathology rating scales and interrater agreement: I. Parents' gender and psychiatric symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry, 27,* 442–450.

Jöreskog, K. G. (1970). Estimation and testing of simplex models. *British Journal of Mathematical and Statistical Psychology, 23,* 121–145.

Kasius, M. C., Ferdinand, R. F., van den Berg, H., & Verhulst, F. C. (1997). Associations between different diagnostic approaches for child and adolescent psychopathology. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 38,* 625–632.

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

Krueger, R. F., Moffitt, T. E., Caspi, A., Bleske, A. & Silva, P. A. (1998). Assortative mating for antisocial behavior: Development and methodological implications. *Behavior Genetics, 28,* 173–186.

Ladd, G. W., & Price, J. M. (1987). Predicting children's social and school adjustment following the transition from preschool to kindergarten. *Child Development, 58,* 1168–1189.

Lamb, M. E., & Nash, A. (1989). Infant–mother attachment, sociability and peer competence. In T. J. Berndt & G. W. Ladd (Eds.), *Peer relations in development* (pp. 219–246). New York: Wiley.

Lange, K., Westlake, J., & Spence, M. A. (1976). Extension to pedigree analysis: III. Variance components by the scoring method. *Annals of Human Genetics, 48,* 47–59.

Mathews, C. A., & Reus, V. I. (2001). Assortative mating in the affective disorders: A systematic review and meta-analysis. *Comprehensive Psychology, 42,* 257–262.

Muthén, B., & Kaplan, D. (1985). A comparison of some methodologies for the factor analysis of nonnormal Likert variables. *British Journal of Mathematical and Statistical Psychology, 38,* 171–189.

Neale, M. C., Boker, S. M., Xie, G., & Maes, H. H. (1999). *Mx: Statistical modeling* (5th ed.). (Available from Virginia Commonwealth University, Department of Psychiatry, Box 900126, Richmond, VA 23298)

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families.* Dordrecht, The Netherlands: Kluwer Academic.

Neuman, R. J., Todd, R. D., Heath, A. C., Reich, W., Hudziak, J. J., Bucholz, K. K., et al.(1999). Evaluation of ADHD typology in three contrasting samples: A latent class approach. *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 25–33.

O'Connor, T. G., Neiderhiser, J. M., Reiss, D., Hetherington, E. M., & Plomin, R. (1998). Genetic contributions to continuity, change, and co-occurrence of antisocial and depressive symptoms in adolescence. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 39,* 323–336.

Parke, R. D., & Kellam, S. G. (Eds.). (1994). *Exploring family relationships with other social contexts.* Hillsdale, NJ: Erlbaum.

Patterson, G. R., Dishion, T. J., & Yoerger, K. (2000). Adolescent growth in new forms of problem behavior: Macro- and micro-peer dynamics. *Prevention Science, 1,* 3–13.

Peterson, J. L., & Zill, N. (1986). Marital disruption, parent–child relationships, and behavior problems in children. *Journal of Marriage and the Family, 48,* 295–307.

Phares, V., Compas, B. E., & Howell, D. C. (1989). Perspectives on child behavior problems: Comparisons of children's self-reports with parent and teacher reports. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 1,* 68–71.

Plomin, R. (1986). Multivariate analysis and developmental behavioral genetics: Developmental change as well as continuity. *Behavior Genetics, 16,* 25–43.

Plomin, R. (1995). Genetics and children's experiences in the family. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 36,* 33–68.

Plomin, R., & Daniels, D. (1987). Why are children in the same family so different from one another? *Behavioral and Brain Sciences, 10,* 1–60.

Plomin, R., Reiss, D., Hetherington, E. M., & Howe, G. W. (1994). Nature and nurture: Genetic contributions to measures of the family environment. *Developmental Psychology, 30,* 32–43.

Richman, N., Stevenson, J., & Graham, P. J. (1982). *Preschool to school: A behavioral study.* New York: Academic Press.

Rietveld, M. J. H., Hudziak, J. J., Bartels, M., van Beijsterveldt, C. E. M., & Boomsma, D. I. (2003). Heritability of attention problems in children: I. Cross-sectional results from a study of twins, age 3 to 12. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 117B,* 102–113.

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

Rowe, D. C. (1981). Environmental and genetic influences on dimensions of perceived parenting: A twin study. *Developmental Psychology, 17,* 203–208.

Rowe, D. C. (1983). A biometrical analysis of perceptions of family environment: A study of twin and singleton sibling kinship. *Child Development, 54,* 416–423.

Rowe, D. C., & Britt, C. L. (1991). Developmental explanations of delinquent behavior among siblings: Common factor versus transmission mechanisms. *Journal of Quantitative Criminology, 7,* 315–332.

Rowe, D. C., Woulbroun, E. J., & Gullwy, B. L. (1994). Peers and friends as nonshared environmental influences. In E. M. Hetherington, D. Reiss, & R. Plomin (Eds.), *Separate social worlds of siblings: The impact of nonshared environment on development* (pp. 159–173). Hillsdale, NJ: Erlbaum.

Rutter, M., Macdonald, H., Le Couteur, A., Harrington, R., Bolton, P., & Bailey, A. (1990). Genetic factors in child psychiatric disorders: II. Empirical findings. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 31,* 39–83.

Schmitz, S., Fulker, D. W., & Mrazek, D. A. (1995). Problem behavior in early and middle childhood: An initial behavior genetic analysis. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 36,* 1443–1458.

Schneider, B. H. (1993). *Children's social competence in context: The contribution of family, school, and culture.* Oxford, England: Pergamon Press.

Sroufe, L. A. (1990). Considering normal and abnormal together: The

essence of developmental psychopathology. *Development and Psychopathology, 2,* 335–347.

Stallings, M. C., Cherny, S. S., Young, S. E., Miles, D. R., Hewitt, J. K., & Fulker, D. W. (1997). The familial aggregation of depressive symptoms, antisocial behavior, and alcohol abuse. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 74B,* 183–191.

Van den Oord, E. J. C. G., Pickles, A. P., & Waldman, I. D. (2003). Normal variation and abnormality: An empirical study of the liability distributions underlying depression and delinquency. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 44,* 180–192.

Van den Oord, E. J. C. G., & Rowe, D. C. (1997). Continuity and change in children's social maladjustment: A developmental behavior genetic study. *Developmental Psychology, 33,* 319–332.

Van den Oord, E. J. C. G., & Rowe, D. C. (1998). An examination of genotype–environment interactions for academic achievement in a U. S. National Longitudinal Survey. *Intelligence, 25,* 205–228.

Van den Oord, E. J. C. G., & Rowe, D. C. (1999). A cousin study of associations between family demographic characteristics and children's intellectual ability. *Intelligence, 27,* 251–266.

Van den Oord, E. J. C. G., Simonoff, E., Eaves, L. J., Pickles, A., Silberg, J., & Maes, H. (2000). An evaluation of different approaches for behavior genetic analyses with psychiatric symptom scores. *Behavior Genetics, 30,* 1–18.

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

Van der Valk, J. C., van den Oord, E. J. C. G., Verhulst, F. C., & Boomsma, D. I. (2003a). Genetic and environmental contributions to stability and change in children's internalizing and externalizing prob-
lems. *Journal of the American Academy of Child and Adolescent Psychiatry, 42,* 1212–1220.

Van der Valk, J. C., van den Oord, E. J. C. G., Verhulst, F. C., & Boomsma, D. I. (2003b). Using common and unique parental views to study the etiology of 7-year-old twins' internalizing and externalizing problems. *Behavior Genetics, 33,* 409–420.

Van der Valk, J. C., Verhulst, F. C., Neale, M. C., & Boomsma, D. I. (1998). Longitudinal genetic analysis of problem behavior in biologically related and unrelated adoptees. *Behavior Genetics, 28,* 365–380.

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

Verhulst, F. C., & Van der Ende, J. (1992a). Six-year developmental course of internalizing and externalizing problem behavior. *Journal of the American Academy of Child and Adolescent Psychiatry, 31,* 927–931.

Verhulst, F. C., & Van der Ende, J. (1992b). Six-year stability of parent reported problem behavior in an epidemiological sample. *Journal of Abnormal Child Psychology, 20,* 595–610.

Verhulst, F. C., Van der Ende, J., & Koot, H. M. (1996). *Handleiding voor de CBCL/4–18* [Dutch manual for the CBCL/4–18]. Rotterdam, The Netherlands: Academic Medical Centre Rotterdam / Erasmus University, Sophia Children's Hospital, Department of Child Psychiatry.

Wilson, R. S. (1986). Continuity and change in cognitive ability profile. *Behavior Genetics, 16,* 45–60.

Wright, J. (1988, April 5). Centre gets $4 million grant to study delinquency. *The Register Guard.*