Developmental Psychology
1996, Vol. 32, No. 4, 590–603

0012-1649/96/$3.00

Family Environment and Adolescent Depressive Symptoms and Antisocial Behavior: A Multivariate Genetic Analysis

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Measures of family environment often show substantial differences between children in the same family and are thus nonshared environment candidates. A key question is whether differential environments are related to differential outcomes when genetic differences between children in the same family are controlled. Parent and child reports and observations of family interactions were used to assess familial negativity and adolescents' depressive symptoms and antisocial behavior in a genetically informative sample of 719 same-sex sibling pairs ranging from 10 to 18 years old. Analyses revealed that parental and sibling negativity is significantly related to adolescent adjustment through nonshared environmental processes, although genetic factors account for most of the association between parental negativity and adolescent adjustment.

One of the most interesting and important findings from the field of behavioral genetics concerns nurture rather than nature. Results from family, twin, and adoption studies converge on the conclusion that environmental influences on behavioral development operate in a nonshared manner, making children in the same family different from one another rather than similar, with only a few exceptions such as antisocial behavior (Plomin, Chipuer, & Neiderhiser, 1994; Plomin & Daniels, 1987). Family environment can be investigated as a source of nonshared environment by studying child-specific environments of siblings (Reiss, 1993). Such studies indicate that children in the same family experience surprisingly different environments (Dunn & Plomin, 1990).

In the present study, the adjustment outcomes of interest were adolescent depressive symptoms and antisocial behavior. Rates of these problems that are quite stable during middle childhood increase over the course of adolescence (e.g., Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; McGee, Feehan, Williams, & Anderson, 1992). Because of this increase, and because actions and decisions undertaken during adolescence can have long-term effects, this is a particularly important time in which to explore depressive symptoms and antisocial behavior. The participants in the present study displayed depressive symptoms and antisocial behavior within the normal range. Symptom checklists that yield dimensional measures of adjustment were used for this unselected sample rather than clinical diagnostic criteria. This strategy has the advantage of pertaining to the adjustment of the vast majority of the population as well as providing greater statistical power than would categorical diagnoses. This does mean, however, that interpretations drawn from this study apply primarily to occasional despondency rather than depression, and to "trouble making" rather than conduct disorder.

In an attempt to identify predictors of antisocial behavior and depressive symptoms, many researchers have turned to the family environment. A lack of family cohesion has been implicated in adolescent depression (Rubin et al., 1992). Simons, Robertson, and Downs (1989) postulated that parental rejection "causes" delinquency. In addition, Patterson, DeBaryshe, and Ramsey (1989) argued that ineffective parenting practices are the original source of antisocial behavior. In their discussion of intergenerational transmission of antisocial behavior, only parenting practices were mentioned as a possible link between parents and their offspring.

These statements of causality have ignored two possible mediators pointed to by genetic research. The first is that common genetic influences could account for associations between parenting practices and children's outcome. The second is that insufficient attention has been paid to nonshared environmental influences that genetic studies have found to be important. Research has begun to identify differential experiences of siblings that relate to differences in the children's developmental outcomes. These associations are seen most clearly for differential

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This research is part of the Nonshared Environment in Adolescent Development project, supported by the National Institute of Mental Health (MH-43373) and the William T. Grant Foundation.

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negative experiences such as conflict and for negative outcomes such as behavioral problems (Daniels, Dunn, Furstenberg, & Plomin, 1985; Dunn, Stocker, & Plomin, 1990; Hetherington, Reiss, & Plomin, 1994). For example, children who experienced more power-assertive discipline than did their sibling are more likely to be anxious (McHale & Pawletko, 1992). In addition, in an ongoing study focused on this issue, the Nonshared Environment and Adolescent Development project (NEAD; Reiss et al., 1994), strong associations were found between child-specific parental treatment, especially conflict, and children's behavior problems (Reiss et al., 1995).

The present article takes the next step in this program of research by examining the extent to which associations between differential sibling or parental negativity and their behavioral problems are mediated by nonshared environmental processes or by genetic factors. That is, one reason why siblings may differ is that biological siblings share on average half of their genes (Plomin, DeFries, & McClearn, 1990). This means that genetically influenced characteristics are expected to produce differences as well as similarities within pairs of siblings. Most domains of behavioral development show at least some genetic influence (Plomin, 1986; Plomin & McClearn, 1993), including behavioral problems (Rutter et al., 1990). More surprisingly, during the past decade it has become clear that most measures of family environment also show genetic influence (Plomin, 1994; Plomin & Bergeman, 1991). In NEAD, significant genetic influence has been found for parent and child reports of family environment (Plomin, Reiss, Hetherington, & Howe, 1994) and for ratings based on videotaped observations of family interaction (O'Connor, Hetherington, Reiss, & Plomin, 1995).

The presence of genetic influence on measures of the family environment is consistent with the idea that socialization is bidirectional. That is, when parents interact with their children, this interaction is affected by the child's behavior as well as that of the parent (Bell, 1968). This finding also supports the idea that children actively create and modify their environments (Scarr, 1992). If genetic factors contribute to measures of family environment as well as to measures of developmental outcomes, it is possible that genetic factors are also involved in associations between family environment and developmental outcomes (Plomin & Neiderhiser, 1992). Recent research supports this hypothesis (reviewed by Plomin, 1994). Concretely, genetic contributions to environment–outcome associations would indicate that the same genetic propensities in a child that elicit negativity from a parent (environment) also result in that child behaving in an antisocial manner (outcome).

The present article proposes a novel approach to the problem of disentangling sibling differences in nonshared environment from differences in genetic effects. The approach involves multivariate genetic analyses, the use of which represents an important advance in genetic research during the past decade. Basic genetic analyses are univariate; they decompose observed variance on a single measure into genetic and environmental components of variance.

Multivariate genetic analysis focuses on the covariance between traits, decomposing this covariance into its genetic and environmental components (Martin & Eaves, 1977; Neale & Cardon, 1992; Plomin & DeFries, 1979). Multivariate genetic analysis has been used in many contexts, primarily to study the extent to which genetic effects on one trait overlap with genetic effects on another trait. For example, genetic effects on measures of cognitive abilities intercorrelate, suggesting a general genetic factor, even though some genetic effects are unique to each measure (Pedersen, Plomin, & McClearn, 1994). Multivariate genetic analysis has also been extended to investigate genetic mediation of the association between environmental measures and developmental outcomes. For example, such analyses have suggested that genetic factors account for about half of the association between the Home Observation for Measurement of the Environment measure of family environment (Caldwell & Bradley, 1978) and Bayley mental development scores at 2 years of age (Braungart, Plomin, Fulker, & DeFries, 1992).

The relevance in the present context is that multivariate genetic analysis can assess the importance of nonshared environmental sources of environment–outcome covariance independent of genetic components of covariance. In this study, we applied multivariate genetic analysis to associations between measures of parental and sibling negativity and measures of adolescents' depressive symptoms and antisocial behavior in NEAD (a nonclinical sample) in order to disentangle nonshared environmental contributions from genetic and shared environmental contributions to these associations. If the association between mothers' negativity and adolescent depressive symptoms is mediated through nonshared environmental factors, this means that to the extent that mothers are differentially negative to their adolescent children, these children will also exhibit differential levels of depressive symptoms. Measures of familial negativity were chosen as promising nonshared environmental candidates because of their relation to adjustment in differential treatment research with siblings. In addition, conflict and other forms of negativity generally show stronger relationships to outcome than do positive aspects of the family environment (Maccoby & Martin, 1983), NEAD being no exception (Reiss et al., 1995). Our hypothesis was that significant nonshared environmental contributions to associations between familial environment and behavioral problems could be found independent of genetic and shared environment contributions for both depressive symptoms and antisocial behavior. Mounting evidence for genetic contributions to environment–outcome associations (Plomin, 1994) led us to expect significant genetic contributions as well.

METHOD

Participants

Participants were 719 same-sex sibling pairs taking part in the NEAD project, a geographically representative sample of two-parent families with a pair of adolescent siblings no more than 4 years apart in age (Reiss et al., 1994). It was also stipulated that the current marriage of the parents in the sample be at least 5 years in duration to ensure that none of the stepfamilies were in the unstable early phases of family formation. All of the children were also required to be residents in the household at least half of each week. Nondivorced families with full siblings were obtained through random-digit dialing of 10,000 telephone numbers throughout the United States; however, most twin and

stepfamilies were recruited through a national market panel of 675,000 households. The 719 families were primarily middle class (average family income was $25,000 to $35,000), reflecting the marital stability criteria, and of European ancestry (94% of mothers and 93% of the fathers were White). The average years of education were 13.6 for mothers and 14.0 for fathers.

The sample included children between 10 and 18 years (M = 14.5, SD = 2.2) and their siblings between 9 and 18 years of age (M = 12.9, SD = 2.2). The present design resulted in six sibling categories representing two family types: nondivorced and stepfamilies. The nondivorced families included 93 monozygotic (MZ) twin pairs, 98 dizygotic (DZ) twin pairs, and 95 full sibling pairs. Stepfamilies included 182 full sibling pairs, 109 half sibling pairs, and 130 unrelated sibling pairs. The three stepsibling groups were matched by age of oldest child and age spacing to increase the comparability of these groups. The sibling pairs include 363 pairs of brothers and 344 pairs of sisters, with nearly equal representation by gender in each of the six groups.

Zygosity of the twins was determined by tester, self-reports, and parent reports of physical similarity (e.g., eye and hair color) using a modified version of the Nichols and Bilbro (1966) zygosity questionnaire for adolescents. This method of zygosity classification has been shown to yield accuracy of over 90% when compared with tests of single-gene markers in blood (Nichols & Bilbro, 1966). Twelve of the twins, however, could not be classified with certainty and were excluded from these analyses.

Procedure

The families were interviewed and videotaped twice in their homes approximately 2 weeks apart. Each session was conducted by two testers and lasted approximately 3 hr. Questionnaires were completed by the parents and siblings concerning the family environment and siblings' adjustment. Family members also specified areas of disagreement, which they then discussed in dyadic, triadic, and tetradic combinations. Although home visits were conducted in part to videotape family interactions, the presence of the testers also facilitated more complete and accurate responding and attenuated collusion between family members as they completed the questionnaires. Each videotaped interaction lasted for 10 min. A global coding system of 5-point Likert scales (Hetherington & Clingempeel, 1992) was used to rate the videotaped interactions. The present study only used the dyadic interactions: the four parent–child dyads and the sibling interaction. Fourteen behaviors were coded, and an average rater reliability of 76% was attained.

Measures

Composites of familial negativity and of adolescent adjustment were used in the present study. Space permits only a brief overview of these measures. The Appendix provides a sample item from each of the questionnaire scales used in the composites. Details of the questionnaire and observational measures can be found in Reiss et al. (1994) and Hetherington and Clingempeel (1992), respectively. To summarize this plethora of measures, we formed composites on the basis of factor analysis (for details see Plomin, Reiss, et al., 1994). In all cases, the measures were standardized and summed with unit weights to form composites.

Questionnaires

Parental Negativity

Composites of negativity were formed for mother's report of her parenting and for child reports of mothering, as well as for both father and child reports of fathering. This domain was indexed by the following measures: Parent Discipline Behavior (Hetherington & Clingempeel, 1992), punitive and yielding to coercion subscales; Parent–Child Disagreements (Hetherington & Clingempeel, 1992); Parent–Child Relationship (Hetherington & Clingempeel, 1992), conflict subscale; and the Conflict Tactics Scale (CTS; Strauss, 1979), symbolic aggression subscale. These measures of negativity were designed to tap how often and how intense disagreements were, as well as to assess feelings of anger.

Sibling Negativity

A composite of sibling negativity was also formulated separately by reporter, yielding composites for mother reports, father reports, and adolescent reports. For parent reports of negative sibling behavior, three subscales—rivalry, aggressiveness, and avoidance—from the Sibling Inventory of Behavior (Schaefer & Edgerton, 1981) were used. The siblings' reports included these and additional measures: the CTS, symbolic aggression, and violence subscales; Sibling Disagreements (created for this project); and the Relationship Quality Survey (modified from Hetherington & Clingempeel, 1992), negative behaviors subscale.

Adolescent Adjustment

Depressive symptoms and antisocial behavior were the targeted areas of adjustment. These again were composited separately by reporter.

Depressive symptoms. Instead of relying on one measure, we used three to provide a more comprehensive picture of adolescent depressive symptoms. These measures were the Child Depression Inventory (Kovacs, 1983); the Behavior Problems Index (BPI; Zill, 1985), depression subscale; and the Behavior Events Inventory (BEI; Patterson, 1982), depression subscale.

Antisocial behavior. Antisocial behavior was measured by the BPI antisocial subscale and by the BEI antisocial subscale.

Observational Measures

Coders gave participants a global rating for several behaviors after watching each dyadic interaction. The global ratings were based on the frequency and intensity of the behaviors.

Familial Negativity

Three ratings were composited to measure negativity. They were anger/hostility, coercion, and transactional conflict. The same codes were used to rate the parents' behavior and how the adolescent siblings treated one another.

Adolescent Adjustment

Verbal and nonverbal evidence of dysphoria, anxiety, and withdrawal from family activity were used to index depressed mood. We used all displays of the adolescents' depressed mood (to their sibling and parents) to form an observational depressive symptoms composite. Antisocial behavior was assessed by the extent to which the adolescent was disrespectful or disruptive during the interactions.

Total Composites

Total composites were formed for each family environment domain (mothers' negativity, fathers' negativity, and sibling negativity) and for the two adolescent adjustment measures. These were formed by standardizing the within-reporter composites and summing these with unit weights. For example, the total mothers' negativity composite was computed by summing the standardized composites of mother's report of

her negativity, the adolescent's report of mother's negativity, and the observational measure of mother's negativity.

Although it can be argued that aggregation of this kind yields environmental measures that are too general, aggregation of informants is also advocated as a means of obtaining a more valid assessment of behavior (e.g., Epstein, 1983; Rushton, Brainerd, & Pressley, 1983). Given that this is the first study of its kind, these general indicators of negativity are a reasonable starting point. To assess the coherence of these composites, we calculated the Cronbach's alpha for each total composite by treating each component measure as an item. These Cronbach's alphas ranged from .71 to .87, pointing to the integrity of these composites.

Data-Analytic Strategy

Before any calculations, we corrected all scores for gender, age, and age difference effects using standardized residuals. This guarded against inflated correlations because all of the sibling pairs are of the same sex and the twins are of the same age.

Phenotypic correlations between familial negativity and depressive symptoms and antisocial behavior. To decompose covariance into its genetic and environmental components, there must be at least a moderate correlation between the variables of interest at the outset. For this reason, we first computed phenotypic correlations between measures of the familial negativity and the measures of depressive symptoms and antisocial behavior. Correlations were computed for the within-reporter composites as well as for the total composites.

Multivariate genetic analyses. Multivariate genetic analyses can be used to decompose the phenotypic covariance between measures of family environment and developmental outcome into genetic, shared environmental, and nonshared environmental components (Plomin, DeFries, & McClearn, 1990). The essence of multivariate genetic analysis is the cross-sibling correlation, which is the correlation between one sibling's environmental measure and the second sibling's outcome measure. According to quantitative genetic theory, if genetic influences are important sources of phenotypic covariances, then the cross-sibling correlations will show the following pattern: MZ twins > DZ twins and full siblings > half siblings > unrelated siblings. If shared environment

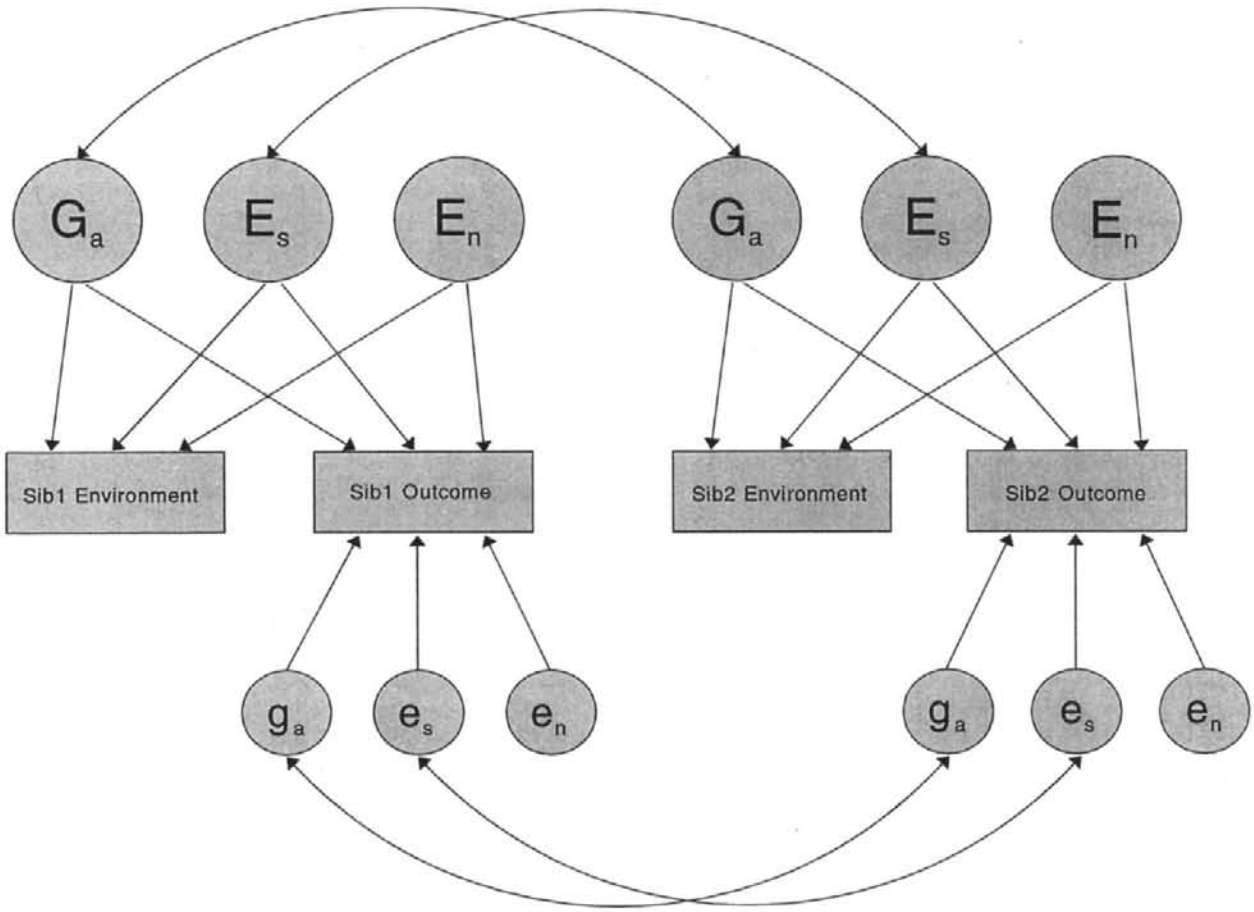

Figure 1. Path diagram of the model. Sib1 Environment, Sib1 Outcome, Sib2 Environment, and Sib2 Outcome are measured variables for the two siblings. The latent variables G_a, E_s, and E_n are the common genetic factor, the common shared environmental factor, and the nonshared environmental factor, respectively, that are shared by the environmental measure and the outcome measure. The latent variables g_a, e_s, and e_n represent the genetic, shared environmental, and nonshared environmental influences, respectively, that are unique to the outcome measure. The curved, two-headed arrows indicate correlations between the variables they connect, and the one-headed arrows represent paths, standardized partial regressions of the measured variable on the latent variable.

is a substantial mediator of the correlation between two variables, the cross-sibling correlation will be similar across all sibling types. In the present design, the cross-sibling correlation between unrelated siblings is the most direct index of shared environment because these siblings share only environmental influences. Nonshared environmental mediation is implicated to the degree that genetic and shared environmental sources of covariance cannot explain phenotypic covariance. The most direct test of this in the NEAD project is the difference between the phenotypic correlation and the cross-sibling MZ correlation.

A model-fitting approach provides a more powerful analysis of sibling resemblance than examining patterns of cross-sibling correlations. Model fitting analyzes the data for different sibling types simultaneously, tests the fit of the model, and makes assumptions explicit (Eaves, Last, Young, & Martin, 1978; Jinks & Fulker, 1970; Loehlin, 1987). In this study, we performed maximum-likelihood model-fitting analyses using LISREL VIII (Jöreskog & Sörbom, 1993). These modeling techniques have been explained in more detail elsewhere (see Boomsma, Martin, & Neale, 1989; Boomsma & Molenaar, 1986; Fulker, Baker, & Block, 1983; Neale & Cardon, 1992).

The bivariate models conducted for this study use cross-sibling covariances to decompose the covariance between a measure of the environment and a measure of the outcome into common and unique genetic and environmental components. Figure 1 represents a bivariate model with six latent variables. Three latent variables (G_a, E_s, and E_n) reflect the genetic and environmental influences common to both measures. The three additional latent variables (g_a, e_s, and e_n) reflect genetic and environmental influences unique to the outcome measure.

This path diagram reflects the assumptions of the model: no assortative mating, no selective placement of stepsiblings, shared and nonshared environmental effects are equal across sibling type, genetic effects are additive in nature, and genotype–environment interaction is negligible. Assortative mating, which occurs when parents choose each other because of their similarities, inflates heritability estimates for comparisons with unrelated or half siblings but deflates heritability estimates for comparisons based on MZ and DZ twins. Thus, the net effect of assortative mating on NEAD heritability estimates is negligible; moreover, assortative mating for psychological traits is modest (Plomin et al., 1990). Selective placement of stepsiblings, which could occur if similarity of stepsiblings contributes to the creation of the stepfamily, would inflate estimates of shared environment. Again, the multiple-group design of NEAD attenuates this potential problem and permits comparisons between the unrelated sibling group and other sibling groups. This is also the case for the assumption that shared and nonshared environmental effects are equal across sibling type. Tests of this assumption in previous NEAD research indicates the reasonableness of this assumption (O'Connor et al., 1995; Plomin, Reiss, et al., 1994). Concerning the assumption of additive genetic effects, we have also tested models that include nonadditive effects, but in all cases results were similar. Genotype–environment interaction (i.e., conditional effects of the environment dependent on genotype) could account for associations between environmental and outcome measures beyond associations uncovered by additive effects of environment. However, it is reasonable and typical to begin with the investigation of additive effects: The present results indicate that additive effects are considerable. Additional information about these assumptions is available (Loehlin, 1992; Neale & Cardon, 1992; Plomin et al., 1990).

The model in Figure 1 takes into account genetic theory at the level of the correlations between the latent variables. The curved double-headed arrow linking common genetic influence (G_a) between the two siblings represents the correlation of the common genetic influence for the two siblings. In the case of the MZ twins, this path is set to 1, because these twins share 100% of their genes. In the case of the DZ twins, and full siblings in both intact and stepfamilies, this path is set to .50 because these sibling pairs share approximately 50% of their segregating genes, assuming additive genetic effects and no assortative mating. For half siblings, the path is set to .25 because their genetic relatedness is .25, and for unrelated siblings the path is set to 0. The curved double-headed arrow linking common shared environmental influence (E_s) for the siblings represents the correlation of the common shared environmental influence. This correlation is set to 1 in all cases, thereby equating the shared environment across each of the sibling types. The common nonshared environment latent variables (E_n) that link the environment and outcome are not joined by a curved double-headed arrow because nonshared environmental influences are not shared by siblings. The path coefficients for the latent variable E_n represent the degree to which sibling differences in environment relate to sibling differences in outcome, having taken genetic (G_a) variability into account. It should be noted here that unlike the univariate case, common nonshared environmental influence does not include measurement error. This is because the model is decomposing covariance rather than variance, and this is free from random error (Pedhazer & Schmelkin, 1991).

The curved double-headed arrows linking the unique genetic and shared environmental influences (g_a and e_s) represent the correlation of the unique influences for the two siblings. The path linking unique genetic effects is set in the same manner as was described earlier, reflecting the differences in genetic relatedness across the sibling groups. The path linking unique shared environmental effects is again set to 1 for all of the sibling groups.

RESULTS

Previous NEAD publications have presented results of genetic analyses of the measures of family environment (O'Connor et al., 1995; Plomin, Reiss, et al., 1994). The present article focuses on the application of multivariate genetic analysis to associations between these measures of family environment and of adolescent depressive symptoms and antisocial behavior.

Phenotypic Correlations Between Familial Negativity and Depressive Symptoms and Antisocial Behavior

We computed correlations for each within-source composite of negativity and each within-source composite of adolescent adjustment. In addition, we calculated total composites (incorporating all sources) of familial negativity and total composites of adolescent adjustment. Because of the many correlations resulting from the within-source composites, only their pattern is described.

The higher correlations were found within source. These higher correlations may be due to rater bias. A more interesting interpretation is that the behavior of children may vary across

Table 1
Phenotypic Correlations Between Total Familial Negativity Composites and Total Adjustment Composites (N = 1350–1374)

Adolescent adjustment	Negativity		
	Mother's	Father's	Sibling
Depressive symptoms	.33*	.37*	.23*
Antisocial behavior	.60*	.57*	.51*

* $p < .001$.

situations, allowing for context-specific associations. The within-source correlations of negativity with depressive symptoms ranged from .06 to .51. The between-source correlations of negativity with depressive symptoms (e.g., mothers' report of her negativity with fathers' report of the adolescents' depressive symptoms) ranged from .04 to .29. The correlations between negativity and antisocial behavior were more substantial. The within-source correlations ranged from .19 to .62, and the between-source correlations ranged from .10 to .37.

The correlations using the total composites are presented in Table 1. Negativity in the family environment is moderately associated with depressive symptoms (.23 to .37) and substantially associated with antisocial behavior (.51 to .60). From this large number of relationships explored, we limited our multivariate genetic analyses to the correlations involving the total composites of negativity and the total composites of depressive symptoms and antisocial behavior. The total composite relationships were chosen because they attenuate the bias of a single reporter while still yielding moderate correlations.

Intraclass and Cross-Sibling Correlations

Intraclass and cross-sibling correlations for the total family environment composites of negativity and total composites of adolescent adjustment are included in Table 2. We calculated intraclass correlations using a double-entry procedure and included these correlations for each of the five main variables of the study to provide basic univariate information as well as to aid in the interpretation of the cross-sibling correlations.

As was described in the Method section, cross-sibling correlations (the correlation between one sibling's environmental measure and the second sibling's outcome measure) can be examined to anticipate the results of model fitting. The "clearest" example in Table 2 is that of mothers' negativity and antisocial behavior. The correlation for MZ twins (.54) is greater than the average correlation for the DZ twins and full sibling in both nondivorced and stepfamilies (.31). The correlation for half-siblings is a bit high at .34, but the .18 correlation for unrelated children in stepfamilies is again lower. Overall, this pattern suggests that there is a substantial genetic contribution to the covariance of mothers' negativity and antisocial behavior. The modest cross-correlation for unrelated siblings indicates that shared environment also contributes to the covariance. The very large MZ cross-correlation (.54), compared with the phenotypic correlation (.60 in Table 1), however, suggests that the nonshared environment contribution will be quite small.

Even when the MZ and DZ cross-correlations do not indicate genetic influence, all possible comparisons of the correlations for the groups can suggest genetic influence on balance. No single comparison is more important than any other. Calculating the average correlation for the siblings that share half of their genes (DZ twins and full siblings in both nondivorced and stepfamilies) and comparing this with the MZ twins is more informative. When this is done, each association except for that of sibling negativity and antisocial behavior indicates genetic mediation. The results for sibling negativity and antisocial behavior are not as straightforward to interpret. In this case, the MZ correlation is out of step with the other sibling groups. The average correlation for the DZ twins and full siblings in both nondivorced and stepfamilies (.46) is greater than the MZ correlation (.40), although it does exceed the half-sibling correlation (.41), which slightly exceeds the correlation for unrelated children in stepfamilies (.40). On balance, the results for all six groups suggest some slight genetic contribution to the phenotypic correlation between sibling negativity and antisocial behavior. In this example, the lower MZ cross-correlation (.40) as compared with the phenotypic correlation (.51 in Table 1) suggests a greater nonshared environmental contribution than the previous example of mothers' negativity and depressive symptoms. However, because the cross-correlations for the other sibling groups are closer to the phenotypic correlation, only a modest contribution of nonshared environment is expected. The benefit of model fitting is that this piecemeal approach is avoided; each comparison is taken into account simultaneously and weighted appropriately.

Table 2
Intraclass and Cross-Sibling Correlations for Total Familial Negativity Composites and Total Adjustment Composites

	MZ (n = 93)	DZ (n = 98)	FN (n = 93)	FS (n = 180)	HS (n = 108)	US (n = 130)
Intraclass correlations						
Mothers' negativity	.77	.53	.51	.52	.49	.19
Fathers' negativity	.67	.68	.45	.47	.44	.45
Sibling negativity	.90	.83	.83	.83	.76	.85
Depressive symptoms	.64	.25	.15	.24	.29	.26
Antisocial behavior	.80	.65	.46	.46	.47	.30
Cross-sibling correlations						
Mothers' negativity and depressive symptoms	.21	.01	.07	.12	.19	.13
Mothers' negativity and antisocial behavior	.54	.34	.28	.30	.34	.18
Fathers' negativity and depressive symptoms	.23	.04	.07	.24	.14	.28
Fathers' negativity and antisocial behavior	.44	.43	.25	.25	.32	.24
Sibling negativity and depressive symptoms	.30	.06	.20	.26	.32	.30
Sibling negativity and antisocial behavior	.40	.51	.48	.41	.41	.40

Note. MZ = monozygotic twins in nondivorced families; DZ = dizygotic twins in nondivorced families; FN = full siblings in nondivorced families; FS = full siblings in stepfamilies; HS = half-siblings in stepfamilies; US = unrelated children in stepfamilies.

Model-Fitting Results

The multivariate genetic model decomposes the phenotypic covariance between a measure of family environment and a measure of children's adjustment into genetic and environmental components of covariance. Using negativity and depressive symptoms as an example, the model "explains" 100% of the variance of depressive symptoms into genetic and environmental components of variance that covary with negativity (the common latent variables G_a, E_s, and E_n) and genetic and environmental components of variance of depressive symptoms that do not covary with negativity (the residual latent variables g_a, e_s, and e_n). If a pair of paths that connect negativity and depressive symptoms through a common latent variable are both significant (according to the standard errors), this indicates that the latent variable contributes significantly to the phenotypic correlation between negativity and depressive symptoms.

Thus, our hypothesis that nonshared environmental processes contribute to the phenotypic correlation between negativity and depressive symptoms, independent of the genetic contribution to the phenotypic correlation, requires that the pair of paths connecting negativity and depressive symptoms via the latent variable E_n are both significant. A significant genetic contribution to the phenotypic correlation is indicated if the pair of paths from the latent common variable G_a are both significant. Similarly, the significance of the pair of paths via E_s indicates a significant contribution of shared environmental processes to the phenotypic correlation. The significance of the path from the residual latent variable e_n indicates whether there is significant nonshared environmental influence on depressive symptoms that does not covary with nonshared environmental effects on negativity. Similarly, the significance of the paths from the residual variables g_a and e_s describes components of variance for the outcome measures independent of genetic and shared environmental effects on negativity.

Mothers' Negativity

Figure 2 depicts the results of multivariate genetic analyses between mothers' negativity and depressive symptoms. The path coefficients are constrained to be identical for the two siblings. Therefore, for clarity of presentation, only one half of the full path diagram (see Figure 1) is shown. The path coefficients indicate how the variance of the depressive symptoms measure has been decomposed into genetic and environmental components (G_a, E_s, E_n) that covary with the mothers' negativity composite and residual components of variance (g_a, e_s, e_n) that do not covary with mothers' negativity. Because the path coefficients are standardized, squaring them indicates the proportion of variance explained by a path. Thus, squaring and summing the six path coefficients leading to the depressive symptoms measure yields 1.0, the total variance of the depressive symptoms measure. The model attributes the variance of depressive

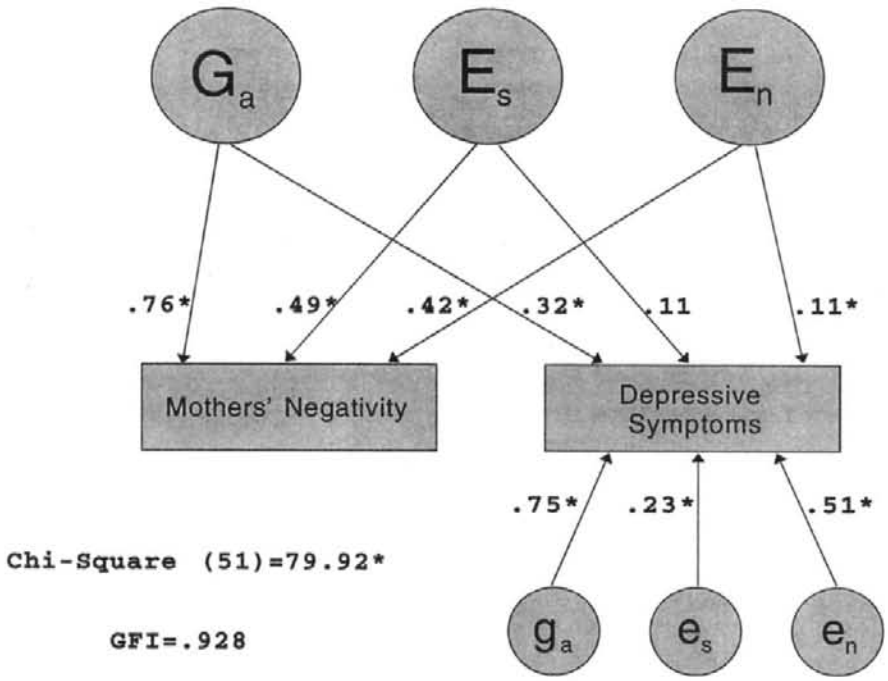

Figure 2. Bivariate model-fitting results between mothers' negativity and depressive symptoms. Asterisks denote loadings significant at $p < .05$. The latent variables G_a, E_s, and E_n are the common genetic factor, the common shared environmental factor, and the nonshared environmental factor, respectively, that are shared by the environmental measure and the outcome measure. The latent variables g_a, e_s, and e_n represent the genetic, shared environmental, and nonshared environmental influences, respectively, that are unique to the outcome measure. GFI = goodness-of-fit index.

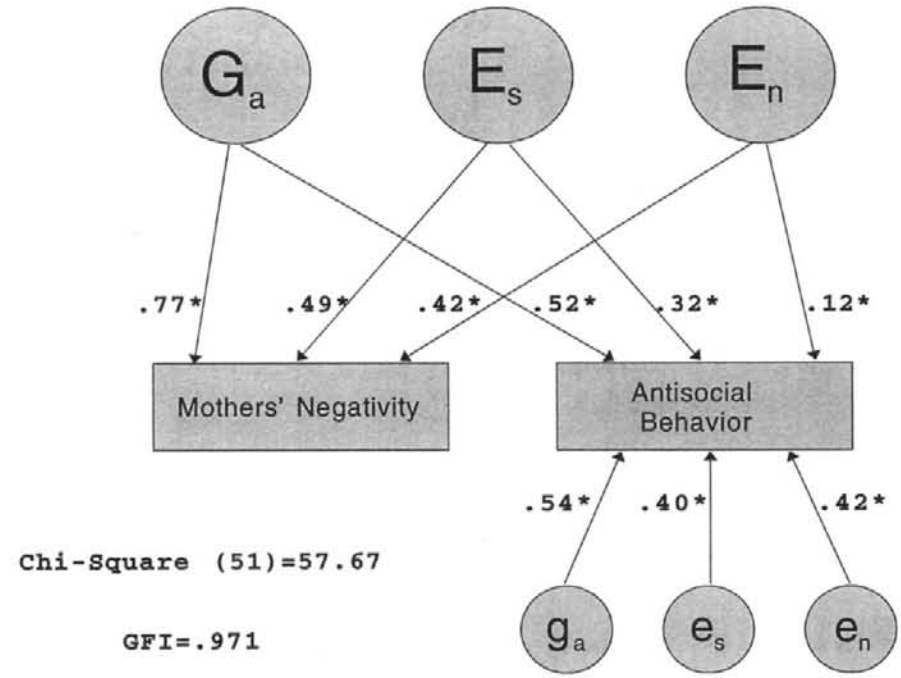

Figure 3. Bivariate model-fitting results between mothers' negativity and antisocial behavior. Asterisks denote loadings significant at $p < .05$. The latent variables G_a, E_s, and E_n are the common genetic factor, the common shared environmental factor, and the nonshared environmental factor, respectively, that are shared by the environmental measure and the outcome measure. The latent variables g_a, e_s, and e_n represent the genetic, shared environmental, and nonshared environmental influences, respectively, that are unique to the outcome measure. GFI = goodness-of-fit index.

symptoms to substantial genetic influence, negligible shared environmental influence, and moderate nonshared environmental influence. Two indexes of fit were used to assess the congruence of the data and the model. The chi-square value of 79.9 is significant, indicating a lack of fit between the model and the data. The goodness-of-fit index (GFI) value of .93, however, suggests a reasonable fit to the data.

The key issue for the present purpose is the nonshared environmental link between mothers' negativity and depressive symptoms. The path coefficients are .42 and .11, and the asterisks indicate that both are statistically significant. This suggests that mothers' negativity is significantly associated with depressive symptoms through nonshared environmental processes independent of genetic and shared environmental processes. As noted previously, this nonshared environmental link is free from measurement error. The significant path coefficients of .76 and .32 for the common genetic factor (G_a) suggest significant genetic mediation as well. The nonsignificant path coefficient of .11 for the shared environment parameter (E_s) implies a nonsignificant contribution of shared environment to the correlation between mothers' negativity and depressive symptoms.

It is possible to go beyond statistical significance to estimate the extent to which the phenotypic correlation between mothers' negativity and depressive symptoms is mediated by nonshared environment, by shared environment, and by genetic factors. Because the path coefficients are standardized, the product of the two paths for each parameter linking negativity and depressive symptoms estimates that parameter's contribution to the phenotypic correlation. For example, for the nonshared environment parameter (E_n), $.42 \times .11 = .05$. This means that .05 of the phenotypic correlation of .33 between negativity and depressive symptoms is due to nonshared environment. A similar proportion of the phenotypic correlation is due to shared environment (i.e., $.49 \times .11 = .05$). Most of the phenotypic correlation is due to genetic mediation (i.e., $.76 \times .32 = .24$). The sum of these components of covariance yields the model's estimate of the correlation of .34 (i.e., $.05 + .05 + .24 = .34$).

The significance of the residual e_n parameter indicates that there are significant nonshared environmental effects on depressive symptoms that are not explained by nonshared environmental effects due to mothers' negativity. Squaring the e_n path coefficient of .51 indicates that 26% of the variance of depressive symptoms is due to such residual nonshared environmental influences, which includes error of measurement. Squaring the E_n path coefficient of .11 suggests that only 1% of the total variance of depressive symptoms is due to nonshared environmental processes involved in mothers' negativity. This nonshared environmental link, however, does not include error of measurement because it is a portion of covariance rather than variance. In summary, nonshared environmental processes contribute significantly to the phenotypic correlation between mothers' negativity and depressive symptoms, even though the magnitude of its contribution is modest.

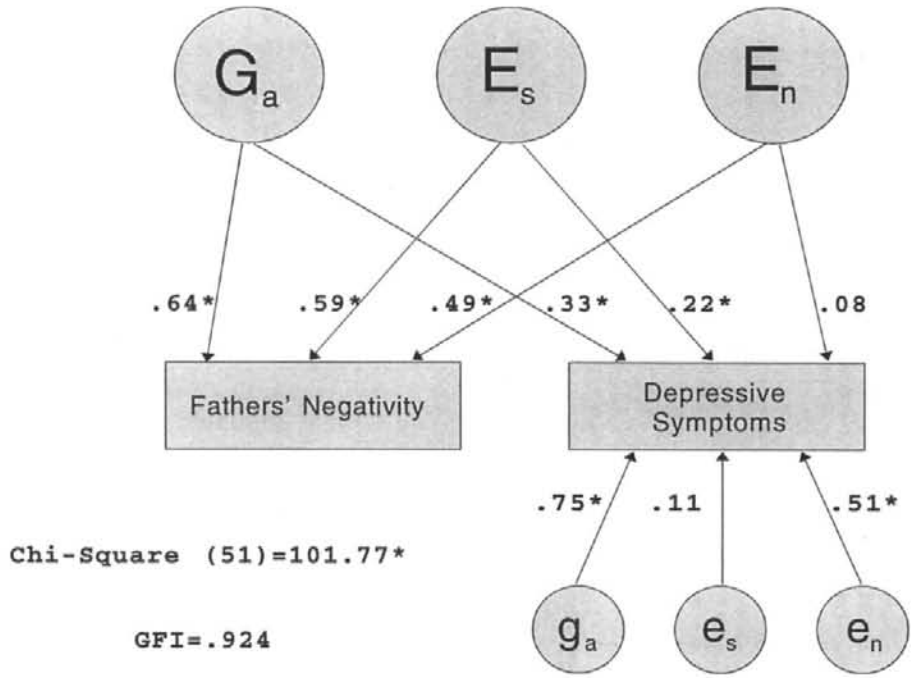

Figure 4. Bivariate model-fitting results between fathers' negativity and depressive symptoms. Asterisks denote loadings significant at $p < .05$. The latent variables G_a, E_s, and E_n are the common genetic factor, the common shared environmental factor, and the nonshared environmental factor, respectively, that are shared by the environmental measure and the outcome measure. The latent variables g_a, e_s, and e_n represent the genetic, shared environmental, and nonshared environmental influences, respectively, that are unique to the outcome measure. GFI = goodness-of-fit index.

Figure 3 illustrates the results of multivariate genetic analyses of mothers' negativity as related to antisocial behavior. As expected from the literature, antisocial behavior shows greater shared environmental influence than depressive symptoms, although nonshared environment and genetic factors are also important. Despite the presence of shared environment and genetic influence, nonshared environment contributes significantly to the correlation between mothers' negativity and antisocial behavior independent of shared environmental and genetic influence. The common nonshared environmental parameter E_n accounts for .05 of the correlation between mothers' negativity and antisocial behavior (i.e., $.42 \times .12 = .05$). Shared environment also contributes significantly to the correlation (i.e., $.49 \times .32 = .16$). Similar to the results for depressive symptoms, genetic factors account for most of the correlation (i.e., $.77 \times .52 = .40$).

Fathers' Negativity

Figures 4 and 5 present the model-fitting results for fathers' negativity. The results are similar to those for mothers' negativity reported in Figures 2 and 3. Although the composite measure of depressive symptoms (Figure 4) just misses significance for nonshared environmental mediation, the magnitude of the nonshared environmental link is similar (i.e., $.49 \times .08 = .04$). Results for fathers' negativity as related to antisocial behavior (see Figure 5) are also similar to those for mothers' negativity (see Figure 3). Nonshared environment contributes significantly to the correlation (i.e., $.51 \times .13 = .07$). For both depressive symptoms and antisocial behavior, genetics and shared environment also significantly mediate the associations with fathers' negativity.

Sibling Negativity

The results of multivariate genetic analyses of relationships between sibling negativity and depressive symptoms and between sibling negativity and antisocial behavior are shown in Figures 6 and 7, respectively. As reported earlier in relation to phenotypic correlations (see Table 1), sibling negativity yielded a relatively low correlation with depressive symptoms. For this reason, the multivariate genetic results are less clear. Nonshared environment contributes negligibly to the phenotypic correlation ($.35 \times .01 = .00$). The estimated phenotypic correlation of .23 is largely due to shared environment (i.e., $.89 \times .28 = .25$). In this case, genetic factors do not contribute significantly to the correlation (i.e., $.30 \times .00 = .00$).

The results for sibling negativity are clearer for antisocial behavior (see Figure 7) because the phenotypic correlation is greater ($r = .51$). Again, the nonshared environment contributes negligibly to the correlation between sibling negativity and antisocial behavior (i.e., $.34 \times .03 = .01$). The genetic contribution is significant and moderate (i.e., $.33 \times .38 = .13$). As in the case of sibling depression, however, most of the correla-

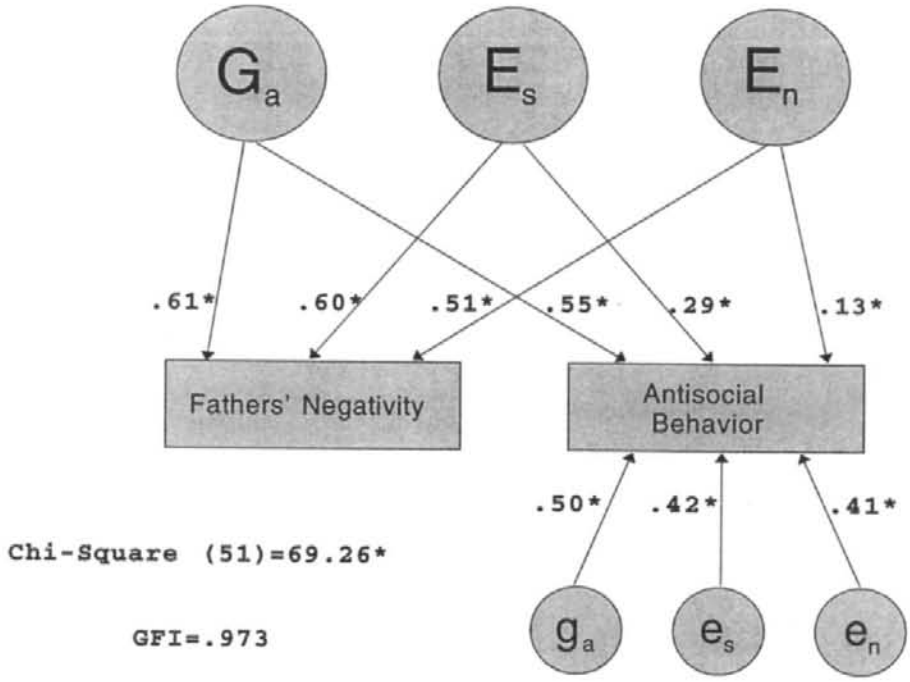

Figure 5. Bivariate model-fitting results between fathers' negativity and antisocial behavior. Asterisks denote loadings significant at $p < .05$. The latent variables G_a, E_s, and E_n are the common genetic factor, the common shared environmental factor, and the nonshared environmental factor, respectively, that are shared by the environmental measure and the outcome measure. The latent variables g_a, e_s, and e_n represent the genetic, shared environmental, and nonshared environmental influences, respectively, that are unique to the outcome measure. GFI = goodness-of-fit index.

tion between sibling negativity and antisocial behavior is explained by shared environmental influences.

Of the six models tested, all had significant chi-square values with the exception of mothers' negativity and antisocial behavior. As previously stated, this indicates a lack of fit between the model and the data. To see whether it was variance differences between the six sibling types that was causing the lack of fit, we tested the models using correlation matrices instead of covariance matrices. This resulted in a good fit for all of the models except for fathers' negativity and depressive symptoms. This suggests that variance differences were a cause of the lack of fit, and not the model itself. Because of the continued lack of fit for fathers' negativity and depressive symptoms, caution in interpretation for that association is warranted.

DISCUSSION

These multivariate genetic analyses of the covariance between measures of familial negativity and measures of adolescents' adjustment supported both hypotheses: (a) Significant nonshared environmental contributions to the environment–adjustment associations were found independent of genetic and shared environmental contributions, and (b) significant genetic contributions were also implicated.

Concerning the first hypothesis, significant nonshared environmental links with depressive symptoms emerged for mothers' negativity. For antisocial behavior, nonshared environment contributed significantly to its covariance with both mothers' negativity and fathers' negativity. These results support previous differential treatment research that had used siblings. Even when genetic differences are controlled, differential treatment affects adolescent adjustment. That is, if adolescents are the object of more parental negativity than is their sibling, they are also more likely to experience adjustment difficulties.

Although nonshared environment contributes significantly to the covariance between parental negativity and adolescent adjustment, the magnitude of its contribution is modest. However, unlike univariate analyses of nonshared environment, the nonshared environmental contribution to the covariance between measures of the family environment and adolescent adjustment is free from measurement error, although it may contain systematic error such as rater bias. It is worth bearing in mind that these results in no way discredit the importance of nonshared environment. Both depressive symptoms and antisocial behavior are influenced by nonshared environmental factors. Familial negativity was the "candidate" source of nonshared environment tested in these analyses. This candidate did not turn out to be a major source of nonshared environment for these adjustment outcomes. Perhaps a nonfamilial measured environment will prove to be the key to the "black box" of nonshared environment for adolescent adjustment.

Finding significant though modest nonshared environmental contributions to correlations between familial negativity and

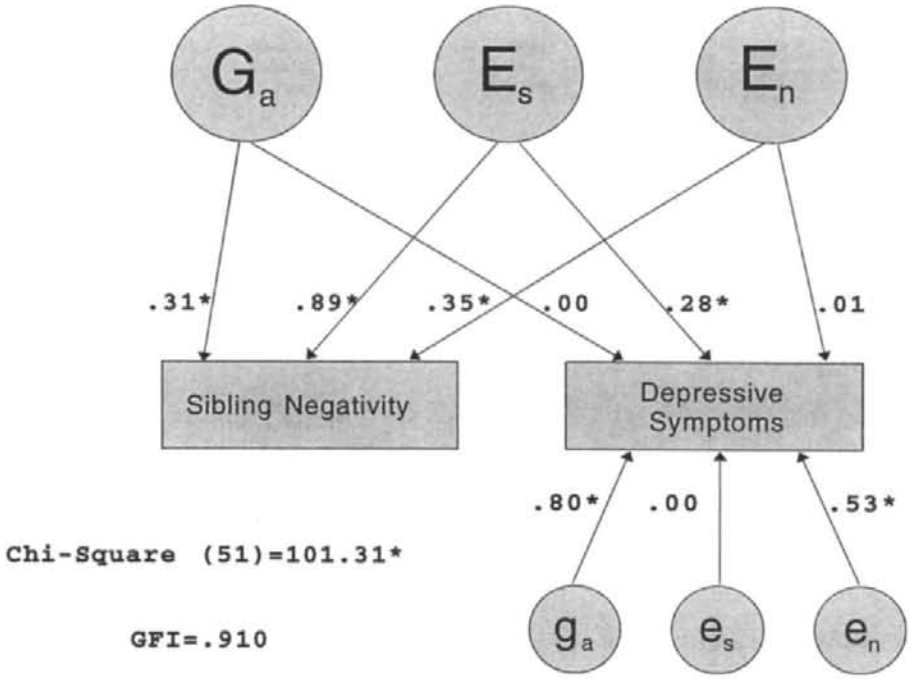

Figure 6. Bivariate model-fitting results between sibling negativity and depressive symptoms. Asterisks denote loadings significant at $p < .05$. The latent variables G_a, E_s, and E_n are the common genetic factor, the common shared environmental factor, and the nonshared environmental factor, respectively, that are shared by the environmental measure and the outcome measure. The latent variables g_a, e_s, and e_n represent the genetic, shared environmental, and nonshared environmental influences, respectively, that are unique to the outcome measure. GFI = goodness-of-fit index.

adolescent adjustment was overshadowed by the results concerning the second hypothesis. Not only is the genetic contribution to the covariance between familial negativity and adolescent adjustment significant, it is very substantial. The genetic contribution to environment–outcome correlations was significant for all comparisons with the exception of sibling negativity and depressive symptoms, and the genetic contribution accounts for most of the phenotypic correlation between measures of parental negativity and adolescent depressive symptoms and antisocial behavior. The genetic mediation may be less for the associations involving sibling negativity than for those involving parental negativity because there is less genetic influence at the univariate level for sibling negativity than for parental negativity (see Plomin, Reiss, et al., 1994).

Finding a genetic contribution to correlations between family environment and adolescent adjustment depends on three requirements: The measure of environment must be heritable, the measure of adjustment must be heritable, and there must be overlap between the genetic effects on the environmental measure and the adjustment measure. Although it is now widely accepted among developmentalists that genetic factors contribute importantly to many behavioral outcome measures, it is only recently that genetic analyses have uncovered substantial genetic influence on measures of the environment, especially family environment (reviewed by Plomin, 1994). Once this conclusion of a genetic contribution to environmental measures is accepted, it is a reasonable step to consider that genetic effects on an environmental measure might overlap with genetic effects on an outcome measure, thus contributing to the phenotypic correlation between them.

The results from this study require that nongenetic studies be interpreted with caution. The vast majority of research relating "environmental" measures with adolescent adjustment has been phenotypic. As was discussed previously, the association between parental negativity and adolescent adjustment is often interpreted to mean that the parent's behavior is "causing" the child's behavior. The present study, however, has found that the association is largely not driven by the behavior of the parents. Instead, it is the children's genes that are reflected in both the parent's behavior and in the adolescent's adjustment. This does not mean that parenting behaviors are unimportant. These analyses describe the overall state of affairs for this sample; they should not be applied to individual families. In addition, these findings do not preclude the potential usefulness of parenting interventions; this is a statement of "what is" rather than "what can be."

The estimate of genetic contribution to the phenotypic correlation between family environment and adolescent outcome is an indication of genotype–environment correlation. Genotype–environment correlation is defined as the extent to which people experience particular environments as a result of their genotype (Plomin, 1994). In the present context, it is the degree to which genetic propensities influence the experience of familial negativity and influence the exhibition of depressive and an-

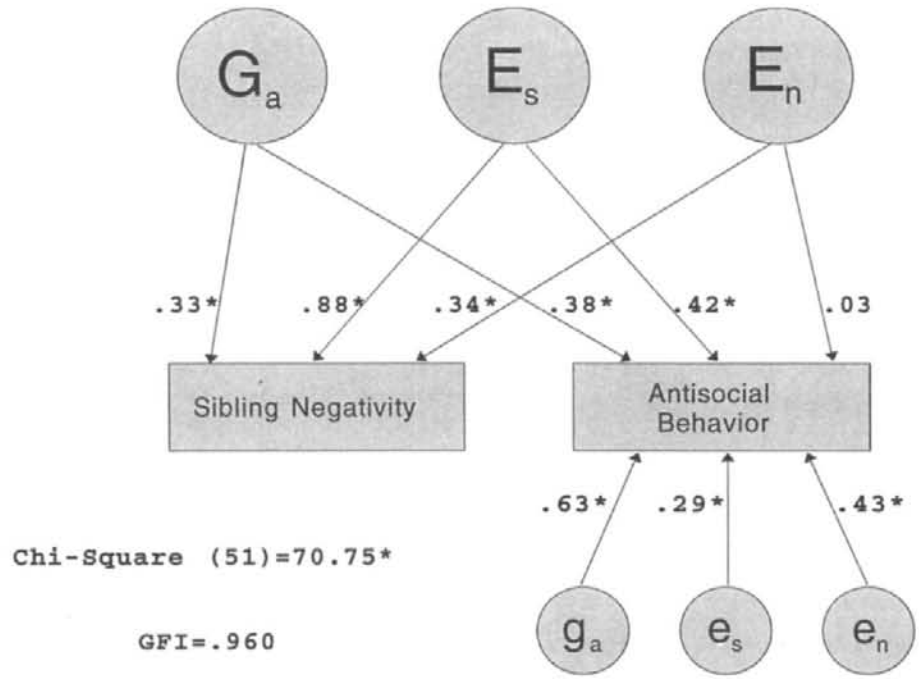

Figure 7. Bivariate model-fitting results between sibling negativity and antisocial behavior. Asterisks denote loadings significant at $p < .05$. The latent variables G_a, E_s, and E_n are the common genetic factor, the common shared environmental factor, and the nonshared environmental factor, respectively, that are shared by the environmental measure and the outcome measure. The latent variables g_a, e_s, and e_n represent the genetic, shared environmental, and nonshared environmental influences, respectively, that are unique to the outcome measure. GFI = goodness-of-fit index.

tisocial behaviors. Genotype–environment correlation has been categorized into three types: passive, reactive, and active (Plomin, DeFries, & Loehlin, 1977). That is, children may inherit environments along with genes from their parents (passive), parents or others may react to genetically influenced characteristics of the child (reactive), or children may seek out their own environmental niche suited to their genetic make-up (active). The bivariate models used here do not differentiate between the three types but provide an estimate of the overall effect. Intuitively, the reactive type of correlation seems to make most sense in this instance. It is quite plausible that an adolescent's genetic propensities that lead to adjustment difficulties would also lead to displays of negativity from family members. Although NEAD was not designed to discriminate types of genotype–environment correlation, it is possible to do so using other designs (Plomin, 1994), and this is an important direction for future research.

A limitation of the present study is that only measures of the family environment have been tested as nonshared environmental candidates. It is likely that the extrafamilial contexts that become increasingly important over the course of adolescence (Brown, 1990) will prove to be important sources of nonshared environment. The method used here also has the limitation that only a single measure of the environment can be tested at a time. More complex environmental processes that have been implicated in the development of depression and antisocial behavior (e.g., Earls, 1994; Harrington, 1994) will require designs that test the cumulative effect of multiple nonshared environmental candidates at once. Another limitation of this study is that for these families with more than two children, additional nonshared environment information related to other siblings is ignored.

Research is clearly needed to identify mediating processes entangled in measures of familial negativity and in their association with adjustment outcomes, as well as to extend the use of multivariate techniques into additional domains. We selected strong associations for analysis because multivariate genetic analysis of covariance requires that the measures covary. However, this selection of strong environment–outcome associations should not have biased the results toward finding that genetic factors are largely responsible for these phenotypic associations. Could it be that the strongest associations observed between parenting and children's adjustment are those that are most strongly mediated by genetic factors? Future research involving positive aspects of the family environment (which yield weaker associations with adjustment) will be able to answer this question.

This is one of the first studies to examine the genetic contribution to the prediction of adolescent adjustment from family environment. Nonetheless, convergence of results from studies using other genetic designs, samples, ages, measures, and approaches is clearly needed. Notably, the participants in this study were drawn from a nonclinical population. The etiology of individual differences in the normal range bear no necessary

relationship to the etiology of extremes. It is thus necessary that future research use participants at the extremes of these adjustment dimensions to see whether the patterns of results found here is similar for selected samples.

Moreover, the present analyses are based on contemporaneous assessments of family environment and adolescent adjustment that may have exaggerated the genetic contribution to their covariance. Especially needed are longitudinal analyses that permit cross-lagged analyses predicting later adjustment from earlier family environment. Three-year follow-up data are being collected in the NEAD project that will be able to address this issue.

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Appendix

Sample Items From Each of the Questionnaire Scales Used in the Composites

Parental Negativity

Parent Discipline Behavior, punitive: "yelled at you about something you did wrong."

Parent Discipline Behavior, yielding to coercion: "given in to you to make things easier for him."

Parent–Child Relationship, conflict: "How often does this person get into disagreements or fights with you?"

Conflict Tactics Scale, symbolic aggression: "insulted or swore."

Sibling Negativity

Sibling Inventory of Behavior, rivalry: "is very competitive with you."

Sibling Inventory of Behavior, aggressiveness: "gets angry with you."

Sibling Inventory of Behavior, avoidance: "stays away from you if possible."

Conflict Tactics Scale, symbolic aggression: "insulted or swore."

Conflict Tactics Scale, violence: "hit or tried to hit with something."

Sibling Disagreements: "your brother/sister took something of yours and didn't ask."

Relationship Quality Scale, negative behaviors: "how much does this person nag you about what you're doing wrong?"

Depressive Symptoms

Child Depression Inventory: Choose between these statements: "I am sad once in a while," "I am sad many times," and "I am sad all the time."

Behavior Problems Index, depression: "I felt or complained that no one loved me."

Behavior Events Inventory, depression: true/false inventory for past 24 hr, "felt withdrawn."

Antisocial Behavior

Behavior Problems Index, antisocial: "I bullied, or was cruel and mean to others."

Behavior Events Inventory, antisocial: "lied" or "cheated."

Received February 7, 1994
Revision received July 16, 1995
Accepted July 16, 1995 ■