Longitudinal Genetic Analysis of Internalizing and Externalizing Problem Behavior in Adopted Biologically Related and Unrelated Sibling Pairs

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To obtain a better understanding of how genetic and environmental processes are involved in the stability and change in problem behavior from early adolescence into adulthood, studies with genetically informative samples are important. The present study used parent-reported data on internalizing and externalizing problem behavior of adoptees at mean ages 12.4, 15.5 and 26.3. In this adoption study adopted biologically related sibling pairs shared on average 50% of their genes and were brought up in the same family environment, whereas adopted biologically unrelated sibling pairs only shared their family environment. The resemblance between these adopted biologically related (N = 106) and unrelated sibling pairs (N = 230) was compared and examined over time. We aimed to investigate (1) to what extent are internalizing and externalizing problem behavior stable from early adolescence into adulthood, and (2) whether the same or different genetic and environmental factors affect these problem behaviors at the 3 assessments. Our results show that both internalizing (rs ranging from .34 to .58) and externalizing behavior (rs ranging from .47 to .69) were rather stable over time. For internalizing and externalizing problem behavior it was found that both genetic and shared environmental influences could be modeled by an underlying common factor, which explained variance in problem behavior from early adolescence into adulthood and accounted for stability over time. The nonshared environmental influences were best modeled by a Cholesky decomposition for internalizing behavior, whereas a time-specific influence of the nonshared environment was included in the final model of externalizing behavior.

Problem behavior shows considerable continuity from childhood to adolescence, among which externalizing problem behaviors, such as oppositional, antisocial or aggressive behavior, are the most common and persistent forms of childhood maladjustment (Campbell, 1995; Esser et al., 1990; Verhulst et al., 1993). Some studies are even suggestive of stability of problem behavior into early adulthood, although only few longitudinal general population studies exist that used comparable measures of psychopathology from adolescence through adulthood. For instance, Achenbach et al. (1995) tested the 6-year continuities and predictive paths of syndromes assessed with the Child Behavior Checklist (CBCL; Achenbach, 1991) from adolescents at 13 to 16 years of age to adulthood, when subjects were assessed with the Young Adult Self-Report (Achenbach, 1997) and the Young Adult Behavior Checklist (YABCL; Achenbach, 1997). It was found that young adult syndrome scores on Withdrawn, Somatic Complaints, Anxious/Depressed, Delinquent Behavior, and Aggressive Behavior were strongly predicted by their adolescent counterpart, with a mean r of .59 for all scales (Achenbach et al., 1995). Ferdinand and Verhulst (1995) also examined the 8-year stability of psychopathology from adolescence into young adulthood. They found that 27.3% of young adults with total problem scores in the deviant range had similar total problem scores 8 years earlier. In the same study sample Hofstra et al. (2000) found that 41% of subjects who showed deviant behavior at age 4 to 16 were classified as deviant 14 years later, and Roza et al. (2003) predicted the onset of DSM-IV mood and anxiety disorders across this 14-year period. Thus, for several types of problem behavior some continuity and stability from childhood into adolescence and early adulthood exists.

Despite continuity in behavior, many children with problem behavior also show changes over time. A review of Koot (1995) indicated that approximately

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50% of children demonstrate improvements in behavior over time. Indeed, the above-mentioned studies of Hofstra et al. (2000) and Ferdinand and Verhulst (1995) also show that a majority of children were not regarded as deviant 8 to 14 years later. Most probably, therefore, the developmental trajectory of problem behavior is best described by both continuity and change.

To obtain a better understanding of how genetic and environmental processes are involved in the stability and change in problem behavior over the course of development from childhood into early adulthood, studies with genetically informative samples, such as twins, are important. Most previous studies have used longitudinal twin models to describe the genetic and environmental contributions to stability and change in problem behavior from childhood into (early) adolescence. These studies can reveal whether new, additional genetic factors are expressed as children grow older and whether stability in behavior is due to the same genetic and environmental influences over the years. In order to do so, several models have been used. The *transmission model* assumes that subsequent levels of functioning are causally linked and earlier experiences successively add to the new level (Bartels et al., 2004). Moreover, in this model, new age-specific influences may also exist and may explain changes (Bartels et al., 2004). In contrast, the *common factor model* (e.g., Van den Oord & Rowe, 1997) describes a stable underlying factor or liability that explains the overlap between successive levels of problem behavior over time. For instance, in a longitudinal twin study in which participants were assessed by their parents at ages 3, 7, 10 and 12, Bartels et al. (2004) showed that genes that were expressed at an earlier age were still active at the next time point and thereby contributed to stability in internalizing (43%) and externalizing (60%) behavior. In addition, a common set of shared environmental factors contributed to 47% of the explained variance of internalizing behavior and 34% of the explained variance of externalizing behavior at all ages. Change in behavior was due to age-specific genetic (explained variance ranged between 13%–40%) and environmental influences (explained variance ranged between 9%–28%). In the same sample of Dutch twins, van Beijsterveldt et al. (2003) focused especially on causes of stability and change of aggression. It was found that childhood aggression showed a strong stability, with correlation coefficients ranging from .41 to .77 across varying intervals, which was due to transmission of existing genetic effects (explained variance ranged between 18% and 53%) and age-specific genetic influences (explained variance ranged between 14% and 48%). The influence of shared environmental factors was modest, 25%, and was best modeled by a common factor, which implies that the same set of shared environmental factors are influencing aggression at each age. Nonshared environmental factors hardly contributed to aggression in childhood. Using teacher reports on behavior of another twin sample at the ages of 7, 8, 9, 10, 11 and 12, Haberstick et al. (2005) found that stability in externalizing behavior resulted from a common genetic factor, that explained 43% to 62% of variance, and some age-to-age transmission of early nonshared environmental effects (explained variance ranged between 38% to 57%), whereas change in behavior was largely due to nonshared environmental effects and to a lesser extent explained by age-specific genetic factors. Ratings for internalizing problem behaviors displayed less stability in their study, with *r*s ranging between .10 and .38, and this stability was mainly due to age-to-age transmission of additive genetic effects, that explained between 29% and 48% of variance. Change in internalizing problem behavior resulted from nonshared environmental factors mostly (explained variance ranged between 52% and 71%), and only partly from genetic effects (Haberstick et al., 2005). In summary, we may deduce from previous twin studies focusing on development from childhood until (early) adolescence, that genetic factors contribute primarily to stability in behavior, whereas nonshared environments contribute largely to change (Bartels et al., 2004; Haberstick et al., 2005; Van Beijsterveldt et al., 2003). To our knowledge, no previous studies have focused on the contributions of genetic and shared and nonshared environmental influences to stability and change in problem behavior from early adolescence into adulthood.

Besides twin studies, another research design that can be used to gain insight in genetic and shared and nonshared environmental contributions to behavior is an adoption study, in which resemblance between adopted biologically related and adopted biologically unrelated sibling pairs is compared. Adopted biologically related sibling pairs share on average 50% of their genes and are brought up in the same family environment, whereas adopted biologically unrelated sibling pairs only share their family environment. This research design is most powerful to detect shared environmental influences on behavior. This might be of particular interest when studying the stability and change in problem behavior from adolescence into early adulthood, because marked changes appear in the shared environment of siblings from adolescence onwards, during which period most young adults leave their families and start their own households. Instead of sharing the same home with their sibling(s), and the daily influence of their shared (adoptive) parents, they now start to structure their own individual lives. Thus, the nonshared environmental effects may become more expressed at later ages and may explain concurrent changes in behavior. For the present study we used parent-reported data on internalizing and externalizing problem behavior of adoptees at mean ages 12.4, 15.5 and 26.3, spanning the period of early adolescence into adulthood. Therefore, the present study aims to examine, within

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an adoption design, (1) to what extent are internalizing and externalizing problem behaviors stable from early adolescence into adulthood, and (2) whether the same or different genetic and shared environmental factors affect internalizing and externalizing problem behavior at each assessment.

Materials and Methods

Participants

The present study is nested within a longitudinal study on problem behavior among international adoptees. The longitudinal study sample consisted of children that were legally adopted by nonrelatives in the Netherlands and who were born outside the Netherlands between January 1, 1972 and December 31, 1975. In total 3519 individual children were selected from the central adoption register of the Dutch Ministry of Justice, which keeps the records of all children adopted by Dutch parents. Of the 3309 parents reached, 2148 initially participated in the study (64.9%). This initial sampling procedure of the study has been described in more detail elsewhere (Verhulst et al., 1990). From the total study population, we selected children who were adopted in pairs within one family for the present study. These adopted sibling pairs were either biologically related (i.e., they shared the same mother and father), or biologically unrelated. At the first assessment in 1986, children were aged between 10 and 15 years, with an average age of 12.5 years (*SD* = 1.2) for the adopted biologically related children and 12.4 years (*SD* = 1.2) for the adopted biologically unrelated sibling pairs. The sample was reapproached in 1989 to 1990 for a second assessment (mean age = 15.8 years, *SD* = 1.2, for biologically related and mean age = 15.6, *SD* = 1.2, for biologically unrelated pairs), and in 1999 to 2002 for a third assessment (mean age = 26.4 years, *SD* = 1.2, for biologically related and mean age = 26.2 for biologically unrelated pairs).

The present study is based on the first, second and third assessment. At the third assessment all 2148 adoptees of the original sample were reapproached; 1475 families participated again, of whom 1115 parents and 1406 adoptees filled out questionnaires. In total 288 adoptees and their parents refused to participate and 76 did not respond. A response rate of 72.1% of the baseline sample was yielded, corrected for deceased adoptees, mentally retarded individuals and participants who had immigrated. For the genetic modeling described in the present article, a subset of 111 adopted biologically related and 221 adopted biologically unrelated sibling pairs was used at T1, 75 biologically related and 154 biologically unrelated sibling pairs at T2, and 53 biologically related and 115 biologically unrelated sibling pairs at T3 (Figure 1). With regard to the biologically related siblings, it should be mentioned that we assumed that adopted biologically related sibling pairs had the same biological mother and father, and would therefore share on average 50% of their genes. We had, however, no DNA to affirm a biological relationship between siblings.

A flow-chart of the response of participants is presented in Figure 1.

All adoptees and their parents gave informed consent. The Medical Ethics Committee of the Erasmus Medical Center approved of the study.

Measures

The CBCL (Achenbach, 1991) and YABCL (Achenbach, 1997) were used to assess internalizing and externalizing behavior in adopted biologically related and unrelated sibling pairs. The adoptive parents completed these checklists. Both instruments have the same scales and are designed to evaluate emotional and behavioral problems in individuals of 4 to 18 years, and 18 years and older, respectively. Many YABCL items have counterparts on the CBCL (Achenbach, 1991), but are adapted for ages 18 to 30 years. The YABCL instrument includes young adult analogs of items from the CBCL plus items tapping different developmental paths followed by young adults. Several studies have shown high correlations (*r*s > .40) between CBCL and YABCL broadband scores (Heijmens Visser et al., 2000; Hofstra et al., 2000). For this study we used the broadband Internalizing (consisting of Anxious/Depressed and Withdrawn) and Externalizing (consisting of Intrusive, Delinquent and Aggressive Behavior) groups. The good reliability and validity for the American CBCL and YABCL have been confirmed for the Dutch versions of the CBCL (de Groot et al., 1994; Verhulst et al., 1985, 1996) and YABCL (Heijmens Visser et al., 2000).

Socioeconomic status (SES) was assessed using a 6-point scale of parental occupation (Van Westerlaak et al., 1975), with 1 as the lowest SES. The six levels of parental occupation were further aggregated to a 'low level' (scores 1–2; no education, elementary school, lower vocational education or lower general secondary education), a 'middle level' (scores 3–4; intermediate vocational education, higher general secondary education, or pre-university education) and a 'high level' (scores 5–6; higher vocational education, university).

Data Analyses

Descriptive statistics for background characteristics and internalizing and externalizing behavior at the first, second and third assessment were calculated for each group of adoptees using SPSS 12.0 for Windows. The distributions of the summed internalizing and externalizing problem behavior scales were skewed and therefore logarithmic transformations were applied. After transformation all scales had a skewness and kurtosis between –1.0 and + 1.0. To assess stability of internalizing and externalizing problem behavior from early adolescence into adulthood,

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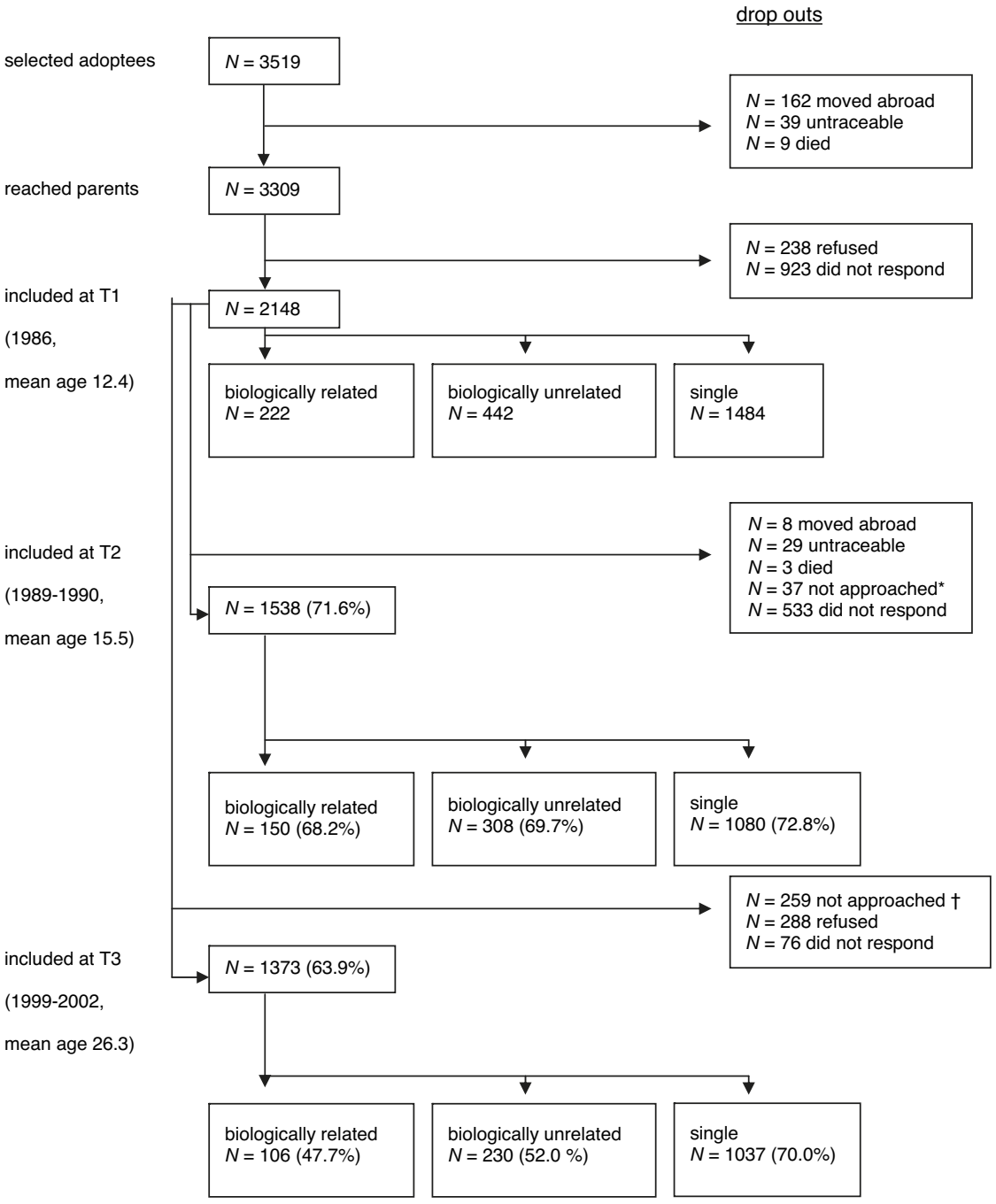

Figure 1

Flow chart of participants.

Note: * 37 adoptive families were not approached because they were already participating in another study at the same moment; † 259 adoptees were not approached of whom 15 had died, 13 were mentally retarded, 72 had emigrated, 100 had withdrawn in an earlier phase, 59 were untraceable and 4 of whom were uncertain that they had been informed of the fact that they had been adopted.

phenotypic correlations and sibling pair correlations were calculated over time, and for the adopted biologically related and unrelated sibling pairs separately. These latter sibling pair correlations give an indication of the genetic and environmental contributions to stability and change in internalizing and externalizing problem behaviors over time.

Genetic Modeling

First, sibling cross-time correlations were calculated (e.g., internalizing score of Sibling 1 at T1 with internalizing score of Sibling 2 at T2), to get a first indication of the importance of genetic and shared environmental factors on the stability of internalizing and externalizing problem behavior. If a

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Table 1

Demographic and Background Factors of Adopted Biologically Related, Biologically Unrelated and Single Adopted Young Adults (*N* = 1475) at T3

	Biologically related (*N* = 143)	Nonbiologically related (*N* = 295)	Singleton (*N* = 1037)	F/χ^2	*df*	*p*
Sex, %, (*N*)						
Female	53.1% (76)	55.9% (165)	55.6% (578)	.34	2	.843
Male	46.9% (67)	44.1% (133)	44.4% (462)			
Mean age at assessment, y, (*SD*)	26.5 (1.1)	26.3 (1.4)	26.2 (1.4)	2.01	2, 1475	.133
Parental SES, %, (*N*)						
Low (score 1 or 2)	7.0% (10)	10.2% (30)	8.8% (92)	9.68	2	.046
Middle (score 3 or 4)	15.4% (22)	26.1% (77)	25.4% (264)			
High (score 5 of 6)	77.6% (111)	63.7% (188)	65.8% (684)			
Country of origin, %, (*N*)						
Korea	51.7% (74)	21.0% (62)	33.8% (352)	140.81	16	<.001
Colombia	27.3% (39)	11.9% (35)	12.4% (129)			
India	2.8% (4)	12.2% (36)	10.6% (110)			
Indonesia	7.7% (11)	8.5% (25)	7.5% (78)			
Bangladesh	2.8% (4)	3.4% (10)	8.5% (88)			
Lebanon	—	11.9% (35)	3.7% (38)			
Austria	.7% (1)	8.5% (25)	3.8% (40)			
Other European	—	4.4% (13)	4.2% (44)			
Other non-European	7.0% (10)	18.3% (54)	15.5% (161)			
Conditions before placement, %, (*N*)						
> 2 changes in caretaking	10.1% (22)	7.0% (30)	7.1% (110)	2.67	2	.264
Physical neglect	46.3% (101)	42.6% (179)	47.4% (654)	2.95	2	.239
Physical abuse	16.8% (35)	11.9% (48)	14.0% (185)	2.93	2	.231
Conditions at placement						
Mean age at placement, y, (*SD*)	3.8 (1.7)	1.7 (1.7)	2.4 (2.0)	58.07	2, 1475	<.001
Poor physical health %, (*N*)	13.8% (18)	17.2% (46)	18.0% (171)	1.41	2	.495

Note: *N* = number; *SD* = Standard Deviation; y = years; *df* = degrees of freedom

cross-correlation is higher for the adopted biologically related sibling pairs than for the adopted biologically unrelated sibling pairs, genetic factors are of importance. If cross-time correlations are similar among biologically related siblings and unrelated siblings, shared environmental factors are of main importance. Second, longitudinal genetic models were fitted to the data to test the contribution of genetic and shared and unshared environmental influences on the stability of internalizing and externalizing problem behavior across the three assessments (mean ages 12.4, age 15.5 and age 26.3). In the present study, we based our models on the phenotypic correlations, sibling correlations, and sibling cross-time correlations to avoid multiple model testing with a relatively small sample size.

Because of our longitudinal design with a long time span between T1-T2 and T3, data from one or more assessments from one member of a sibling pair may be missing from the dataset. We therefore fitted our models to the log-transformed raw data with the maximum-likelihood method using the Mplus statistical software (Muthén & Muthén, 2005).

For each tested model, the total variances and covariances were decomposed into additive genetic (A), shared environmental (C) and nonshared environmental (E) contributions. We used a full model, in which all variance components were expressed as Cholesky decompositions, as a reference model. In this model, the first factor contributes to all three assessments, the second factor influences the second and third assessment, and the third factor is related to the third assessment only. The fit of our models were evaluated in comparison to the full model. We examined the fit of the models for each variance component (A, C, and E) separately. This was done by testing one variance component within a restricted model, while the other variance components were expressed as Cholesky decompositions. Goodness-of-fit of the models was assessed by the likelihood ratio χ^2–tests. Nested models were compared by using the likelihood ratio chi-square test, which uses the difference between –2-log likelihood of the full model from that of the restricted (nested) model, distributed as a χ^2. The degrees of freedom (*df*) for this test are derived by calculating the difference between the number of

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Table 2

Means, Standard Deviations and Sample Sizes for Parent-Reported Internalizing and Externalizing Problem Behaviors at age 12.4, 15.5 and 26.3

	Internalizing behavior			Externalizing behavior		
	Mean	*SD*	*N*	Mean	*SD*	*N*
T1 CBCL: age 12.4						
Biologically related	5.64	5.79	222	7.22	9.11	222
Biologically unrelated	5.70	5.69	442	7.55	8.97	442
T2 CBCL: age 15.5						
Biologically related	7.73	7.31	150	8.55	9.66	150
Biologically unrelated	6.89	6.67	308	8.78	10.33	308
T3 YABCL: age 26.3						
Biologically related	5.99	5.34	98	6.89	10.56	98
Biologically unrelated	5.16	5.06	228	6.39	9.22	228

estimated parameters in the full model and that in the restricted model. Furthermore, the Akaike's Information Criterion (AIC) can be used to select the best model, which will have the lowest value of the AIC. If a restricted model fits the data significantly worse than the full model, the model is rejected.

Results

Attrition

Analyses of adoptees that dropped out of the study showed that significantly more women (74.4%) than men (63.5%) of the original sample participated at the third assessment ($\chi^2 = 28.53$, $df = 1$, $p < .001$) and that dropouts had somewhat lower SES than those who remained (4.51 vs. 4.65, respectively, $t = -2.1$, $p = .037$). Also, the mean CBCL total problems score (the summing of the scores for each of the 118 problem items) at first assessment was significantly higher for the dropouts than for those who remained in the study (mean = 25.42, $SD = 23.49$ vs. mean = 20.15, $SD = 18.66$, $t = 4.78$, $p < .001$). The dropouts had a similar average age as those who remained in the study (12.37 vs. 12.35 respectively, $t = .39$, $p = .696$).

Background Characteristics

Descriptive statistics for background factors are presented in Table 1. There were a few significant differences between the groups of adopted biologically related sibling pairs, adopted biologically unrelated sibling pairs, and single adopted children. Parents of adopted biologically related sibling pairs had a slightly higher SES and their children were significantly older at adoption than in the other groups. Also, the country of origin significantly differed across the groups. Parent report of externalizing problems differed between Colombian and Korean adoptees, with Colombian children scoring significantly higher than Korean. Of the adopted biologically unrelated sibling pairs 87.3% were from the same countries and only six sibling pairs were adopted at the same time. All of the adopted biologically related sibling pairs were adopted at the same time and obviously from the same country of origin.

Descriptives on Internalizing and Externalizing Problem Behavior

The untransformed mean scores on internalizing and externalizing problem behavior assessed with the CBCL (T1 and T2) and the YABCL (T3) are presented in Table 2. No significant differences were found for scores on internalizing and externalizing problem behaviors of adopted biologically related and unrelated sibling pairs (Table 2).

Phenotypic Correlations

In Table 3 the phenotypic correlations of log-transformed internalizing and externalizing scores for each follow-up interval are given separately for biologically related and adopted biologically unrelated sibling pairs. For internalizing problem behavior, correlations between T1 and T2 (with a short time interval of 3.5 years) are highest ($r = .58$ and $r = .55$, for adopted biologically related and unrelated sibling pairs, respectively). Correlations are, however, mostly of similar strength between T1 and T3 (with a time interval of approximately 14 years), and T2 and T3 (with a time

Table 3

Phenotypic Correlations for Internalizing (Above Diagonal) and Externalizing (Below Diagonal) Problem Behavior Across Time for Adopted Biologically Related and Unrelated Sibling Pairs

Internalizing Externalizing	T1	T2	T3
Biologically related			
T1 (age 12.4)	1	.58	.51
T2 (age 15.5)	.58	1	.40
T3 (age 26.3)	.47	.49	1
Biologically unrelated			
T1 (age 12.4)	1	.55	.34
T2 (age 15.5)	.69	1	.50
T3 (age 26.3)	.53	.59	1

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interval of 11 years), with one exception between T1 and T3 for biologically unrelated siblings. Likewise, for externalizing problem behavior, correlations between T1 and T2 are strongest, but only slightly lower between T1 and T3, and T2 and T3. This structure suggests that a transmission model is not likely to explain the development of internalizing or externalizing problem behavior. In such a model, subsequent levels of problem behavior are influenced by prior levels, implying that effects of closely in time related events will be larger, thus predicting higher correlations among adjoining assessments than among those occurring more distantly in time. The common factor model may explain our correlation structure over time, since this model assumes that a common factor exerts its effects at each assessment and does not imply that correlations between assessments vary as function of the length of the time lag.

Table 4

Sibling Correlations (Diagonal) and Sibling Cross-Time Correlations for Internalizing (Above Diagonal) and Externalizing (Below Diagonal) Problem Behavior for Adopted Biologically Related and Unrelated Sibling Pairs

Internalizing Externalizing	T1	T2	T3
Biologically related			
T1 (age 12)	.33[a]/.46[b]	.11	.31
T2 (age 15.5)	.19	.18[a]/.34[b]	.34
T3 (age 26)	.07	.14	.52[a]/.39[b]
Biologically unrelated			
T1 (age 12)	.34[a]/.20[b]	.35	.24
T2 (age 15.5)	.19	.38[a]/.19[b]	.19
T3 (age 26)	.23	.21	.16[a]/.28[b]

Note: [a]internalizing problem behavior; [b]externalizing problem behavior.

Sibling Correlations and Sibling Cross-Time Correlations

Table 4 shows that sibling correlations for internalizing problem behavior are similar between biologically related siblings and unrelated siblings at T1, even lower among biologically related siblings at T2, but much higher among biologically related siblings at T3, when compared to unrelated siblings.

Sibling cross-time correlations for internalizing problem behavior show an inconsistent picture, so no

Table 5

Model Fitting Results of Longitudinal Models for Internalizing and Externalizing Problem Behavior

Model	−2 LL	*df*	Compared to model	$\Delta\chi^2$	Δdf	*p*	AIC
Internalizing							
1. Full	3428.79	33					
A: Cholesky							
C: Cholesky							
E: Cholesky							
2. A: Common factor	3429.69	35	1	0.90	2	*ns*	−3.10
3. C: Common factor	3431.91	36	1	3.12	3	*ns*	−2.88
4. E: Time-specific	3449.86	36	1	21.07	3	< .001	15.07
5. Final model	3436.37	39	1	7.58	6	*ns*	−4.42
A: Common factor							
C: Common factor							
E: Cholesky							
Externalizing							
1. Full	3910.79	33					
A: Cholesky							
C: Cholesky							
E: Cholesky							
2. A: Common factor	3915.24	35	1	4.45	2	*ns*	0.45
3. C: Common factor	3914.76	36	1	3.97	3	*ns*	−2.03
4. E: Time-specific	3913.69	36	1	2.90	3	*ns*	−3.10
5. Final model	3929.16	42	1	18.37	11	*ns*	−3.63
A: Common factor							
C: Common factor							
E: Time-specific							

Note: −2 LL: −2 Log likelihood; *df* = degrees of freedom; A = additive genetic factor; C = common environment factor; E = nonshared environment factor; *ns* = not significant.

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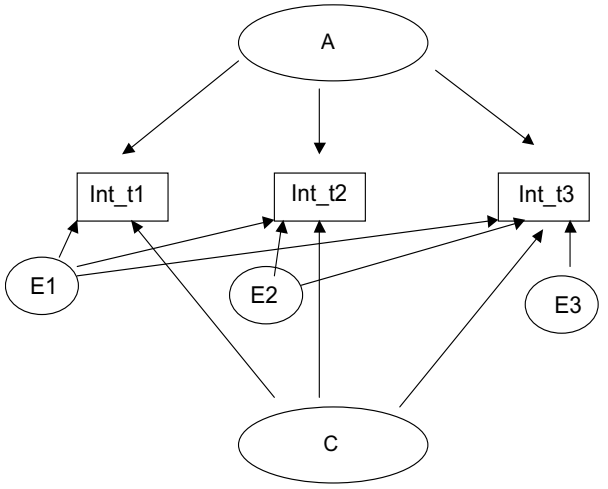

Figure 2

Final model for internalizing problem behavior.

Note: Int_t1, Int_t2, Int_t3: Internalizing behavior at T1, T2 and T3, respectively.

A: additive genetic influence: Modeled by a single common factor.

C: shared environmental influence: Modeled by a single common factor.

E1, E2, E3: nonshared environmental influence at T1, T2 or T3: Modeled by a Cholesky decomposition.

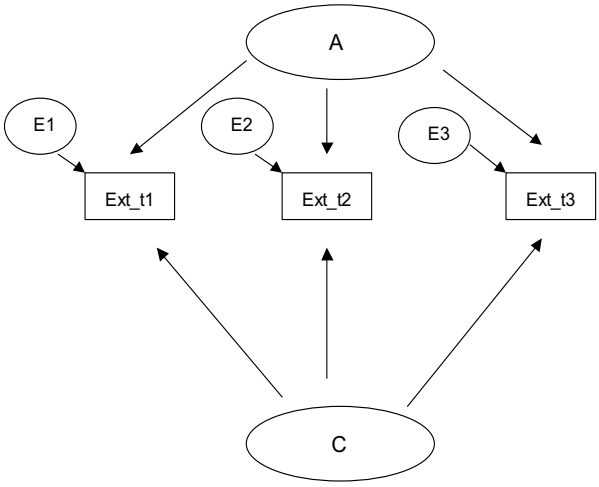

Figure 3

Final model for externalizing problem behavior.

Note: Ext_t1, Ext_t2, Ext_t3: Externalizing behavior at T1, T2, and T3, respectively.

A: additive genetic influence: Modeled by a single common factor.

C: shared environmental influence: Modeled by a single common factor.

E1, E2, E3: nonshared environmental influence at T1, T2 or T3: Modeled by time-specific factors.

conclusion about the underlying model can be drawn on these cross-time correlations.

For externalizing problem behavior, sibling correlations of adopted biologically related sibling pairs are approximately twice as high as those of unrelated siblings at T1 and T2, and higher, but certainly not twice as high, at T3. Sibling cross-time correlations show a less clear picture, with a tendency of lower cross-time correlations between related siblings.

Genetic Modeling

Based on the phenotypic correlations over time, we tested a common factor model for the A and C variance component, and modeled time-specific nonshared environmental (E) factors for externalizing and internalizing problem behavior from adolescence into adulthood.

Model fitting yielded the results presented in Table 5. For *internalizing* problem behavior it was found that both genetic and shared environmental influences could be modeled by an underlying common factor, which explained variance in internalizing behavior from early adolescence into adulthood and accounted for stability over time. The nonshared environmental influences were best modeled by a Cholesky decomposition. The final model is shown in Figure 2. For *externalizing* problem behavior, a common factor model for both genetic and shared environmental influences also fitted the data. These two common factors may account for stability of externalizing behavior over time. Moreover, a time-specific influence of the nonshared environment was included in the final model (Figure 3), which may explain the change in behavior.

The percentages of the total time (or age) specific variance are presented in Table 6. An increase in additive genetic effects and a decrease in shared environmental effects were observed for internalizing problem behavior from age 12.4 (T1; 10.2% and 32%, respectively, for A and C) to age 15.5 (T2; 23% and

Table 6

Contribution of A, C and E to Explained Variance of Internalizing and Externalizing Problem Behavior at First (T1, age 12.4), Second (T2, age 15.5) and Third (T3, age 26.3) Assessment

	T1	T2	T3
Internalizing			
A (common factor)	10.24%	23.04%	22.66%
C (common factor)	32.00%	23.52%	24.21%
E1 (time specific)	57.76%	33.64%	13.62%
E2 (time specific)#	N/A	19.80%	19.97%
E3 (time specific)#	N/A	N/A	19.54%
Externalizing			
A	43.16%	91.58%	28.52%
C	18.15%	1.40%	19.01%
E1	38.69%	N/A	N/A
E2	N/A	7.02%	N/A
E3	N/A	N/A	52.42 %

Note: N/A: not applicable; E1: nonshared environmental influence at T1; E2: nonshared environmental influence at T2; E3: nonshared environmental influence at T3.

To calculate the contribution of the nonshared environmental influence on T2, the percentages of explained variance by E1 and E2 are summed up (33.64 % + 19.80 % = 53.44%); likewise, the contribution of nonshared environmental influence to the total explained variance at T3 is derived by summing up E1, E2 and E2, which results in 52.99%.

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23.5%, for A and C, respectively) and age 26.3 (T3; 22.7% and 24.2%, for A and C, respectively). Thus, the amount of explained variance by A and C was more or less stable from T2 to T3. For externalizing problem behavior, a different pattern was found. At age 12.4 genetic effects explained 43.2% of the variance, while at age 15.5 the contribution of A was more than doubled (91.6%). This contribution was much lower at age 26 when 28.5% of variance could be explained by genetic effects. For the shared environmental influence on externalizing problem behavior, a similar proportion of variance (18%–19%) was explained at T1 and T3, whereas almost no influence of C (1.4%) was found at T2. For internalizing and externalizing behavior, the nonshared environment explained more than 50% of variance at T3. This proportion was rather stable from T1 to T3 for internalizing behavior, whereas for externalizing behavior an increase was found from T1 (38.7%) to T3 (52.4%), but with a very low contribution at T2 (7%).

Discussion

In the present study, we used genetic modeling techniques to describe the processes that may explain stability and change in internalizing and externalizing problem behavior from early adolescence into young adulthood. By using an adoption design in which resemblance between adopted biologically related and adopted biologically unrelated sibling pairs were compared with regard to internalizing and externalizing problem behavior, the present study extended prior twin studies that focused on developmental trajectories of behavior at earlier ages (Bartels et al., 2004; van Beijsterveldt et al., 2003).

Our first aim was to examine to what extent internalizing and externalizing problem behavior were stable from early adolescence, at mean age 12.4, into adulthood, at mean age 26.3. Both internalizing and externalizing problem behaviors tended to increase from age 12.4 to age 15.5, and showed a decline from age 15.5 to adulthood. Despite this trend in mean values, a rather high phenotypic correlation was found over time, with *r*s ranging from .34 to .58 for internalizing behavior and *r*s ranging from .47 to .69 for externalizing behavior. Thus, our findings suggest that internalizing and externalizing problem behavior in our sample of adoptees showed a moderate to high stability from early adolescence into adulthood.

Even though the time span between the first assessment at mean age 12.4 and the last assessment at mean age 26.3 was rather long, the phenotypic correlation was mostly of a similar strength when compared to the correlation between the first and second assessment, which were only 3.5 years apart. This suggested that a transmission model would probably not explain our data. This is in contrast to the findings of Bartels et al. (2004) and van Beijsterveldt et al. (2003), who found evidence for transmission of additive genetic effects on problem behavior during childhood. The main difference between the study by Bartels et al. (2004) and van Beijsterveldt et al. (2003) and the present study is that our study bridged a very different developmental period: from early adolescence into adulthood as compared to the period from childhood into early adolescence in the studies of Bartels et al. (2004) and van Beijsterveldt et al. (2003). This may explain why we found a different developmental model. Based on our phenotypic correlations we decided to test only a common factor model in our relative small sample, to avoid Type I errors that may result from multiple testing.

We found that a common genetic factor may underlie the stability of internalizing and externalizing behavior from age 12 through age 26. The same conclusion may be drawn for the shared environmental influences, which were modeled by a single common factor over time. The latter finding is in line with results from the previous twin studies of Bartels et al. (2004) and van Beijsterveldt et al. (2003). For internalizing problem behavior, the influence of this common genetic factor was lowest at mean age 12.4, whereas its influence was more strongly expressed at age 15.5 and age 26.3. This finding may be in line with the gradual increase in prevalence of internalizing behaviors, in particular depression, from early adolescence onwards, which is most profound in females, as shown in other studies (e.g., Roza et al., 2003). Unfortunately, our small sample size did not allow separating female–female sibling pairs from female–male sibling pairs and male–male sibling pairs, to test whether the expression of a common underlying genetic factor is different for females compared to males.

For externalizing problem behavior, our findings also suggest that a common genetic factor underlies the stability over time. Especially at age 15.5, a genetic factor is strongly expressed and explains almost all variance in externalizing behavior. This finding may be in line with the 'adolescence-limited' type of antisocial behavior, as described by Moffitt (1993). In her theory, Moffitt distinguishes two developmental pathways of antisocial behavior: a life-course persistent pathway, for children who commit antisocial behaviors throughout their lives and an adolescence-limited pathway, for those who only commit antisocial behaviors during adolescence. Thus, genetic influences on certain aspects of externalizing behavior, such as antisocial behavior, may be most expressed during adolescence. In adulthood, the expression of the genetic factor appears to have attenuated and the nonshared, or unique, environment starts to play a more important role. The latter finding may be the result of the marked changes that appear in the shared environment of siblings from adolescence onwards, in which period most young adults leave their families and start their own households. Thus, the nonshared environmental effects may become more expressed at later ages and may explain changes in behavior. It may be, for instance, that the structure and rules of adoptive

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parents in the adolescent period overrule most nonshared environmental influences. In adulthood, individuals have to structure their own lives and individual specific, nonshared environmental factors, consequently gain increased importance. Such an increasing effect for the nonshared environment on internalizing problem behavior may be expected as well, but in the present study, the nonshared environment exerted a rather stable and large influence on internalizing behavior.

Thus, our findings are in line with a common factor model, which describes a stable underlying factor or liability that explains the overlap between successive levels of problem behavior over time. In our study, the time period ranged from early adolescence into adulthood, reflecting an important developmental period in which an own identity is developed (e.g., Eriksson, 1968), relationships with others than (adoptive) parents gain in importance, and finally, autonomy and independence are to be achieved. Our findings suggest that, within this developmental period, an underlying genetic and shared environmental factor explains stability in behavior, whereas the nonshared environmental influences at each specific age-period may account for changes over time. The latter finding with regard to the nonshared influences was also supported by a study of Haberstick et al. (2005), albeit in a different developmental period.

The present study has some limitations that should be taken into account. First, our loss to follow-up may have caused selection bias in which individuals with less problem behavior remained in the study and those with more problems dropped out. This may have influenced the estimates in our models. Also, we focused on parental ratings of problem behavior of adolescents and adults, because we had these rating available at each assessment. Rater bias may have influenced our outcomes in a continuous way, that is, by influencing the ratings similarly at each assessment. Alternatively, rater bias may change over time, leading to different rating styles at different ages. Such a change in rater bias may be particularly at stake when the adoptees are adults and are no longer living at home with their adoptive parents. Parents may be less aware of how their adoptive children behave as adults, and may consequently rate less problem behavior when compared to the adolescent period. However, in a previous cross-sectional study (van den Berg et al., submitted), we focused on T3 data based on self-reported and parent-reported problem behavior and found that very comparable models fitted to data from both kinds of informants. Thus, most probably, the rating bias did not affect our genetic models in the present study. Furthermore, the mean scores on internalizing and externalizing behavior of adoptees were higher than those of the general Dutch population. This may be partly explained by the adverse circumstances in which the majority of these children lived the first part of their lives (Tieman et al., 2005). This is also evident from the high prevalences of physical neglect and physical abuse that were reported among these adoptees (Table 1). Thus, we cannot generalize our findings to the general population. Another limitation of the current study is the fact that the biologically related adoptees were significantly older at the time of adoption and were always adopted from the same country. In addition to genetic factors, these two factors may make biologically related siblings more alike than unrelated siblings, although it should be mentioned that of the adopted biologically unrelated sibling pairs also 87.3% were from the same country. Finally, we may have underestimated genetic influences by assuming that adopted biologically related sibling pairs had the same biological mother and father, and would therefore share on average 50% of their genes. We had no DNA to affirm a biological relationship between siblings.

In sum, our study is the first to model genetic and environmental effects on stability and change of internalizing and externalizing behavior from early adolescence into adulthood. Both behaviors show a moderate to high stability over a period of 14 years, and are probably influenced by a common underlying genetic and a common shared environmental factor, in addition to nonshared environmental factors, which accounted for a large part of the variance.

It is of interest to gain more insight into these shared and nonshared environmental influences, since they may offer tools for prevention and intervention programs.

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