Developmental Psychology
1999, Vol. 35, No. 3, 680–692

0012-1649/99/$3.00

Relationships Between Parenting and Adolescent Adjustment Over Time: Genetic and Environmental Contributions

Jenae M. Neiderhiser and David Reiss
George Washington University

E. Mavis Hetherington
University of Virginia

Robert Plomin
Institute of Psychiatry

The predictive association between parenting and adolescent adjustment has been assumed to be environmental; however, genetic and environmental contributions have not been examined. This article represents one effort to examine these associations in which a genetically informative design was used. Participants were 395 families with adolescent siblings who participated in the Nonshared Environment in Adolescent Development (D. Reiss et al., 1994) project at 2 times of assessment, 3 years apart. There were 5 sibling types in 2 types of families: 63 identical twins, 75 fraternal twins, and 58 full siblings in nondivorced families and 95 full, 60 half, and 44 genetically unrelated siblings in stepfamilies. Results indicate that the cross-lagged associations between parental conflict–negativity and adolescent antisocial behavior and depressive symptoms can be explained primarily by genetic factors. These findings emphasize the need to recognize and examine the impact that adolescents have on parenting and the contribution of genetic factors to developmental change.

Understanding the pathways to adolescent maladjustment (e.g., antisocial behavior and depression) has been an important focus of developmental research. The role of family process, especially parenting, in both producing and maintaining psychopathology during adolescence has also been explored extensively. One of the clearest and most replicated associations in this area is between conflictual parent–child relationships and adolescent maladjustment. For example, harsh, inconsistent, and ineffective discipline has been associated with later antisocial behavior (Loeber & Dishion, 1983; Patterson, DeBaryshe, & Ramsey, 1989), whereas insecure parent–child relationships, including parental rejection and forcefulness, have been traced to later internalizing problems (Rubin, Hymel, Mills, & Rose-Krasnor, 1991).

Until recently this association has been considered unidirectionally, such that parent–child conflict was thought to lead to negative adolescent adjustment for primarily "family environmental" reasons. Evidence of genetic contributions to parent–child relationships and to environmental measures in general (Plomin, 1994; Plomin, Reiss, Hetherington, & Howe, 1994), coupled with evidence of bidirectional coercive family interaction patterns (Patterson, 1982), has led to a shift in perspective that is likely to be more tenable. In this vein, Haapasalo and Tremblay (1994) described the pathway to aggression as a combination of predisposing individual factors, including genetic contributions, and "aggression-promoting learning conditions," such as conflictual parent–child relationships. Although this is a step in the right direction, Plomin (1994) and Reiss (1995), among others, have challenged researchers to consider the possibility that genetic factors contribute not only to individual characteristics but also to interpersonal relationships and, more important, to the associations between interpersonal relationships and individual behavioral characteristics.

One of the first studies to examine systematically genetic and environmental contributions to associations between parent–child relationships and adolescent adjustment was conducted by Pike, McGuire, Hetherington, Reiss, and Plomin (1996). All of the associations between conflictual parent–child relationships and adolescent antisocial behavior and depressive symptoms showed evidence of substantial and significant genetic contributions. In other words, the same genetic factors in the adolescents that contributed to conflictual parent–child relationships also contributed to the adolescents' maladjustment.

A next step in the effort to trace the pathway to adolescent maladjustment is to examine these data longitudinally. There are three possible directional hypotheses for associations between parent–child relationships and adolescent adjustment. The first hypothesis has guided much of the research in this area: Conflictual parent–child relationships result in adolescent adjustment

Jenae M. Neiderhiser and David Reiss, Center for Family Research, Department of Psychiatry and Behavioral Sciences, George Washington University; E. Mavis Hetherington, Department of Psychology, University of Virginia; Robert Plomin, Social, Genetic and Developmental Psychiatry Research Centre, Institute of Psychiatry, London, England.

This article is part of the Nonshared Environment in Adolescent Development project, supported by the National Institutes of Mental Health (5R01MH43373 and 5R01MH48825) and the William T. Grant Foundation.

Correspondence concerning this article should be addressed to Jenae M. Neiderhiser, Center for Family Research, Department of Psychiatry and Behavioral Sciences, George Washington University, 2300 Eye Street, NW, Room 613, Ross Hall, Washington, DC 20037. Electronic mail may be sent to jenaemn@gwis2.circ.gwu.edu.

problems (Baumrind, 1991; Loeber & Stouthamer-Loeber, 1986; Stattin & Klackenberg-Larsson, 1990). A second hypothesis proposes that adolescents who are disruptive or depressed elicit negativity from their parents (Hetherington & Clingempeel, 1992; Patterson, 1982). Finally, the third hypothesis suggests that the associations between parent–child relationships and adolescent adjustment may be bidirectional. Studies that have explored this third hypothesis have found evidence for a coercive family interaction pattern in which the parents' behavior both influences and is influenced by the adolescents' behavior (Patterson, 1982; Vuchinich, Bank, & Patterson, 1992).

A recent article by Reiss et al. (1995) emphasized and detailed the importance of families to adolescent mental health. There were two issues that were motivated, but could not be answered, by that study. First, does parental behavior lead to certain patterns of adolescent adjustment, or do certain patterns of adolescent adjustment lead to specific parental behaviors? As described previously, our analysis attempts to address that question. Second, because genetic models were not examined, the nonshared family effects reported by Reiss et al. may have been due to nonshared genetic factors as well as to nonshared environmental factors. Nonshared environmental factors are defined as all nongenetic influences that cause siblings to differ from one another.

In the current article we examine the associations between parental conflict–negativity and adolescent adjustment by using a cross-lagged genetic model. In a phenotypical sense, a cross-lagged model is designed to examine the longitudinal association between two different measures independent of stability in both measures and of the contemporaneous associations (Figure 1). The cross-lagged genetic model is designed to operate similarly while estimating genetic and environmental influences on the associations between measures. A test of the directional hypotheses described earlier is possible through the use of this model. Our approach to the cross-lagged model is a very conservative one (Kenny, 1979). The most likely result of the use of this more conservative approach is a decrease in the effect size of the cross-lagged paths. Although this approach may decrease the likelihood of finding significant effects, we believe that this is the most appropriate model to examine the research questions proposed in the present article.

The cross-lagged path that is the focus of this article is the path from parental conflict–negativity to adolescent adjustment 3 years later (l1). This path was chosen because most research that has examined the associations between parenting and adolescent adjustment has considered parenting as a predictor. It is also possible, however, that adolescent adjustment can contribute to change in parental conflict–negativity (l2). Because the majority of research has considered the path from parenting to change in adjustment, the first model is presented in detail and is then compared with the alternate model (the path from adjustment to change in parenting).

On the basis of previous results from this study and others, we expected that genetic factors would make important contributions to the contemporaneous associations between parenting and adolescent adjustment, to the stability from Time 1 to Time 2 of parenting, and to the stability of adjustment. Lacking any precedent for a prediction of genetic influences on cross-lagged associations, our predictions were shaped by the prevailing literature: We expected environmental factors to account for most, if not all, of the cross-lagged associations.

Method

The data used in this analysis were obtained from two measurement occasions 3 years apart from the Nonshared Environment in Adolescent Development (NEAD; Reiss et al., 1994) project, which includes extensive environmental and developmental outcome assessments.

Sample

The sample consisted of 395 families who participated at both times of data collection. Families included five sibling types residing in two types of families: 63 identical (MZ) twins, 75 fraternal (DZ) twins, and 58 full siblings (FI) in nondivorced families and 95 full siblings (FS), 60 half siblings (HS), and 44 genetically unrelated siblings (US) in stepfamilies. The sibling types differ according to the degree of genetic relatedness: MZ twins share 100% of their segregation genes, DZ twins and full siblings share 50%, HS share 25%, and US share none of their segregating genes.

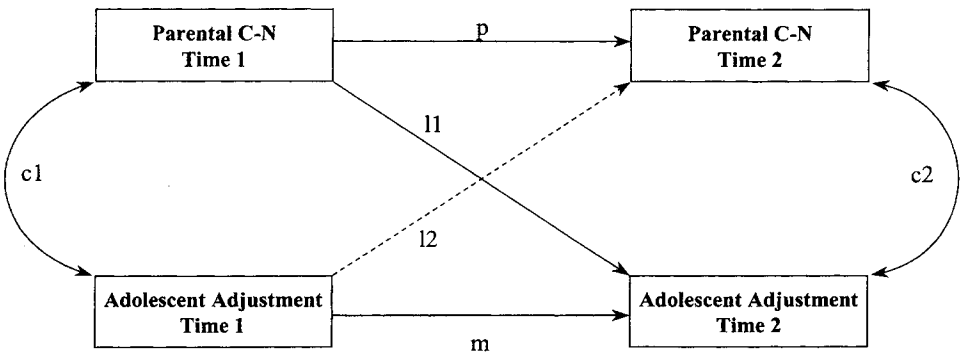

Figure 1. Phenotypic cross-lagged model for parental conflict–negativity (C–N) and adolescent maladjustment. Stability for parenting is indicated by the paths labeled p and for adolescent maladjustment by the paths labeled m. The contemporaneous associations are indicated by the paths labeled c1 and c2 for Time 1 and Time 2, respectively. Finally, the cross-lagged path is represented by the path l1, from parental conflict–negativity at Time 1 to adolescent maladjustment at Time 2. The cross-lagged path indicated by the dashed line (l2) represents an alternative direction for the cross-lagged association.

The nondivorced families with full siblings were initially recruited through random digit dialing of 10,000 telephone numbers throughout the United States. A small subsample of the other siblings types was also drawn from random digit-dialing procedures; however, most of the other sibling types were recruited through a national market survey of 675,000 families. These sources yielded the Time 1 sample of 720 families.

The 720 families were primarily middle class (average family income ranged from $25,000 to $35,000—with 12% earning less than $20,000/year and 32% earning more than $50,000/year), reflecting the marital stability criteria—and were primarily Caucasian (94% of the mothers and 93% of the fathers). The average years of education were 13.6 for mothers and 14.0 for fathers. Only a few parents received less than a high school education (7% of mothers and 10% of fathers), somewhat more completed their education with high school graduation (42% of mothers and 35% of fathers), and the remainder received at least some post–high school education. Table 1 includes detailed information about the sample at both times of measurement. Further details regarding the sampling strategy and recruitment can be found elsewhere (Plomin et al., 1994; Reiss et al., 1995).

To be included in Time 2 data collection, 3 years later, both of the target adolescents were required to reside in the home at least half of the time. Failure to meet these criteria resulted in a reduced sample of 395 families (15% of the ineligible families experienced a divorce, 79% of the adolescents had moved out of the home, and the remaining 6% were unable to be classified). Only 9% of the families who were eligible to participate at Time 2 refused to do so. There were no mean differences in demographic characteristics (parents' education, family income, sex of the siblings, and age difference between siblings) for families who were eligible for participation only at Time 1 versus families who were eligible to participate at both times, $F(5, 681) = 1.26$, $p < .28$. For the 27 eligible families who refused to participate at Time 2, analyses indicated significant main effects for age and variables related to age (i.e., the adolescents were older and received less parental monitoring) when compared with eligible families who chose to participate at Time 2.

Table 1
Sample Characteristics for the Nonshared Environment in Adolescent Development Project at Times 1 and 2

Sample	Time 1	Time 2[a]
Adolescent characteristics[b]		
Age range	10–18	13–21
Mean age for Child 1	13.5 ± 2.0	16.2 ± 2.1
Mean age for Child 2	12.1 ± 1.3	14.7 ± 1.9
Mean age difference	1.61 ± 1.29	1.47 ± 1.34
Female sibling pairs (%)	48.4	49.4
Parent characteristics[c]		
Existence of stepfamilies		
Mean length of time	8.5 ± 3	11.6 ± 3.5
Range	5–18	8–21
Mother's age	38.1 ± 5.2	40.5 ± 4.8
Father's age	41.0 ± 6.5	43.0 ± 6.1
Mother's education	13.8 ± 2.3	13.9 ± 2.4
Father's education	13.9 ± 2.7	14.0 ± 2.6
Family income (*Mdn*)	$25,000–$35,000	$25,000–$35,000

Note. All characteristics presented in years unless otherwise indicated. [a] Represents a time period 3 years later. [b] Sibling pairs were required to be the same sex and to reside in the home at least half of the time. [c] Both parents were required to reside together in the home.

Procedure

At Time 1, most families were visited twice by two interviewers, whereas all families who participated in the Time 2 assessment were visited once by one interviewer. Both parents and the two adolescents completed questionnaires and were videotaped during the visit. Additional questionnaire data were obtained from take-home questionnaires, which were mailed ahead and collected by the interviewer. Family members specified areas of disagreement that were then discussed and videotaped in 10-min dyadic, triadic, and tetradic combinations. A global coding system of 5-point Likert scales (Hetherington & Clingempeel, 1992) was used to rate the videotaped interactions. In the present study we focused on the dyadic interactions between the parents and the adolescents.

Twins were rated for physical similarity (e.g., eye and hair color) by the interviewer, the parents, and the self-reports using a questionnaire designed for adolescents (Nichols & Bilbro, 1966). If any differences in physical characteristics were reported or if respondents reported that people never were confused about the identity of the twins, the twin pair was classified as dizygotic. Ten of the twin pairs could not be classified as either monozygotic or dizygotic and were excluded from these analyses (7% of the twin pairs). Questionnaire methods of assigning zygosity have been found to be at least 90% accurate when compared with tests of single-gene markers in blood phenotypes (Nichols & Bilbro, 1966; Spitz et al., 1996).

Measures

To obtain ratings of parenting and adolescent adjustment that were less likely to be measure or rater specific, we created composite measures. Table 2 describes the measures used to create the composites, including reliability coefficients. The composites were created by summing the standardized scores across all of the respondents for maternal conflict–negativity (mother, adolescent, and observer ratings), paternal conflict–negativity (father, adolescent, and observer ratings), and antisocial behavior and depressive symptoms (mother, father, adolescent, and observer ratings). More detailed descriptions of the measures and the construction of the composites can be found in previous reports from NEAD (Pike et al., 1996; Plomin et al., 1994; Reiss et al., 1995).

Analyses

Before genetic analysis, phenotypic cross-lagged correlations were examined to determine whether they were of sufficient magnitude to be included in genetic analyses. Genetic and environmental influences on associations between measures are limited by the size of the phenotypic correlation; that is, genetic and environmental influences on the covariance between two measures can only be present if the two measures covary at least modestly (Plomin, DeFries, & McClearn, 1990). Once phenotypic association was established, genetic and environmental effects on these cross-lagged associations were examined by using multivariate genetic model-fitting techniques.

Figure 2 represents the genetic model that includes the cross-lagged association between parenting at Time 1 and adolescent adjustment at Time 2 and is described in detail later. It should be noted that the alternate model represented by Figure 3 operates in the same way.

The cross-lagged genetic model was designed to operate in two steps. First, genetic and environmental influences that contribute to the stability of parenting and maladjustment and to the contemporaneous associations between the two at each time of measurement were estimated. Second, genetic and environmental influences on the cross-lagged association were estimated. This second step results in an estimate of genetic and environmental contributions to the cross-lagged association that is independent of stability and the contemporaneous associations. In other words, in which Figure 2 is used as an example, to what degree are the same genetic and environmental factors that influence conflictual parent–child relationships

Table 2
Summary of Composite Measures

Measure[a]	Reference	Subscale	Item type	Reliability[b]
Family environment: Parental conflict–negativity				
Parent Discipline Behavior	Hetherington & Clingempeel (1992)	Coercion	7-point Likert	α = .85–.92
		Punitiveness	7-point Likert	α = .82–.87
Conflict Tactics Scale	Strauss (1979)	Symbolic aggression	5-point Likert	α = .72–.95
Parent–Child Relationships	Hetherington & Clingempeel (1992)	Conflict	7-point Likert	α = .72–.78
Videotaped dyadic interactions	Hetherington & Clingempeel (1992)	Anger–hostility, coercion, and transactional conflict	Global coding, 5-point Likert	κ = .55–.73 IC = .59–.68
Adolescent adjustment: Antisocial behavior				
Behavior Problems Index	Zill (1985)	Antisocial behavior	3-point Likert	α = .72–.78
Videotaped dyadic interactions	Hetherington & Clingempeel (1992)	Disrespectful, disruptive, or both	Global coding, 5-point Likert	κ = .65–.77 IC = .79
Adolescent adjustment: Depressive symptoms				
Behavior Problems Index	Zill (1985)	Depressed behavior	3-point Likert	α = .72–.77
Child Depression Inventory	Kovacs (1983)	Total score	3-point Likert	α = .83–.87
Videotaped dyadic interactions	Hetherington & Clingempeel (1992)	Depressed mood	Global coding, 5-point Likert	κ = .58 IC = .75

[a] For all measures, raters included the mother, father, and adolescent except videotaped interactions, which were rated by an observer. [b] Reliabilities are Cronbach's alpha (α) for all scales except videotaped dyadic interactions. For the videotaped dyadic interactions, reliabilities are weighted kappa (κ) and intraclass correlations (IC).

also the same genetic and environmental factors that influence the adolescent's maladjustment 3 years later?

The models illustrated in Figures 2 and 3 represent only one member of the sibling pair. If both siblings were illustrated in the model, then all of the genetic factors would be connected by double-headed arrows with values corresponding to the degree of genetic similarity (1.0 for MZ; .5 for DZ, FI, and FS; .25 for HS; and 0 for US). The shared environmental factors are set to be the same for both members of the twin and sibling pairs because they reside in the same household, and the nonshared environmental factors are, by definition, uncorrelated for the two siblings.

Figures 2 and 3 contain 12 latent factors. The latent factors G1, Es1, and En1 represent genetic, shared, and nonshared environmental contributions to stability in parental conflict–negativity and in the contemporaneous association between parenting and adolescent adjustment at Time 1 (Paths p and c1 in Figure 1). Genetic, shared, and nonshared environmental influences on stability in adolescent adjustment and the contemporaneous associations between parent conflict–negativity and adolescent adjustment at Time 2 (Paths m and c2 in Figure 1) are represented by the latent factors G2, Es2, and En2. The focus of the present article is the genetic, shared environmental, and nonshared environmental contributions to the cross-lagged associations. In Figure 2, the cross-lagged association between parental conflict–negativity at Time 1 and adolescent adjustment at Time 2 (Path l1 in Figure 1) is represented by latent factors G3, Es3, and En3. Finally, in Figure 2, any genetic and environmental influences on adolescent adjustment at Time 2 that are independent of stability and parenting are represented by the latent factors g4, es4, and en4.

Figure 3 represents the alternate model, the cross-lagged association of adolescent adjustment at Time 1 and parental conflict–negativity at Time 2. The first two sets of latent factors are the same; however, the latent factors G3, Es3, and En3 now represent Path l2 from Figure 1, and the residual genetic and environmental influences (g4, es4, and en4) represent unique genetic and environmental influences on parental conflict–negativity at Time 2. It is important to note that the genetic cross-lagged models represented by Figures 2 and 3 are not nested. Therefore, it is impossible to establish which model best represents the data, as is typically done in phenotypic cross-lagged analyses. It is possible, however, to compare the two models to evaluate which model provides a more parsimonious fit (the fewest number of paths and the smallest chi-square and root mean square error of approximation [RMSEA]; see the explanation of fit indices later).

This genetic model is a derivation of Cholesky factoring or triangular decomposition (Neale & Cardon, 1992). This approach is used most often for longitudinal analysis of a single measure or for a multivariate analysis of simultaneously measured variables that have been ordered according to a hypothesis (Loehlin, 1996). The cross-lagged genetic model combines these two functions. The variables are ordered so that common variance that is due to stability and contemporaneous associations are decomposed into genetic and environmental components before genetic and environmental contributions to the cross-lagged association are estimated. Because there was no hypothesis for weighting stability differently from the contemporaneous associations, these associations were considered simultaneously.

The significance of each genetic and environmental component was tested by using a nested model design in which each set of paths was dropped from the model and the change in chi-square was computed. For example, to test the significance of the G3 parameter in Figure 2, we set Paths h31 and h34 to zero and computed the change in chi-square with two degrees of freedom. The overall fit of the model was tested by chi-square and the RMSEA (Steiger, 1990). A nonsignificant chi-square and an RMSEA that is low (.05 or below) indicate that the model accurately represents the data. Previous research has found that chi-square is likely to reject a model that fits the data well, but imperfectly, is very sensitive to sample size, and improves when more parameters are added to the model (Mulaik et al., 1989; Neale & Cardon, 1992; Tanaka, 1993). Because RMSEA also incorporates parsimony into its assessment of fit, it is often used as an alternative fit index (Browne & Cudeck, 1993). Further discussion of fit indices is available elsewhere (Bollen & Long, 1993; Loehlin, 1992; Neale & Cardon, 1992; Plomin et al., 1990).

The assumptions of the model are reflected in the path diagram (Figure 2): there is no assortative mating and no selective placement of stepsiblings, shared and nonshared environmental effects are the same across sibling types, genetic effects are additive, and gene–environment interaction is negligible. A more detailed discussion of these assumptions for the NEAD sample can be found in Pike et al. (1996). Additional, more general, discussion of the assumptions of quantitative genetic model fitting is available elsewhere (Loehlin, 1992; Neale & Cardon, 1992; Plomin, DeFries, McClearn, & Rutter, 1997). Variance–covariance matrices, computed separately for each of the six sibling types, were used for the model-fitting analyses (Cudeck, 1989; Neale & Cardon, 1992).

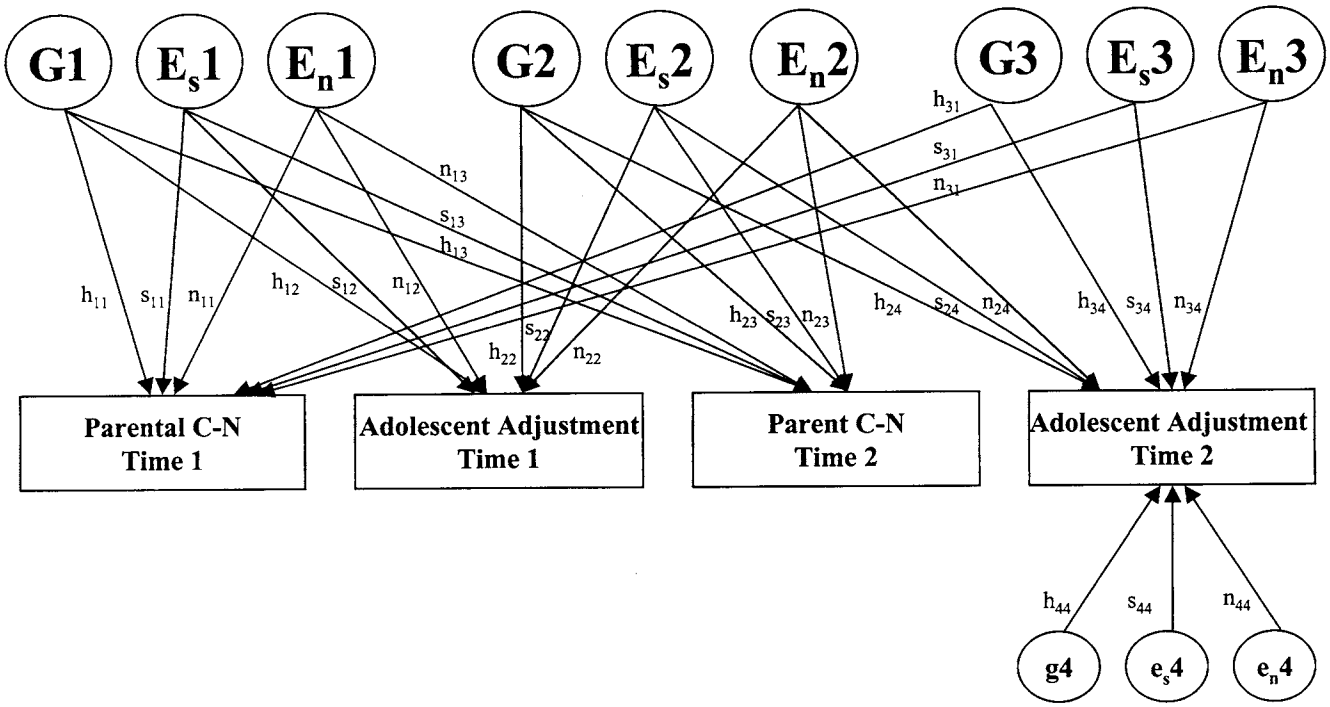

Figure 2. Cross-lagged genetic model. Latent factors are represented by circles and phenotypic measures are represented by rectangles. Genetic, shared environmental, and nonshared environmental contributions to stability in parental conflict–negativity (C–N) and to the association between parental C–N and adolescent adjustment at Time 1 are represented by the latent factors G1, E_s1, and E_n1, respectively. Latent factors G2, E_s2, and E_n2 represent genetic, shared environmental, and nonshared environmental contributions to stability in adolescent adjustment and to the contemporaneous association between parental C–N and adjustment at Time 2. Genetic, shared, and nonshared environmental contributions to the cross-lagged association between parental C–N at Time 1 and adolescent adjustment at Time 2 are represented by the latent factors G3, E_s3, and E_n3. The latent factors g4, e_s4, and e_n4 represent genetic, shared, and nonshared environmental influences that are unique to adolescent adjustment at Time 2.

Results

The composite scores were corrected for age, sex, and their interaction by using standardized partial residuals from the regression of scores on these variables (McGue & Bouchard, 1984). Nontwin sibling scores were also corrected for age differences within sibling pairs to control for any decrease in the nontwin sibling correlations that was due to age differences between the siblings. None of these demographic variables accounted for more than 9% of the variance for any of the measures of parenting or adolescent adjustment.

Correlations

The phenotypic cross-lagged correlations were significant for all of the associations between parental conflict–negativity and adolescent maladjustment, although the magnitude of the correlations was lower for depressive symptoms than for antisocial behavior. Maternal and paternal conflict–negativity at Time 1 correlated .48 and .38, respectively, with antisocial behavior at Time 2 and .24 and .15 with depressive symptoms at Time 2. The correlations were very similar for the opposite direction relationship. Antisocial behavior at Time 1 correlated .48 and .46, respectively, with maternal and paternal conflict–negativity at Time 2, and depressive symptoms at Time 1 correlated .24 and .22 with maternal and paternal conflict–negativity at Time 2. These correlations were not corrected for stability or for the contemporaneous associations; however, they are a reasonable starting point for subsequent analyses. Concurrent correlations between parental conflict–negativity and adolescent antisocial and depressive symptoms have been reported elsewhere (Pike et al., 1996) and are available from Jenae M. Neiderhiser by request (*r*s range from .24 to .63). Finally, there was substantial stability in both parental conflict–negativity (r = .64 for maternal and r = .50 for paternal) and adolescent adjustment (r = .63 for antisocial behavior and r = .66 for depressive symptoms).

Model Fitting

The cross-lagged genetic model was applied to the associations between parental conflict–negativity and adolescent adjustment (see Figures 2 and 3). Standardized parameter estimates and fit indices are presented in Tables 3 and 4; for clarity of presentation, the parameter estimates are presented in a three-part format. The standardized parameter estimates for cross-lagged associations are

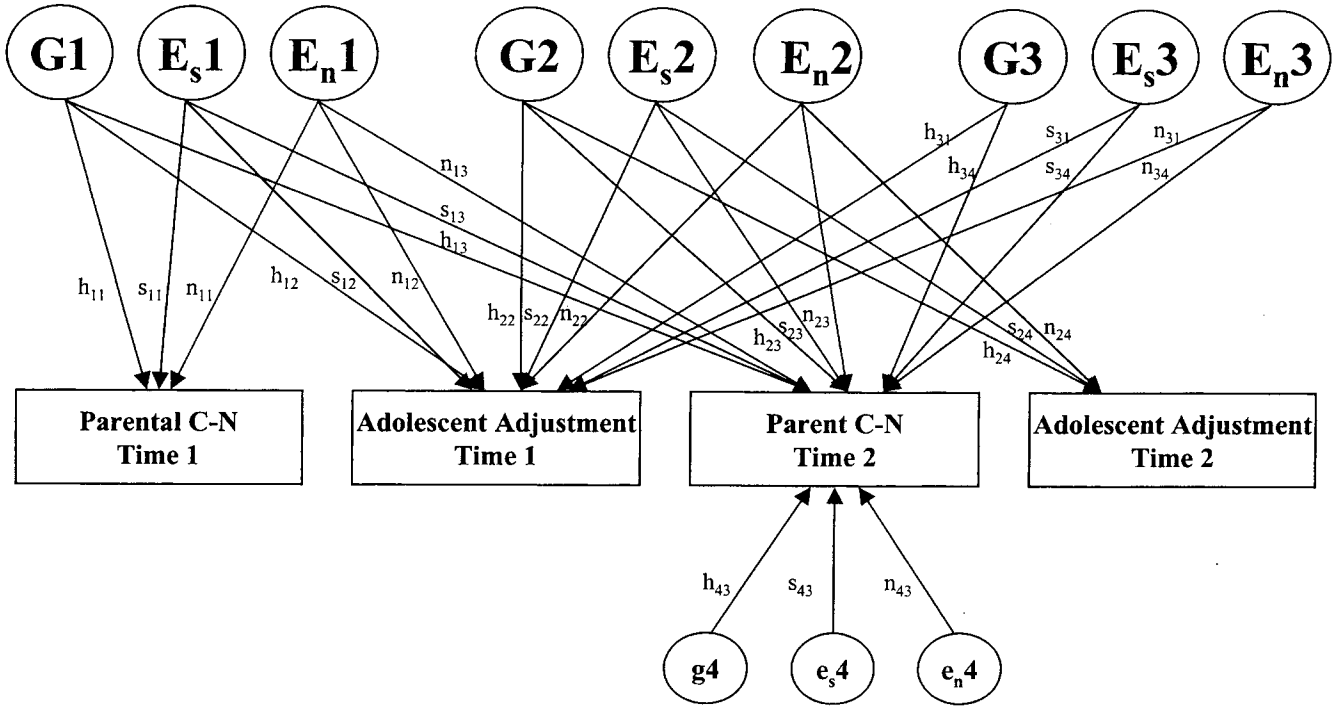

Figure 3. Alternate cross-lagged genetic model. Genetic, shared environmental, and nonshared environmental contributions to stability in parental conflict–negativity (C–N) and to the association between parental C–N and adolescent adjustment at Time 1 are represented by the latent factors G1, E_s1, and E_n1, respectively. Latent factors G2, E_s2, and E_n2 represent genetic, shared environmental, and nonshared environmental contributions to stability in adolescent adjustment and to the contemporaneous association between parental C–N and adjustment at Time 2. Genetic, shared, and nonshared environmental contributions to the cross-lagged association between adolescent adjustment at Time 1 and parental C–N at Time 2 are represented by the latent factors G3, E_s3, and E_n3. The latent factors g4, e_s4, and e_n4 represent genetic, shared, and nonshared environmental influences that are unique to parental C–N at Time 2.

presented first, in the top part of Tables 3 and 4. The parameter estimates for stability and the contemporaneous associations are located in the second and third sections, and the residuals are located in the fourth section of Tables 3 and 4. Finally, the last part of Tables 3 and 4 contains the chi-square and RMSEA values. For all of the models, the chi-square values were significant; however, all of the RMSEA values were .05 or below. Taken together, these two fit indices indicate that the fit of the models was acceptable. One exception is the RMSEA for the alternate model examining the association between depressive symptoms at Time 1 and maternal conflict–negativity at Time 2, which exceeds the .05 cutoff for acceptable fit.

The percentages of genetic, shared environmental and nonshared environmental contributions to the model-estimated phenotypic associations were also computed (Tables 5 and 6). These values were derived from the parameter estimates reported in Tables 3 and 4. For example, the percentage of the genetic contribution to the cross-lagged association between maternal conflict–negativity at Time 1 and adolescent antisocial behavior at Time 2 was computed as follows. First, the paths leading to each of the latent factors G3, Es3, and En3 were multiplied and summed (h31 × h34 + s31 × s34 + n31 × n34). This value represents the *estimated correlation* between maternal conflict–negativity and adolescent antisocial behavior after stability and the contemporaneous associations have been accounted for [(−.39 × −.45 + .00 × .00 + .36 × .02) = .17]. The percentage of the genetic contribution to this estimated phenotypic correlation was then computed by dividing the product of the paths leading to the G3 factor (−3.9 × −.45 = .18) by the r_{est} (.18/.17 = 100).

Maternal conflict–negativity and adolescent adjustment. For maternal conflict–negativity and adolescent antisocial behavior, the cross-lagged association was influenced primarily by genetic factors. All of the estimated phenotypic correlation can be explained by genetic factors (Table 5). Genetic factors were also important for the stability of both measures (79% of conflict–negativity and 73% of antisocial behavior) and for the contemporaneous association between maternal conflict–negativity and antisocial behavior at Time 1 (93%). Shared and nonshared environmental influences had their greatest effect on the contemporaneous associations between maternal conflict–negativity and antisocial behavior at Time 2, accounting for 35% and 63% of the correlation, respectively. The only unique influences on antisocial behavior at Time 2 were nonshared environmental factors (see Table 3).

For maternal conflict–negativity and adolescent depressive symptoms, genetic influences on the cross-lagged association were

Table 3
Standardized Maximum-Likelihood Parameter Estimates and Fit Indices From the Longitudinal Cross-Lagged Genetic Model

Parameter	Maternal conflict–negativity		Paternal conflict–negativity	
	Antisocial behavior	Depressive symptoms	Antisocial behavior	Depressive symptoms
	Cross-lagged results			
h_{31}	−.39[a]	−.27	−.39[a]	−.20[a]
h_{34}	−.45	−.27	−.43	−.23
s_{31}	.00	.00	.00	.15
s_{34}	.00	.00	.00	.13
n_{31}	.36	.42	.38	.43
n_{34}	−.02	.06	.02	.06
	Longitudinal conflict–negativity and contemporaneous at Time 1			
h_{11}	−.76[b]	−.76[b]	−.65[b]	.70[b]
h_{12}	−.50	−.29	−.41	.19
h_{13}	−.55	−.69	−.53	.67
s_{11}	.32	.41	−.48[c]	−.50[c]
s_{12}	.00	.00	−.15	−.12
s_{13}	.33	.00	−.33	−.27
n_{11}	.20[d]	−.09[d]	−.20[d]	−.14[d]
n_{12}	.44	−.48	−.52	−.56
n_{13}	−.11	.40	.04	.22
	Longitudinal adjustment and contemporaneous at Time 2			
h_{22}	−.72[e]	.75[e]	.68[e]	.74[e]
h_{23}	−.02	.07	.37	.00
h_{24}	−.50	.60	.77	.62
s_{22}	.18	.00	.29	−.12
s_{23}	.38	.37	−.34	.37
s_{24}	.44	.28	.00	.00
n_{22}	−.12[f]	.35[f]	.02	.27[f]
n_{23}	−.66	.40	.02	.54
n_{24}	−.45	.33	−.22	.41
	Unique to adjustment at Time 2			
h_{44}	.00	.00	.00	.00
s_{44}	.00	.00	.00	.00
n_{44}	.38[g]	.62[g]	.42[g]	.61[g]
	Goodness-of-fit indices			
$\chi^2(189, N = 395)$	390.61*	314.08*	364.33*	338.93*
RMSEA	.05	.04	.05	.05

Note. See Figure 2 for an explanation of the labels for the parameter estimates (e.g., h_{11}). RMSEA = root mean square error of approximation.
[a] Significant G3 factor. [b] Significant G1 factor. [c] Significant E_s1 factor. [d] Significant E_n1 factor. [e] Significant G2 factor. [f] Significant E_n2 factor. [g] Significant e_n4 factor.
* $p \leq .05$.

not significant. Nonetheless, the parameter estimates for genetic influences (70%) were larger than those for either the shared (0%) or the nonshared (30%) environmental factors (Table 3). Similar to the results for antisocial behavior, genetic factors were the largest contributor for most of the remaining associations. The one exception was the contemporaneous association between maternal conflict–negativity and depressive symptoms at Time 2 that could be explained primarily by shared (37%) and nonshared (48%) environmental factors. Finally, only nonshared environment remained unique to depression at Time 2.

Paternal conflict–negativity and adolescent adjustment. As expected from the high correlation between maternal and paternal conflict–negativity ($r = .63$ at Time 1 and $r = .59$ at Time 2), the results for father were nearly identical to the results for mother. Genetic factors contributed the most to the cross-lagged associations between paternal conflict–negativity and to both measures of adolescent adjustment (94% for antisocial behavior and 50% for depressive symptoms). There were some differences in the significance of factors; nonetheless, the relative contributions of genetic and environmental factors to each association between conflict–

Table 4
Standardized Maximum-Likelihood Parameter Estimates and Fit Indices From the Alternate Longitudinal Cross-Lagged Genetic Model

Parameter	Maternal conflict–negativity		Paternal conflict–negativity	
	Antisocial behavior	Depressive symptoms	Antisocial behavior	Depressive symptoms
Cross-lagged results				
h_{31}	.50[a]	.64[a]	.25	.69[a]
h_{34}	.08	−.18	−.21	.18
s_{31}	−.30[b]	.00	.27	.00[b]
s_{34}	.00	.00	−.10	.47
n_{31}	.42	.41	.45	.41
n_{34}	−.04	.08	.01	.03
Longitudinal conflict–negativity and contemporaneous at Time 1				
h_{11}	−.84[c]	−.81[c]	−.73[c]	.82[c]
h_{12}	−.24	−.47	−.49	.44
h_{13}	−.48	−.35	−.40	.54
s_{11}	.31	−.36	−.53[d]	−.28
s_{12}	.27	−.32	.05	.00
s_{13}	.00	.01	−.25	.00
n_{11}	.46	.46[e]	−.53[e]	.50[e]
n_{12}	−.04	.14	.05	.09
n_{13}	.01	.02	−.25	.23
Longitudinal adjustment and contemporaneous at Time 2				
h_{22}	.53[f]	.21[f]	.45[f]	.25[f]
h_{23}	.00	.40	.36	.00
h_{24}	.71	.78	.70	.77
s_{22}	−.14[g]	−.12	.12[g]	.26[g]
s_{23}	.00	−.26	.38	.00
s_{24}	−.54	−.14	.55	.17
n_{22}	.22[h]	.14[h]	.23[h]	.14[h]
n_{23}	.10	.31	.02	.08
n_{24}	.45	.61	.45	.61
Unique to parental conflict–negativity at Time 2				
h_{44}	.48[i]	−.22	.15	.39[i]
s_{44}	.00	.00	−.36[j]	.00
n_{44}	.72[k]	.68[k]	.55[k]	.49[k]
Goodness-of-fit indices				
$\chi^2(189, N = 395)$	387.42*	443.71*	341.52*	323.49*
RMSEA	.05	.06	.05	.04

Note. See Figure 3 for an explanation of the labels for the parameter estimates (e.g., h_{11}). RMSEA = root mean square error of approximation.
[a] Significant G3 factor. [b] Significant E_S3 factor. [c] Significant G1 factor. [d] Significant E_S1 factor. [e] Significant E_n1 factor. [f] Significant G2 factor. [g] Significant E_S2 factor. [h] Significant E_n2 factor. [i] Significant g4 factor. [j] Significant E_S4 factor. [k] Significant e_n4 factor.
* $p < .05$.

negativity and adolescent adjustment were nearly identical for mothers and fathers. One interesting difference between maternal and paternal conflict–negativity was that there is evidence that shared environmental factors were more important for associations involving paternal conflict–negativity than for maternal. For example, shared environmental factors were significant for none of the maternal conflict–negativity associations; whereas for paternal conflict–negativity, shared environmental factors were significant for the Time 1 contemporaneous associations with both antisocial behavior and depressive symptoms.

Alternate model. There were some differences in the results from the model that examined the cross-lagged association of adolescent adjustment at Time 1 to parental conflict–negativity at Time 2 (Path l2 in Figure 1). For the models examining the cross-lagged associations between adolescent antisocial behavior at Time 1 and maternal or paternal conflict–negativity, the estimated phenotypic correlation for the cross-lagged association was smaller, especially for maternal conflict–negativity. The opposite was true when depressive symptoms were examined; the estimated phenotypic correlations were somewhat larger. In addition, the

Table 5
Genetic and Environmental Contributions to the Phenotypic Associations Between Parental Conflict–Negativity and Adolescent Adjustment From the Longitudinal Cross-Lagged Model and Genetic and Environmental Correlations

Measure	Percentage contribution to estimated phenotypic correlation: G	E_S	E_n	r_{est}
Maternal conflict–negativity: Antisocial behavior				
Cross-lagged	100	0	0	.17
C–N stability	79	21	0	.51
ASB stability	73	16	10	.31
C–N/ASB @ Time 1	93	0	7	.41
C–N/ASB @ Time 2	2	35	63	.48
Maternal conflict–negativity: Depressive symptoms				
Cross-lagged	70	0	30	.10
C–N stability	100	0	0	.51
DEP stability	79	0	21	.57
C–N/DEP @ Time 1	85	0	15	.26
C–N/DEP @ Time 2	15	37	48	.27
Paternal conflict–negativity: Antisocial behavior				
Cross-lagged	94	0	6	.18
C–N stability	68	32	0	.49
ASB stability	100	0	0	.52
C–N/ASB @ Time 1	61	16	23	.44
C–N/ASB @ Time 2	100	0	0	.28
Paternal conflict–negativity: Depressive symptoms				
Cross-lagged	50	20	30	.10
C–N stability	77	23	0	.58
DEP stability	81	0	19	.57
C–N/DEP @ Time 1	48	22	30	.27
C–N/DEP @ Time 2	0	0	100	.22

Note. r_{est} = estimated phenotypic correlation between the parental conflict–negativity (C–N) variable and the adolescent antisocial behavior (ASB) variable; DEP = adolescent depressive symptoms.

patterns of genetic and environmental contributions to the cross-lagged associations were different in the alternate models. The most striking differences were for the cross-lagged associations between adolescent adjustment at Time 1 and paternal conflict–negativity at Time 2. Shared environmental influences were present for the cross-lagged association between antisocial behavior at Time 1 and paternal conflict–negativity at Time 2, whereas shared environmental contributions were zero for the association between depressive symptoms and paternal conflict–negativity. Conversely, shared environment was not present for paternal conflict–negativity at Time 1 and antisocial behavior at Time 2, as was the case when the opposite direction cross-lagged association was examined (see Table 5).

When the fit of the two models was compared to assess whether one provides a more parsimonious fit (see the bottom section of Tables 3 and 4), no clear conclusions can be drawn. Only for the two models that examined the association between maternal conflict–negativity and adolescent depressive symptoms was there a substantial difference between the original and alternate models' chi-square values and RMSEAs. In this case, the model that included a cross-lagged path from maternal conflict–negativity at Time 1 and depressive symptoms at Time 2 was more parsimonious, although the differences between the two indices of model fit were modest. The fit indices for the other models were all nearly identical, therefore, because these models are not nested and thus no conclusions can be drawn in regard to whether the original model or the alternate model is the best explanation of the data. For this reason, no clear judgments in regard to the direction of effects can be made.

Discussion

By examining cross-lagged associations between parent–child conflict and adolescent maladjustment for genetic and environmental contributions, we addressed the question of how parenting contributes to adolescent maladjustment. We had expected that the independent covariance between parental conflict–negativity at Time 1 and change in adolescent antisocial behavior and depres-

Table 6
Genetic and Environmental Contributions to the Phenotypic Associations Between Parental Conflict–Negativity and Adolescent Adjustment From the Alternate Longitudinal Cross-Lagged Model and Genetic and Environmental Correlations

Measure	Percentage contribution to estimated phenotypic correlation: G	E_S	E_n	r_{est}
Antisocial behavior: Maternal conflict–negativity				
Cross-lagged	67	0	33	.06
C–N stability	100	0	0	.40
ASB stability	68	14	18	.56
C–N/ASB @ Time 1	67	27	6	.30
C–N/ASB @ Time 2	0	0	100	.05
Depressive symptoms: Paternal conflict–negativity				
Cross-lagged	79	0	21	.14
C–N stability	97	0	37	.29
DEP stability	59	7	33	.27
C–N/DEP @ Time 1	68	21	11	.56
C–N/DEP @ Time 2	57	7	35	.54
Antisocial behavior: Paternal conflict–negativity				
Cross-lagged	38	23	38	.13
C–N stability	69	0	31	.42
ASB stability	65	14	20	.49
C–N/ASB @ Time 1	65	29	5	.55
C–N/ASB @ Time 2	53	45	2	.47
Depressive symptoms: Paternal conflict–negativity				
Cross-lagged	92	0	8	.13
C–N stability	80	0	20	.55
DEP stability	59	13	28	.32
C–N/DEP @ Time 1	88	0	12	.41
C–N/DEP @ Time 2	0	0	100	.05

Note. r_{est} = estimated phenotypic correlation between the parental conflict–negativity (C–N) variable and the adolescent antisocial behavior (ASB) variable; DEP = adolescent depressive symptoms.

sive symptoms would be explained by primarily environmental factors. In contrast to our environmental hypothesis, the results indicate that the cross-lagged associations were influenced primarily by genetic factors. These results suggest that most of the nonshared family factors found in phenotypic research with this sample (Reiss et al., 1995) were nonshared genetic factors rather than nonshared environmental. The findings and their implications are discussed in turn.

Two general patterns of results emerged, which were similar for both maternal and paternal conflict–negativity and differed somewhat according to adolescent adjustment. Cross-lagged associations between parental conflict–negativity and adolescent antisocial behavior could be attributed primarily to genetic factors, once genetic and environmental contributions to stability and to the contemporaneous associations were accounted for. The importance of genetic contributions to the cross-lagged associations between parental conflict–negativity and depressive symptoms was less than for antisocial behavior. Nonetheless, genetic factors contributed the most to the cross-lagged associations, although not significantly for maternal conflict–negativity and depressive symptoms. The decreased importance of genetic influences on depression was not surprising given that the phenotypic correlations between both maternal and paternal conflict–negativity and adolescent depressive symptoms were smaller than the correlations for antisocial behavior. Although genetic factors appear to play a role in the association between parental conflict–negativity and change in depressive symptoms, it is more difficult to decompose the phenotypic covariance into genetic and environmental components when only modest levels of covariance exist.

As noted, our analytic models were developed to explore how interindividual differences in parenting are associated with changes in adolescent adjustment. Whether intraindividual changes in parenting and adolescent's adjustment were associated was not tested. We focused on one variant of this approach: a model that examines the association of parenting at Time 1 with change in adolescent maladjustment from early-to-middle adolescence. We found that there was substantial change in genetic influence across this 3-year time period, and when the effects of these genetic changes were partialled out there was little or no additional influence on change contributed by Time 1 parenting.

There are three possible explanations for this finding. The first relies on the fact that adolescents and parents share 50% of their genes. Thus, genetic factors responsible for change in adolescent maladjustment may overlap with genetic factors associated with irritability and negative child rearing in their parents. In other words, this could simply be a passive genotype–environment correlation (see Scarr, 1992, or Plomin, 1994, for a more detailed explanation of genotype–environment correlation). Second, characteristics of the child that lead to or are part of change in maladjustment and symptomatology in adolescents may elicit negative behavior from parents. This second explanation, if true, would suggest evocative genotype–environment correlation. In other words, there is something about the way the child behaves that causes the parents to respond in a certain manner that is then associated with adolescent adjustment 3 years later for genetic reasons. When considering this evocative explanation, it is important to consider that, whatever these genetically influenced characteristics of the child are that elicit negative parenting, they are *independent* of the measured adjustment constructs included in the models (antisocial behavior and depressive symptoms). In other words, the parents are responding to some genetically influenced characteristic in the child that is not measured in our model but that is associated with the adolescent's adjustment 3 years later. Possible examples could include attributes like temperament or academic performance.

The third explanation does not concern genotype–environment correlation. It is possible that negative parenting at Time 1 may arouse and frighten the adolescent, which could then elicit genetic change in adolescents whose genomes include genes that influence antisocial behavior or depressive symptoms. Until recently, an explanation of this kind would appear improbable. However, several recent studies suggest that exceedingly stressful environmental circumstances may elicit molecular mechanisms of genetic expression in rodents (Larsen & Mau, 1994; Nilsson et al., 1993; Smith, Banerjee, Gold, & Glowa, 1992; Watanabe, Stone, & McEwen, 1994) and even in humans (Glaser et al., 1990; Platt, He, Tang, Slater, & Goldstein, 1993; Post, Weiss, & Leverich, 1994), although none of these studies have focused on antisocial behavior or depressive symptoms or on individual differences in adaptive behavior.

An advantage of model fitting is the ability to test alternate models. In this case, there was only modest power to test an alternate cross-lagged genetic model (Path l2 in Figure 1) because the models are not nested; however, the two models were compared to evaluate which model provides a more parsimonious fit. In this case, the alternate model provided similar parameter estimates and fit indices as did the original model, thus providing further support for the bidirectionality of these associations. Unfortunately, this conclusion leaves an issue proposed by an earlier article from this project (Reiss et al., 1995) unresolved: What is the direction of effects in the association between parental behavior and adolescent adjustment? There was, however, one exception to these inconclusive findings in regard to the direction of effects: Maternal conflict–negativity at Time 1 and change in depressive symptoms (the original model) was more parsimonious than the alternative. This one finding, although modest, provides support for the directional hypothesis that has guided much of the literature in this area: Maternal conflict–negativity is associated with change in adolescent depressive symptoms (high conflict is correlated with increased depression). Nonetheless, these results are surprising because the cross-lagged association was due almost entirely to overlapping genetic influences, as described earlier.

There were, however, some differences in the patterns of findings for the two cross-lagged models. Genetic contributions to the cross-lagged association between antisocial behavior at Time 1 and parenting at Time 2 were substantially lower than for the cross-lagged associations in the opposite direction. In fact, for paternal behavior, genetic influences were not significant for this cross-lagged association. In part, these differences may be explained by the more modest estimated phenotypic associations once the stability and contemporaneous associations had been accounted for (see Tables 5 and 6). The pattern of findings for depressive symptoms was also somewhat different when the alternate model was examined; however, these differences were less pronounced than those for antisocial behavior. Despite these modest differences between the two cross-lagged models, the overall findings were much the same. In most cases, genetic influences explained the majority of the cross-lagged association between

parenting and adolescent adjustment, regardless of the direction of effects.

Genetic and environmental contributions to the contemporaneous associations between parental conflict–negativity and adolescent adjustment differed at Time 1 and Time 2. For example, at Time 1, the association between maternal conflict–negativity and antisocial behavior can be explained almost exclusively by overlapping genetic influences, whereas at Time 2 this same association was influenced very little by genetic factors. These findings, although surprising, are consistent with the findings from NEAD, which examined a substantial portion of the parenting and adolescent adjustment constructs available for this sample (Reiss, 1997; Reiss, Neiderhiser, Hetherington, & Plomin, in press). When five parenting composites were associated with seven adolescent adjustment composites in a series of bivariate analyses, different patterns of findings emerged for Time 1 and Time 2. These findings suggest that the contributions of genetic and environmental factors change over time. When models like the present one are used, evidence of this change is provided.

One finding that emerged for both of the genetic cross-lagged models was unexpected and warrants further discussion. Shared environment was somewhat more important for father's parenting than for mother's parenting. This finding could be the result of a tendency of the father to treat the adolescents more similarly; in other words, a father tends to be less sensitive to individual differences than a mother. Other studies have found similar results for associations between composite and self-report measures of father's versus mother's parenting and adolescent adjustment (Pike, 1994; Pike et al., 1996), which may suggest that this pattern of findings is robust and should be examined more carefully in future studies. When genetic and environmental contributions to the correlations between mother's and father's parenting have been examined, there is evidence that genetic contributions to this overlap are modest for most parenting constructs (Reiss et al., in press), a finding that would support different patterns of genetic and environmental contributions for associations between mother's and father's parenting with adolescent adjustment. There is a recent study, however, that does not support this pattern of results, at least for sons. Elkins, McGue, and Iacono (1997) found that when the correlation between mother's and father's parenting to their twin sons was examined for genetic and environmental contributions, all of the shared environment influences on father's parenting were correlated with the shared environmental influences on mother's parenting. Additional exploration of differences in the patterns of findings for mothers and fathers will help to resolve these somewhat different findings.

The measures used in this study were composites across several raters, providing a less rater-biased and more global estimation of family process and adolescent adjustment. Some would argue, however, that the perceptions of the individuals are also important gauges of family interaction and adjustment (Hinde, 1979; Kreppner & Lerner, 1989; Powers, Welsh, & Wright, 1994). One future direction for research in this area is to break apart the composites of parenting and adolescent adjustment to investigate differences between raters. When the measures of parenting and adolescent adjustment used in the present study were examined separately by respondent at Time 1, substantial differences in the patterns of genetic and environmental contributions to these associations emerged (Pike, 1994), and adolescent's perceptions of their parent's behavior significantly mediated the genetic influences on the association between parenting and adolescent adjustment (Neiderhiser, Pike, Hetherington, & Reiss, 1998). It may be that genetic contributions to the cross-lagged associations will decrease and the environmental contributions will increase as a function of whose ratings are examined.

It is worth mentioning that this study took a very conservative approach to the causal modeling of the cross-lagged associations. Many recent articles examining cross-lagged associations have used models that control only for the stability of one measure and the Time 1 contemporaneous associations (Conger & Conger, 1994; Cramer, 1988). The fact that we found evidence of any cross-lagged associations by using this conservative approach is noteworthy, especially because they were explained primarily by genetic influences.

In summary, the results of this study indicate that genetic factors contribute importantly to the longitudinal associations between parenting and adolescent adjustment assessed 3 years later, especially for antisocial behavior. The importance of these findings for developmental researchers is twofold. First, these findings stress the need to consider child factors when examining the impact of parenting on adolescent adjustment. The importance of child effects on parents was first introduced by Bell (1968, 1979) and has since become more accepted by developmentalists. It is commonly thought, however, that these child effects can be controlled for if the child's behavior is included in, or removed from, a model of parenting to later child adjustment. In the current article, genetically influenced adolescent factors were found to have a substantial impact on the association between parenting and adolescent adjustment. This finding suggests that simply using an "environmental" variable to predict later child adjustment, corrected for earlier adjustment, does not rule out the possibility of a child effect. The child effect, in this case, would be different than is usually conceptualized, in that genetically influenced characteristics of the child may have elicited certain styles of parenting behavior, which are associated with the child's later behavior independent of stability. One example of how this could operate longitudinally is through child temperament. If the child is very irritable and difficult to soothe as an infant, the parents may withdraw from the child and adopt a more negative style of parenting very early in the child's life. This negative style of parenting then continues as the child matures into an irritable toddler, and coercive patterns of parent–child interactions may be set into place. By the time the child has reached adolescence, the coercive styles of parent–child interaction may be well established, although they began, in this example, as a parental response to a genetically influenced characteristic of the child (temperament). The second implication for developmentalists is that genetic factors do not influence only those measures that are stable. There have been several recent articles that have stressed the changing nature of genetic influences throughout the life span (e.g., Matheny, 1989; Neiderhiser, Reiss, & Hetherington, 1996; O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998; Plomin et al., 1993; Van den Oord & Rowe, 1997). The present article is consistent with these findings of genetic influences on change as well as stability and takes one step further in finding that these genetic influences on change in adolescent adjustment are associated with parenting for mostly genetic reasons.

References

Baumrind, D. (1991). The influence of parenting styles on adolescent competence and substance use. *Journal of Early Adolescence, 11,* 56–95.

Bell, R. Q. (1968). A reinterpretation of the direction of effects in socialization. *Psychological Review, 75,* 107–116.

Bell, R. Q. (1979). Parent, child, and reciprocal influences. *American Psychologist, 34,* 821–826.

Bollen, K. A., & Long, J. S. (Eds.). (1993). *Testing structural equation models.* Newbury Park, CA: Sage.

Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.

Conger, K. J., & Conger, R. D. (1994). Differential parenting and change in sibling differences in delinquency. *Journal of Family Psychology, 8,* 287–302.

Cramer, D. (1988). Self-esteem and facilitative close relationships: A cross-lagged panel correlation analysis. *British Journal of Social Psychology, 27,* 115–126.

Cudeck, R. (1989). Analysis of correlation matrices using covariance structure models. *Psychological Bulletin, 105,* 317–327.

Elkins, I. J., McGue, M., & Iacono, W. G. (1997). Genetic and environmental influences on parent–son relationships: Evidence for increasing genetic influence during adolescence. *Developmental Psychology, 33,* 351–353.

Glaser, R., Kennedy, S., Lafuse, W. P., Bonneau, R. H., Speicher, C., Hillhouse, J., & Kiecolt-Glaser, J. K. (1990). Psychological stress-induced modulation of Interleukin 2 Receptor gene expression and Interleukin 2 production in peripheral blood leukocytes. *Archives of General Psychiatry, 47,* 707–712.

Haapasalo, J., & Tremblay, R. E. (1994). Physically aggressive boys from ages 6 to 12: Family background, parenting behavior, and prediction of delinquency. *Journal of Consulting and Clinical Psychology, 62,* 1044–1052.

Hetherington, E. M., & Clingempeel, W. G. (1992). Coping with marital transitions: A family systems perspective. *Monographs of the Society for Research in Child Development, 57*(2–3, Serial No. 227).

Hinde, R. (1979). *Toward understanding relationships.* San Diego, CA: Academic Press.

Kenny, D. A. (1979). *Correlation and causality.* New York: Wiley.

Kovacs, M. (1983). *Children's Depression Inventory: A self-rated depression scale for school-aged youngsters.* Unpublished manuscript, University of Pittsburgh School of Medicine.

Kreppner, K., & Lerner, R. M. (Eds.). (1989). *Family systems and life-span development* (pp. 149–163). Hillsdale, NJ: Erlbaum.

Larsen, P. J., & Mau, S. E. (1994). Effect of acute stress on the expression of hypothalamic messenger ribonucleic acids encoding the endogenous opioid precursors Preproenkephalin A and Proopiomelanocortin. *Peptides, 15,* 783–790.

Loeber, R., & Dishion, T. (1983). Early predictors of male delinquency: A review. *Psychological Bulletin, 94,* 68–99.

Loeber, R., & Stouthamer-Loeber, M. (1986). Family factors as correlates and predictors of juvenile conduct problems and delinquency. In N. Morris & M. Tonry (Eds.), *Crime and justice: An annual review of research* (Vol. 7, pp. 29–149). Chicago: University of Chicago Press.

Loehlin, J. C. (1992). *Latent variable models: An introduction to factor, path, and structural analysis* (2nd ed.). Hillsdale, NJ: Erlbaum.

Loehlin, J. C. (1996). The Cholesky approach: A cautionary note. *Behavior Genetics, 26*(1), 65–69.

Matheny, A. P., Jr. (1989). A longitudinal twin study of stability of components from Bayley's Infant Behavior Record. *Child Development, 54,* 356–360.

McGue, M., & Bouchard, T. J., Jr. (1984). Adjustment of twin data for the effects of age and sex. *Behavior Genetics, 14,* 325–343.

Mulaik, S. A., James, L. R., Alstine, J. V., Bennett, N., Lind, S., & Stilwell, C. D. (1989). Evaluation of goodness-of-fit indices for structural equation models. *Psychological Bulletin, 105,* 430–445.

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

Neiderhiser, J. M., Pike, A. E., Hetherington, E. M., & Reiss, D. (1998). Adolescent perceptions as mediators of parenting: Genetic and environmental contributions. *Developmental Psychology, 34,* 1459–1469.

Neiderhiser, J. M., Reiss, D., & Hetherington, E. M. (1996). Genetically informative designs for distinguishing developmental pathways during adolescence: Responsible and antisocial behavior. *Development and Psychopathology, 8,* 779–791.

Nichols, R. C., & Bilbro, W. C., Jr. (1966). The diagnosis of twin zygosity. *Acta Genetica, 16,* 265–275.

Nilsson, L., Mohammed, A. K. H., Henriksson, G. G., Folkesson, R., Winblad, B., & Bergstrom, L. (1993). Environmental influence on somatostatin levels and gene expression in the rat brain. *Brain Research, 628,* 993–998.

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, 39,* 323–336.

Patterson, G. R. (1982). *Coercive family process.* Eugene, OR: Castalia.

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist, 44,* 329–335.

Pike, A. (1994). *The nonshared experience of parental negativity and its relation to adolescent depression and antisocial behavior.* Unpublished master's thesis, Pennsylvania State University.

Pike, A., McGuire, S., Hetherington, E. M., Reiss, D., & Plomin, R. (1996). Family environment and adolescent depression and antisocial behavior: A multivariate genetic analysis. *Developmental Psychology, 32,* 590–603.

Platt, J. E., He, X., Tang, D., Slater, J., & Goldstein, M. (1993). C-FOS expression in vivo in human lymphocytes in response to stress. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 19,* 65–74.

Plomin, R. (1994). *Genetics and experience: The interplay between nature and nurture* (Vol. 6). Thousand Oaks, CA: Sage.

Plomin, R., DeFries, J. C., & McClearn, G. E. (1990). *Behavioral genetics: A primer* (2nd ed.). New York: Freeman.

Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (1997). *Behavioral genetics* (3rd ed.). San Francisco: Freeman.

Plomin, R., Emde, R., Braungart, J. M., Campos, J., Corley, R., Fulker, D. W., Kagan, J., Reznick, S., Robinson, J., Zahn-Waxler, C., & DeFries, J. C. (1993). Genetic change and continuity from 14 to 20 months: The MacArthur Longitudinal Twin Study. *Child Development, 64,* 1354–1376.

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.

Post, R. M., Weiss, S. R. B., & Leverich, G. S. (1994). Recurrent affective disorder: Roots in developmental neurobiology and illness progression based on changes in gene expression. *Development and Psychopathology, 6,* 781–813.

Powers, S. I., Welsh, D. P., & Wright, V. (1994). Adolescents' affective experience of family behaviors: The role of subjective understanding. *Journal of Research on Adolescence, 4,* 585–600.

Reiss, D. (1995). Genetic influence on family systems: Implications for development. *Journal of Marriage and the Family, 57,* 543–560.

Reiss, D. (1997). Mechanisms linking genetic and social influences in adolescent development: Beginning a collaborative search. *Current Directions in Psychological Science, 6,* 100–105.

Reiss, D., Hetherington, E. M., Plomin, R., Howe, G. W., Simmens, S. J., Henderson, S. H., O'Connor, T. J., Bussell, D. A., Anderson, E. R., &

Law, T. (1995). Genetic questions for environmental studies: Differential parenting of siblings and its association with depressive symptoms and antisocial behavior in adolescents. *Archives of General Psychiatry, 52,* 925–936.

Reiss, D., Neiderhiser, J. M., Hetherington, E. M., & Plomin, R. (in press). *The relationship code: Deciphering genetic and social patterns in adolescent development.* Cambridge, MA: Harvard University Press.

Reiss, D., Plomin, R., Hetherington, E. M., Howe, G. W., Rovine, M., Tryon, A., & Hagan, M. S. (1994). The separate worlds of teenage siblings: An introduction to the study of the Nonshared Environment in Adolescent Development. In E. M. Hetherington, D. Reiss, & R. Plomin (Eds.), *Separate social worlds of siblings: The impact of nonshared environment on development* (pp. 63–109). Hillsdale, NJ: Erlbaum.

Rubin, K. H., Hymel, S., Mills, R. S. L., & Rose-Krasnor, L. (1991). Conceptualizing different developmental pathways to and from social isolation in childhood. In D. Cicchetti & S. L. Toth (Eds.), *Rochester Symposium on Developmental Psychopathology* (Vol. 2, pp. 91–122). Hillsdale, NJ: Erlbaum.

Scarr, S. (1992). Developmental theories for the 1990's: Development and individual differences. *Child Development, 63,* 1–19.

Smith, M. A., Banerjee, S., Gold, P. W., & Glowa, J. (1992). Induction of c-fos mRNA in rat brain by conditioned and unconditioned stressors. *Brain Research, 578,* 135–141.

Spitz, E., Moutier, R., Reed, T., Busnel, M. C., Marchaland, C., Roubertoux, P. L., & Carlier, M. (1996). Comparative diagnoses of twin zygosity by SSLP variant analysis, questionnaire, and dermatoglyphic analysis. *Behavior Genetics, 26*(1), 55–63.

Stattin, H., & Klackenberg-Larsson, I. (1990). The relationship between maternal attributes in the early life of the child and the child's future criminal behavior. *Development and Psychopathology, 2,* 99–111.

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research, 25,* 173–180.

Strauss, M. A. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics (CT) Scales. *Journal of Marriage and the Family, 41,* 75–85.

Tanaka, J. S. (1993). Multifaceted conceptions of fit in structural equation models. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 10–39). Newbury Park, CA: Sage.

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

Vuchinich, S., Bank, L., & Patterson, G. R. (1992). Parenting, peers, and the stability of antisocial behavior in preadolescent boys. *Developmental Psychology, 28,* 510–521.

Watanabe, Y., Stone, E., & McEwen, B. S. (1994). Induction and habituation of c-fos and zif/268 by acute and repeated stressors. *NeuroReport, 5,* 1321–1324.

Zill, N. (1985). *Behavior problems scales developed from the 1981 Child Health Supplement to the National Health Interview Survey.* Washington, DC: Child Trends.

Received July 18, 1997
Revision received March 13, 1998
Accepted August 12, 1998 ■