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Genetic and Environmental Influences on the Relationships Between Family Connectedness, School Connectedness, and Adolescent Depressed Mood: Sex Differences

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This study investigated (a) genetic and environmental contributions to the relationship between family and school environment and depressed mood and (b) potential sex differences in genetic and environmental contributions to both variation in and covariation between family connectedness, school connectedness, and adolescent depressed mood. Data are from 2,302 adolescent sibling pairs (mean age = 16 years) who were part of the National Longitudinal Study of Adolescent Health. Although genetic factors appeared to be important overall, model-fitting analyses revealed that the best-fitting model was a model that allowed for different parameters for male and female adolescents. Genetic contributions to variation in all 3 variables were greater among female adolescents than male adolescents, especially for depressed mood. Genetic factors also contributed to the correlations between family and school environment and adolescent depressed mood, although, again, these factors were stronger for female than for male adolescents.

Researchers often find that differences in family and school environments are associated with differences in adolescent depressed mood. For example, the transition to secondary schooling is thought to be a precipitating factor in adolescent depression (Petersen et al., 1993). Likewise, Pedersen (1994) found that parental care, especially paternal care, differentiated adolescents with anxious–depressed symptomatology from adolescents without such symptoms. Similarly, Kandel and Davies (1982) reported that less closeness to parents predicted depressed mood in adolescents, particularly in female adolescents. Finally, a recent report from the National Longitudinal Study of Adolescent Health (Add Health) indicated that even after both demographic variables and specific risk factors were controlled for, family connectedness still explained approximately 15% of the variation in adolescent emotional distress. Similarly, school connectedness explained between 13% and 18% of the variation in adolescent emotional distress (Resnick et al., 1997).

Although the relationships between family and school environments and depression in adolescence are well documented, at least two central questions remain. The first question concerns the nature of these relationships. Specifically, the mechanisms through which the associations between environment and depression appear are often ambiguous. It may be that parental behaviors and school environments directly affect child and adolescent adjustment; in other words, more positive parenting and better school environments lead causally to more well-adjusted children. A second hypothesis is that characteristics of children and adolescents alter the responses of others toward them. This hypothesis has two parts: First, the behavior and characteristics of children and adolescents cause differential responses from the people in their environments. Alternatively (and not mutually exclusively), less well-adjusted children and adolescents may perceive their environments more negatively. A final hypothesis is that the relationships between processes and adjustment are, in fact, spurious; these associations are caused by some other factor entirely. There is an increasing body of research from behavioral genetic studies

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that suggests that genetics may be the elusive "third factor" responsible for at least part of the well-documented associations between environmental measures and adolescent adjustment. Moreover, in nongenetic research, it has been suggested that less positive family processes are simply mediators of the relationship between depressed mood in parents and depressed mood in adolescents (Petersen et al., 1993), which implies that some of the influence on variation in adolescent depressed mood may be genetic. Therefore, the first goal of this study was to use genetically informative data to estimate the genetic and environmental contributions to the covariation between family and school environments and adolescent depressed mood.

It is commonly acknowledged that genetic factors account for a substantial proportion of variance in a multitude of phenotypes, including adolescent body weight, cognitive ability, psychopathology, and delinquent behavior (Plomin & McClearn, 1993; Rowe, 1994). Given that parents provide their offspring with genes as well as environments, it has been hypothesized that the relationship between parenting behaviors and child and adolescent adjustment is caused by genetic similarities. This is generally thought of as a passive gene → environment correlation (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). For example, one might hypothesize that more depressed parents provide less nurturing environments for their children. Research on the effects of economic hardship and depression on parenting suggests that this is often the case (Conger et al., 1992, 1993; McLoyd, 1990). Thus, adolescents of depressed parents are more likely to "inherit" both a genetic predisposition toward depression and a less nurturing environment. Because other environmental influences, such as schools, do not contain genes, it is somewhat less intuitive to explain associations between such measures as school environment and adjustment in terms of genetic influences. Nevertheless, adolescents are not randomly distributed across environments; parents play a large role in determining the other environments to which their children are exposed.

Furthermore, children and adolescents are acknowledged to be active agents in creating their own environments, in both intra- and extrafamilial settings (Bell, 1968; Scarr & McCartney, 1983). Moreover, to the extent that genetically influenced characteristics of the individual modify the responses of others, then differences in genes can account for differential responses of nonfamily environments. In the behavioral genetic literature, these processes are referred to as active and reactive gene → environment correlations (Plomin et al., 1977; Scarr & McCartney, 1983). An example of an active gene → environment correlation is depressed adolescents isolating themselves from others, thereby prohibiting positive family and school relationships from forming. An example of a reactive gene → environment correlation is parents and teachers finding it harder to be more nurturing toward adolescents who are depressed.

Empirical support for the third-factor hypothesis comes from the accumulating body of research that has found significant genetic influences on "environmental" measures (see Plomin, 1995; Plomin & Bergeman, 1991; Plomin, Loehlin, & DeFries, 1985; Rowe, 1994), including social support (Kendler, 1997; Kessler, Kendler, Heath, Neale, & Eaves, 1992), life events (Foley, Neale, & Kendler, 1996; Kendler, Neale, Kessler, Heath, & Eaves, 1993), and objective measures of the home environment (Braungart, Fulker, & Plomin, 1992; Coon, Fulker, DeFries, & Plomin, 1990). Beginning with the early studies of twins conducted by Rowe (1981, 1983), researchers have discovered that perceptions of parenting are also genetically influenced. Genetic influences on perceptions of parenting have been found in twin samples (Kendler, 1996; Neale et al., 1994; Rowe, 1981, 1983), sibling samples (O'Connor, Hetherington, Reiss, & Plomin, 1995; Plomin, Reiss, Hetherington, & Howe, 1994), adoptive samples (Braungart et al., 1992; Coon et al., 1990), and even samples of reared-apart twins (Hur & Bouchard, 1995; Plomin, McClearn, Pedersen, Nesselroade, & Bergeman, 1988). Moreover, observational measures of parenting behaviors have been found to be genetically influenced (Lytton, 1977; O'Connor et al., 1995), suggesting that the genetic influences are not restricted to child and adolescent perceptions of parenting. Heritability estimates typically range from .15 to .60, with approximately one quarter to one third (on average) of the variation in parenting behaviors explained by differences in genes. Shared environmental influences are usually small and sometimes do not significantly differ from zero (e.g., Neale et al., 1994; Plomin et al., 1994), although it should be noted that heritability is lower and shared environmental influences are higher for dimensions of parenting that reflect negative (e.g., O'Connor et al., 1995; Plomin et al., 1994) or control-related (e.g., Neale et al., 1994; Plomin et al., 1988; Rowe, 1981, 1983) behaviors.

Furthermore, a great deal of research has estimated the genetic and environmental influences on depression, and most studies have reported heritability estimates between .25 and .50 (Kendler et al., 1994; Neale et al., 1994; Reiss et al., 1995; Tambs, Harris, & Magnus, 1995; Thaper & McGuffin, 1994; Wierzbicki, 1987) for both depressed mood and clinical depression (although the magnitude of heritability may be lower in clinically depressed individuals; see Eley, 1997; Rende, Plomin, Reiss, & Hetherington, 1993). Moreover, significant heritabilities have been found in older adult (e.g., McGue & Christensen, 1997), adult (e.g., Kendler et al., 1994; Kessler et al., 1992; Neale et al., 1994; Silberg et al., 1990; Tambs et al., 1995), and adolescent (e.g., Eley, 1997; Reiss et al., 1995; Wierzbicki, 1987) samples, although there is some evidence that in young children (i.e., younger than 10 years), familial resemblance for depressed mood may be due entirely to shared environmental factors (Thaper & McGuffin, 1994).

Given that (a) there is a substantial phenotypic relationship between environment and adolescent depression and (b) a significant part of the variation in both the environment and depression is due to genetic differences in adolescents, it is reasonable to examine whether part of the correlation between environment and depression is mediated genetically. Only a few studies to date have done so. Specifically, Bergeman, Plomin, Pedersen, and McClearn (1991) reported that 65% of the correlation between perceived social support and depressive symptomatology in their sample of older Swedish twins could be explained by common genetic influences. Similarly, in a study of adult female–female twins, Neale et al. (1994) found that the most parsimonious model that explained the relationship between retrospective reports of parenting style and depression was a model that contained common genetic influences.

Finally, to date, only one published study has examined the possible role of genetic factors in explaining the relationship between perceptions of parenting and adolescent depression. Pike, McGuire, Hetherington, Reiss, and Plomin (1996) found that more than 70% of the correlation between adolescent reports of maternal

negativity and adolescent depression was due to common genetic factors. Moreover, common shared environmental factors were much lower and did not achieve statistical significance. In contrast, however, the same study reported that genetic factors explained somewhat less of the relationship between paternal negativity and depression (55%) and that common shared environmental influences accounted for more than one third of the correlation (Pike et al., 1996). Thus, the first goal of the present study was to add to the handful of studies examining whether the relationship between the environment and adolescent depressed mood is mediated genetically.

One way in which this study adds to the existing research is that it focuses on two different types of "environmental" protective factors: family connectedness and school connectedness. Although genetic influences have been found for a variety of parenting behaviors (see Plomin, 1995; Plomin & Bergeman, 1991; Rowe, 1994), to our knowledge, this is the first study that has examined potential genetic influences on variation in adolescent reports of school connectedness. Because the school environment does not contain genes, it is possible that genetic influences mediate the relationship between parenting and depressed mood but not the relationship between school connectedness and depressed mood.

The second goal of this study was to investigate whether genetic and environmental influences on variation in family connectedness, school connectedness, and depressed mood and on the covariation among these variables are different for male adolescents and female adolescents. Many behavioral genetic studies of environmental measures lack adequate sample sizes or are restricted to same-sex pairs, inhibiting a thorough investigation of sex differences. Thus, little is known about sex differences in the relative genetic and environmental influences on adolescent perceptions of parenting. Furthermore, sex differences in mean levels have consistently been found in studies of adolescent depression and depressed mood (Allgood-Merten, Lewinsohn, & Hops, 1990; Angold & Rutter, 1992; Gore, Aseltine, & Colton, 1992; Kandel & Davies, 1982; Weissmann & Klerman, 1977; see also Birmaher et al., 1996; Petersen et al., 1993, for reviews). Although factors that contribute to differences in mean levels across groups do not necessarily contribute to individual differences, the reasons given for the higher rates of depression among female adolescents may suggest different etiologies.

On the one hand, it has been noted that sex differences in depression do not appear until adolescence (for reviews, see Birmaher et al., 1996; Brooks-Gunn, 1992; Petersen et al., 1993). This finding has led to the speculation that biological differences that are accentuated during puberty are responsible for the greater levels of depression found among female adolescents and women (Angold & Rutter, 1992; Brooks-Gunn, 1992; Petersen et al., 1993). This finding may suggest that certain genes related to depression are "turned on" during adolescence; thus genetic influences on variation in depressed mood may be greater for female than for male adolescents. There is some evidence for higher heritability of depression among women than men (e.g., Tambs et al., 1995), although not all studies find sex differences (e.g., Kendler et al., 1994; McGue & Christensen, 1997).

On the other hand, it has been hypothesized that gender differences in socialization, responsiveness to the environment, or both may be related to mean-level differences in depression (Angold & Rutter, 1992; Birmaher et al., 1996; Kandel & Davies, 1982). This hypothesis might imply that nonshared environmental influences have a greater impact on variation in depressed mood among female than among male adolescents. Finally, Gore et al. (1992) reported that level of parental education and standard of living failed to predict depressive symptoms in male adolescents but were significant predictors of depression in female adolescents. Thus, it is possible that shared environmental factors may be more important for female adolescents. Given the potential for sex differences in the etiology of adolescent depression, it seems judicious to also examine whether genetic and environmental influences on the associations between family and school environments and depressed mood differ for male and female adolescents; although given the lack of previous research, no definite hypotheses can be proposed.

A final strength of the present study is that it uses data from Add Health. In addition to being one of the largest and most recent studies of adolescent health and health-related behaviors, Add Health is the data set on which Resnick et al.'s (1997) study was based. According to the first published report from this data set, family connectedness and school connectedness were the most powerful predictors of multiple indicators of adolescent adjustment, including measures such as emotional distress, suicidality, violence, and substance use (Resnick et al., 1997). However, Resnick et al.'s study did not use data from the genetically informative sibling-pairs subsample, nor were sex differences examined. Thus, our use of data from the same study allows for an interesting comparison of Resnick et al.'s results with our own.

In conclusion, on the basis of previous research, we expected to find genetic influences on our two environmental measures: family connectedness and school connectedness. However, genetic factors may be less important for variation in school connectedness than for variation in family connectedness. Likewise, we expected that genetic factors would contribute to variation in depressed mood and that the heritability of depression might differ for male and female adolescents. Finally, we expected that at least part of the relationship between our environmental variables and depressed mood would be mediated by genetic factors, although the strength of the mediation might be moderated by sex.

Method

Sample and Design of the National Longitudinal Study of Adolescent Health

The present study used data from Add Health, a large, nationally representative, longitudinal study of adolescent health and health-related behaviors and of the causes and consequences of these behaviors. Add Health began with a total sample of more than 90,000 adolescents surveyed in school. The primary sampling frame was all high schools in the United States that had an 11th grade and an enrollment of at least 30 students. A random sample of 80 high schools was selected from this sampling frame, taking into consideration enrollment size, region, school type, ethnicity, and urbanicity. The largest feeder school for each high school was also included in the sample. Seventy-nine percent of the schools initially contacted agreed to participate. Schools that refused to participate were replaced by another school in the same sampling stratum, resulting in a final sample of 134 schools. Within these schools, an in-school questionnaire was completed by 90,118 of 119,233 eligible students (76%) in 7th grade through 12th grade.

By using both school roster information and information provided by adolescents during school interviews, a random sample of 15,243 adoles-

cents was also selected for detailed home interviews. This sample was stratified by sex and age. In-home interviews were completed by 12,188 (80%) of these adolescents. In addition to this core sample, a number of subsamples were also selected for home interviews. These subsamples included samples of adolescents with disabilities, adolescents from well-educated African American families, adolescents from typically understudied racial and ethnic groups, and a sibling-pairs sample. Overall, 20,745 adolescents completed in-home interviews. Other information obtained during Add Health included follow-up in-home interviews 1 year later of more than 17,000 adolescents, parent interviews, school administrator interviews, and data on community characteristics. Details on Add Health have been reported elsewhere (Resnick et al., 1997).

Sibling-Pairs Sample

The sibling-pairs sample was selected by using information from school rosters. Specifically, all adolescents who reported having a twin, a half sibling, or an unrelated sibling between 11 and 20 years of age residing in the same household were selected for inclusion in the home interview subsample. Home interview data were obtained from both the target adolescent and his or her sibling. The sibling pairs were selected regardless of whether they were present on the day of the school interview and regardless of whether the siblings attended the same school. An additional probability sample of full-sibling pairs was also selected for home interviews. Thus, the Add Health sibling-pairs sample consisted of 783 twin pairs, 1,252 full-sibling pairs, 442 half-sibling pairs, and 662 unrelated sibling pairs, resulting in a total sample size of 3,139 pairs, or 7,278 adolescents. Final determination of pair type was based on household roster information from the in-home questionnaire.

Twins' zygosity was determined primarily on the basis of both self-report of zygosity and 4 items concerning similarity and confusability of physical appearance. Self-report questions such as those used in this study are commonly used in large-scale twin research and have good reliability with twin zygosity determined on the basis of DNA. For example, Spitz et al. (1996) reported that a composite created by using 4 items similar to the ones used in the present study correctly identified more than 92% of twins. In the present study, definitive zygosity ratings were made only when self-reports of zygosity matched sibling reports of physical similarity. Furthermore, 89 twin pairs of uncertain zygosity were able to be classified on the basis of molecular genetic markers. Twins were designated as monozygotic (MZ) if they were the same on five or more genetic markers (error rate = 4/1,000 or less) and as dizygotic (DZ) if they were different on one or more markers. These two methods (i.e., self-report and genetic markers) resulted in a determination of 289 MZ twin pairs and 451 DZ twin pairs. We were unable to determine the zygosity of an additional 43 twin pairs.

Table 1 shows certain demographic characteristics of the sibling-pairs sample compared with the full Add Health sample. The mean age in both samples was approximately 16 years (range = 11–20 years). As can be seen in Table 1, both samples encompassed a wide variety of racial and ethnic groups and covered a broad range of socioeconomic statuses, as indexed by level of maternal education. Complete data were available for 89% of the sibling-pairs sample. However, certain sibling pairs were excluded from the present analyses. Specifically, we excluded the 43 twin pairs (1%) whose zygosity could not be determined. These undecided twin pairs reported that they were DZ but were classified as MZ according to self-reports of confusability of appearance. Combining the undecided twin pairs with the DZ twin pairs would most likely have overestimated the influence of shared environment. Likewise, adding them to the MZ twin pairs would have underestimated the influence of shared environment. Similarly, we excluded an additional 202 unrelated pairs (6%) whose relationship was not exactly a sibling relationship. Specifically, 172 pairs (5%) were identified as cousins residing in the same household. An additional 30 pairs (1%) were identified as aunt/uncle–niece/nephew pairs

Table 1
Demographic Characteristics of the National Longitudinal Study of Adolescent Health Sample

Variable	Full sample (N = 20,745)	Sibling-pairs sample (N = 6,278)
Mean age (in years)	16.1[a]	16.0[a]
Ethnicity (%)		
Caucasian	50.2	49.1
African American	20.8	22.8
Hispanic[b]	8.9	9.0
Asian[c]	2.3	2.0
Filipino	2.6	2.9
Cuban	2.2	1.0
Native American	0.5	0.5
Central–South American	1.4	1.3
Puerto Rican	2.1	1.7
Other	7.2	7.2
Missing	1.7	2.4
Maternal education (%)		
Less than 8th grade	5.4	5.3
Some high school	9.3	9.7
High school graduate equivalent[d]	31.7	31.8
Some college or 2-year degree	17.8	17.4
4-year college graduate	17.5	17.0
Professional training	7.0	5.9
Don't know	4.3	5.8
Missing	7.0	7.6

[a] SD = 1.7 years. [b] Includes Mexican Americans and Chicanos. [c] Includes Chinese, Japanese, Korean, and Vietnamese adolescents. [d] Includes high school graduate, general equivalency diploma, or business or trade school instead of high school.

or as adolescents living together in a group home for delinquent children. We reasoned that these adolescents were less likely to be treated as siblings than, for example, stepsiblings or adoptive siblings living together. Thus, combining them with the unrelated group would most likely have underestimated shared environmental influences. We also excluded 172 unrelated sibling pairs (5%) who reported that they had lived in the same family for less than 3 years. Because we were interested, in part, in examining the genetic and environmental contributions to variation in adolescent self-reports of family environment, we felt that including these pairs would have biased our results in favor of finding lower shared environmental influences. Finally, because there is some evidence that the relative contributions of genetic and environmental factors to the etiology of clinical depression differ from those that contribute to the broader spectrum of depressed mood (Eley, 1997; Rende et al., 1993), we excluded 87 sibling pairs (3%) in which at least one sibling was identified as an outlier (i.e., greater than 3.5 SDs from the mean). These outliers were approximately equally distributed across sibling groups, thus excluding them should not have biased our results. In sum, the data used in all of these analyses were from 2,302 pairs (73% of the pairs sample). Table 2 presents the numbers of sibling pairs in each group, divided by sex type, as well as their mean age, the mean length of time (and proportion of life) they had lived together, and the mean age difference between the siblings.

Procedure

Data for this study were drawn from the Wave 1 home interviews, which were conducted between May 1995 and December 1995. Trained assistants visited the adolescents in their homes to administer the interviews by means of laptop computers. Interviews typically took 1–2 hr. To ensure greater confidentiality and accuracy of responses, we had the adolescents enter their responses on the laptop computers, and for a portion of the

Table 2
Descriptions of Sibling Pairs

Type of pair	Pairs (*n*)	Mean length of time together (in years)	Mean proportion of life lived together[a]	Mean age difference (in years)
		Female–female pairs		
MZ	132	15.4	1.00	0.01[b]
DZ	99	15.3	1.00	0.01[b]
FS	317	14.5	.93	2.27
HS	96	13.6	.90	2.91
UR	47	9.4	.62	1.71
		Male–male pairs		
MZ	131	15.6	1.00	0.02[b]
DZ	121	15.5	1.00	0.01[b]
FS	321	14.6	.93	2.12
HS	96	14.1	.91	2.66
UR	39	9.5	.60	1.54
		Male–female pairs		
DZ	176	15.4	1.00	0.02[b]
FS	474	14.4	.93	2.18
HS	161	13.9	.91	2.70
UR	92	10.8	.70	1.83

Note. MZ = monozygotic twin pairs; DZ = dizygotic twin pairs; FS = full-sibling pairs; HS = half-sibling pairs; UR = unrelated sibling pairs.
[a] Proportion of life lived together was obtained by dividing the length of time siblings had lived together by each sibling's age. These scores were then averaged across siblings. [b] The slight age differences found among the MZ and DZ twin pairs reflect the fact that in a few cases, siblings were interviewed on different days.

interviews, they could hear the questions through headphones. All adolescents received the same interview. Whenever possible, siblings were interviewed on the same day.

Measures

Depressed mood. Adolescent depressed mood was measured with a slightly modified version of the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977; 19 items; α = .87). For example, adolescents were asked to indicate how often during the past 7 days they had felt "depressed," "tired," "[that their] appetite was poor," and "that life was not worth living." Responses ranged from 0 (*never*) to 3 (*most of the time or all of the time*). Items were averaged to create a single scale score, which was reversed (to create a positive correlation between adolescent depressed mood and the two environmental measures) and then standardized within grade level by sex. Prior research has indicated that the CES-D is a valid and reliable measure of depressed mood in adolescents (Radloff, 1991).

Family connectedness. Adolescent family connectedness was assessed with a 7-item scale (α = .83). Items included "People in your family understand you," "Your family pays attention to you," and "Your family has fun together." Additional items tapped into the parent–child relationship (e.g., "Mom cares about me," "Dad cares about me"). For these items, adolescents were given the maximum score from their response to the question concerning either mom or dad. Thus, the parent–child relationship items reflected the degree to which adolescents were close to at least one of their parents. This scale was identical to the family connectedness scale used in Resnick et al.'s (1997) study (T. Beuhring, personal communication, December 17, 1997). The 7 items were averaged to create a single scale score, with higher scores representing greater family connectedness, and then standardized within grade level.

School connectedness. Adolescent school connectedness was assessed with an 8-item scale (α = .77) regarding school connectedness during the current or previous (for those adolescents interviewed during the summer) school year. Items included "Your teachers care about you," "You feel close to people at school," "Teachers treat students fairly," and "You feel safe at school." Items were averaged to create a single scale score, with higher scores representing greater school connectedness, and then standardized within grade level. This scale was identical to the school connectedness scale used in Resnick et al.'s (1997) study (T. Beuhring, personal communication, December 17, 1997).

Coefficient of genetic relatedness. The coefficient of genetic relatedness (R) used in the structural equation modeling represented the average proportion of genes shared by siblings. For MZ twins, R = 1.00; for DZ twins and full siblings, R = .50; for half siblings, R = .25; and for unrelated siblings, R = .00.

Results

Descriptive Statistics

Table 3 presents the means and the standard deviations for each of the three variables, by sex type and sibling group. As can be seen in Table 3, there were some slight differences in mean levels across sibling groups. For example, there was some indication that unrelated siblings reported greater school connectedness than the other groups. Likewise, half siblings and unrelated siblings reported lower family connectedness than other sibling groups. However, neither of these patterns was consistent across both gender and sibling type. More important, the standard deviations were quite similar across the different groups. Thus, the results

Table 3
Means (and Standard Deviations) by Sex Type and Sibling Group

	Male adolescents				Female adolescents			
Sibling group	*n*	Depressed mood	Family connectedness	School connectedness	*n*	Depressed mood	Family connectedness	School connectedness
Same-sex								
MZ twins	262	0.04 (0.99)	0.19 (0.87)	0.11 (0.97)	264	0.16 (0.94)	0.07 (0.96)	0.06 (1.05)
DZ twins	242	0.11 (0.90)	0.10 (0.81)	0.00 (0.92)	198	0.12 (0.87)	−0.04 (0.97)	0.02 (0.93)
Full siblings	642	0.09 (0.93)	0.10 (0.84)	0.16 (0.91)	634	0.06 (0.95)	0.01 (0.94)	0.13 (0.93)
Half siblings	192	−0.02 (0.96)	0.02 (0.84)	0.02 (1.03)	192	0.12 (0.86)	−0.26 (1.02)	−0.03 (0.97)
Unrelated	78	0.01 (0.83)	0.01 (0.86)	0.17 (0.94)	94	0.03 (0.97)	−0.06 (0.91)	0.16 (0.91)
Opposite-sex								
DZ twins	176	−0.03 (0.98)	0.10 (0.87)	−0.07 (0.99)	176	0.19 (0.89)	0.13 (0.90)	0.10 (1.13)
Full siblings	474	0.11 (0.89)	0.08 (0.85)	0.00 (0.97)	474	0.09 (0.90)	−0.00 (0.96)	0.11 (0.92)
Half siblings	161	−0.16 (1.05)	−0.10 (0.97)	−0.13 (1.06)	161	−0.04 (0.92)	−0.29 (1.06)	0.05 (0.95)
Unrelated	92	0.19 (0.85)	0.07 (0.99)	0.21 (0.96)	92	−0.05 (0.99)	−0.29 (1.17)	−0.06 (0.94)

Note. Sample sizes for same-sex pairs are based on double-entered data. Scale scores for depression have been reversed: Higher scores indicate less depression. Depressed mood has been standardized within age and sex. Both family and school connectedness were standardized within grade level. MZ = monozygotic; DZ = dizygotic.

from our structural equation models should not have been biased by differences in mean levels or variances. Table 4 presents the intercorrelations among the three variables, separately by sex. All correlations were significant and similar to the results reported by Resnick et al. (1997), ranging from .27 to .38.

Sibling Correlations

Next, we computed the within-sibling correlations for each of the three variables. Table 5 presents these correlations by sex type and sibling group. As can be seen in Table 5, genetic influences were indicated for each of the three variables and for both sexes because, in general, the within-sibling correlations increased as the level of genetic relatedness increased. However, there was also evidence for significant shared environmental influences. Specifically, the within-sibling correlations for unrelated male–male pairs were significant for all three variables. The correlation for unrelated female–female pairs was also significant for depressed mood. Likewise, correlations for half siblings were more than one half the magnitude of the correlations for full siblings and DZ twins, suggesting that a purely genetic model cannot explain 100% of the familial resemblance for these three variables. Finally, the pattern of correlations suggests that either genetic or shared environmental influences may differ for male and female adolescents because the correlations for male–female sibling pairs were lower than those for the same-sex pairs.

Table 4
Correlations Among Variables

Variable	1	2	3
1. Depressed mood	—	.27***	.35***
2. Family connectedness	.37***	—	.38***
3. School connectedness	.37***	.35***	—

Note. Correlations below the diagonal are for female adolescents; those above the diagonal are for male adolescents. N = 2,285 for female adolescents; N = 2,319 for male adolescents. Scale scores for depressed mood have been reversed so that higher scores represent less depression.
*** $p < .001$.

Structural Equation Modeling

To obtain the best estimates of genetic and environmental influences on variation in depressed mood, family connectedness, and school connectedness and on the covariation among these variables, we analyzed the data by using the structural equation modeling program Mx (Neale, 1997), which is based on maximum-likelihood model fitting. Figure 1 shows the full path model used in the analyses. In this model, the latent variables A_c, C_c, and E_c represent the additive genetic influences, shared environmental influences, and nonshared environmental influences, respectively, that are common to family connectedness, school connectedness, and depressed mood. The latent variables A_s, C_s, and E_s represent the additive genetic influences, shared environmental influences, and nonshared environmental influences, respectively, that are specific to each of the three variables. The coefficient of genetic relatedness represents the average proportion of genes shared among siblings and differed across the different sibling groups (for the coefficients of genetic relatedness for each group, see the *Coefficient of genetic relatedness* section). In contrast, for same-sex siblings, shared environmental influences (both common and specific) were correlated 1.00 across siblings, regardless of their level of genetic relatedness (the shared environment correlation for opposite-sex siblings can be estimated to test whether male and female adolescents living in the same household experience similar environments; see below). Nonshared environmental influences, by definition, were not correlated across siblings.

The variation in each of the three measured variables can be decomposed into variance due to six sources: common genetic, common shared environmental, common nonshared environmental, specific genetic, specific shared environmental, and specific nonshared environmental influences. Genetic, shared environmental, and nonshared environmental influences that contributed to the correlations among the three variables were estimated by using the

Table 5
Sibling Correlations

Type of pair	Pairs (*n*)	Depressed mood	Family connectedness	School connectedness
Female–female				
MZ twins	132	.52***	.57***	.57***
DZ twins	99	.27**	.31**	.25**
Full siblings	317	.26***	.26***	.29***
Half siblings	96	.21*	.35***	.14
Unrelated siblings	47	.30*	.18	.19
Male–male				
MZ twins	131	.40***	.51***	.47***
DZ twins	121	.36***	.28**	.27**
Full siblings	321	.26***	.27***	.26***
Half siblings	96	.21*	.24*	.35***
Unrelated siblings	39	.44**	.39**	.40**
Male–female				
DZ twins	176	.19**	.37***	.29***
Full siblings	474	.14**	.11**	.18***
Half siblings	161	−.04	.10	.02
Unrelated siblings	92	.16	−.10	.10

Note. MZ = monozygotic; DZ = dizygotic.
* $p < .05$. ** $p < .01$. *** $p < .001$.

parameters that connected A_c, C_c, and E_c to the three measured variables (i.e., Parameters *a–i*).

We investigated sex differences in three ways: First, by estimating the coefficient of genetic relatedness among male–female pairs, it is possible to determine whether the same genes influence variation and covariation for male adolescents and for female adolescents.[1] Similarly, estimating the shared environment correlation for male–female pairs determines whether male and female adolescents living in the same household experience the same environment. Finally, sex differences in the magnitudes of genetic and environmental influences can be tested by comparing models that allow Parameters *a–r* to differ for male–male and female–female pairs with models that constrain the parameters to be equal across sex.[2] Boundary constraints were imposed so that all parameters would be nonnegative. All models were fit to variance–covariance matrices.

Table 6 presents the results from a series of hierarchically nested models. Model 1 (the full model) is the model that allowed all parameters to vary across sexes and allowed both the coefficient of genetic relatedness to differ from .50 and the shared environment correlation to differ from 1.00 for male–female pairs. Although the chi-square for this model was significant, this was expected, given the large sample size (Neale, 1997). According to the other two goodness-of-fit indices, however, this model fit the data very well. Specifically, the Akaike's Information Criterion (AIC) was negative, and the value of the root-mean-square error of approximation was less than .05 (Neale, 1997). In the full model, the coefficient of genetic relatedness for male–female pairs was estimated at .50 (95% confidence interval, .24–.50), and the shared environment correlation for male–female pairs was estimated at .36 (95% confidence interval, .02–1.00). The remaining models (Models 2–10) tested for sex differences. Models can be compared using two criteria: the change in chi-square ($\Delta\chi^2$) and the AIC. If the change in chi-square is nonsignificant, then each of the two models being compared fits the data equally well. Thus, the more parsimonious model is retained. Likewise, the AIC is an indicator of both goodness of fit and parsimony (Akaike, 1987; Neale, 1997). In general, a more negative AIC indicates a better fitting, more parsimonious model.

As can be seen in Table 6, neither the model that constrained the coefficient of genetic relatedness parameter to equal .50 in male–female pairs (but allowed the shared environment correlation to vary; Model 2) nor the model that constrained the shared environment correlation in male–female pairs to equal 1.00 (while allowing the coefficient of genetic relatedness parameter to vary; Model 3) resulted in a significantly poorer fitting model: $\Delta\chi^2(1, N = 2{,}302) = 0.00, p < .99$, and $\Delta\chi^2(1, N = 2{,}302) = 3.79, p < .10$, for Models 2 and 3, respectively, indicating that there were not qualitative differences in genetic or environmental influences between male and female adolescents. Moreover, a model that simultaneously constrained the coefficient of genetic relatedness to equal .50 and the shared environment correlation to equal 1.00 (Model 4) did not fit the data significantly more poorly than the full model, $\Delta\chi^2(2, N = 2{,}302) = 3.79, p < .10$. Thus, these constraints were placed on all subsequent models. Model 4 was then used as the comparison model for all subsequent analyses.

In contrast, we found evidence for sex differences in the magnitudes of genetic and environmental influences (i.e., a quantitative sex difference). Specifically, Model 5 tested whether all parame-

[1] If different genes influenced depressed mood, family connectedness, and school connectedness among male and female adolescents, then the coefficient of genetic relatedness between opposite-sex DZ twin pairs and full siblings should differ from .50. Because this coefficient for same-sex half siblings was .25, the estimated coefficient for opposite-sex half siblings was constrained to be one half the estimated coefficient for opposite-sex DZ twins and full siblings.

[2] Adolescents in the male–female pairs were first sorted so that the male adolescent was always Sibling 1 and the female adolescent was always Sibling 2. Then, Sibling 1 was given the parameters used for the male–male sibling pairs, and Sibling 2 was given the parameters used for the female–female sibling pairs.

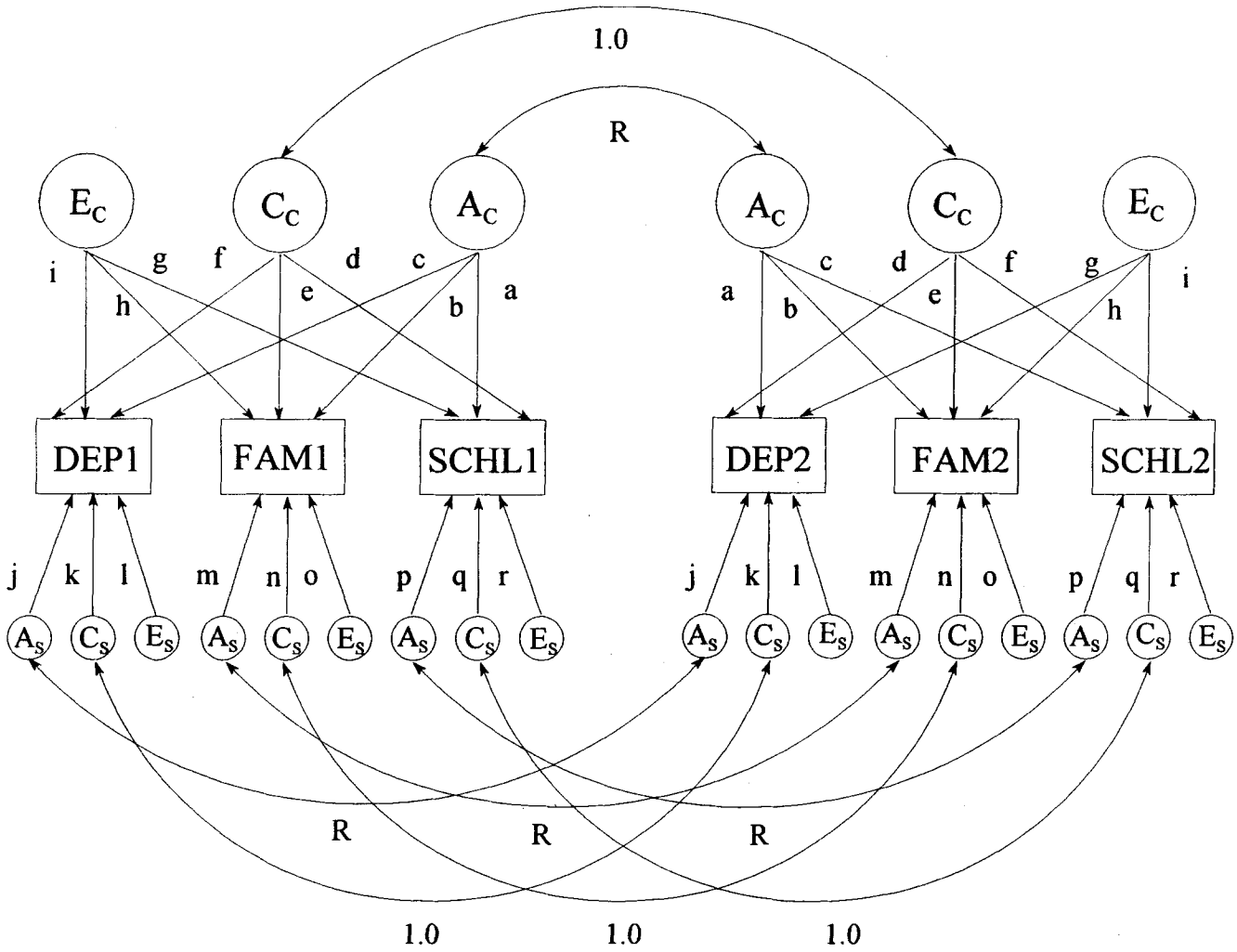

Figure 1. The full path model. Parameters a through r are allowed to vary across male and female adolescents. R = coefficient of genetic relatedness; E_c = common nonshared environmental factor; C_c = common shared environmental factor; A_c = common additive genetic factor; DEP1 = Sibling 1 depressed mood; FAM1 = Sibling 1 family connectedness; SCHL1 = Sibling 1 school connectedness; DEP2 = Sibling 2 depressed mood; FAM2 = Sibling 2 family connectedness; SCHL2 = Sibling 2 school connectedness; A_s = specific additive genetic factors; C_s = specific shared environmental factors; E_s = specific nonshared environmental factors.

ters could be constrained to be equal across sexes. This model resulted in a significantly poorer fit, $\Delta\chi^2(18, N = 2{,}302) = 92.35$, $p < .001$; consequently, at least some of the relative genetic or environmental influences differed for male and female adolescents. The next two models tested whether the parameters common to (Model 6) or specific to (Model 7) the three variables could be constrained to be equal across sexes. Both of these models also resulted in significant changes in chi-squares: $\Delta\chi^2(9, N = 2{,}302) = 30.82$, $p < .001$, for Model 6; $\Delta\chi^2(9, N = 2{,}302) = 29.84$, $p < .001$, for Model 7.

Although the models that constrained parameters to be equal across sexes for all three variables simultaneously did not fit the data as well as the full model, it was possible that the parameters for a single variable could be constrained to be equal across sexes. Therefore, the final three models (Models 8, 9, and 10) tested whether the parameters could be constrained for adolescent depressed mood, family connectedness, and school connectedness, respectively. As can be seen in Table 6, all three models resulted in significant changes in chi-squares: $\Delta\chi^2(6, N = 2{,}302) = 24.70$, $p < .001$, for depressed mood; $\Delta\chi^2(6, N = 2{,}302) = 44.70$, $p < .001$, for family connectedness; and $\Delta\chi^2(6, N = 2{,}302) = 15.00$, $p < .05$, for school connectedness. Thus, the best-fitting model was Model 4, which allowed for quantitative, but not qualitative, differences in the genetic and environmental contributions to the variation in and covariation between depressed mood and family and school connectedness.

Table 7 presents the standardized parameter estimates and their 95% confidence intervals from Model 4. As can be seen in Table 7, the genetic influences on all three variables (both common and spe-

Table 6
Comparison of Model Fits

Model	χ^2	*df*	AIC	RMSEA	$\Delta\ \chi^{2a}$	*df*
1. Full model	362.47***	256	−149.5	.049	—	—
Sex equality						
2. Same genes	362.47***	257	−151.5	.049	0.00	1
3. Same environment	366.26***	257	−147.7	.051	3.79	1
4. Same genes and environment	**366.26*** **	**258**	**−149.7**	**.051**	**3.79**	**2**
5. All parameters equal	458.61***	276	−98.4	.067	92.35***	18
6. Common ACE equal	397.08***	267	−136.9	.054	30.82***	9
7. Specific ACE equal	396.10***	267	−137.9	.057	29.84***	9
8. Depression parameters only	390.96***	264	−137.0	.054	24.70***	6
9. Family parameters only	410.96***	264	−117.0	.061	44.70***	6
10. School parameters only	381.25***	264	−146.7	.053	14.99*	6

Note. The best-fitting model is indicated in boldface. All analyses were based on an *N* of 2,302 sibling pairs. AIC = Akaike's Information Criterion; RMSEA = root-mean-square error of approximation; A = genetic influences; C = shared environmental influences; E = nonshared environmental influences.
[a] Model 1 is used as the comparison model for the change in chi-square for Models 2–4. The change in chi-square for Models 5–10 is based on comparisons with Model 4.
*$p < .05$. ***$p < .001$.

cific) were larger for female than for male adolescents. Conversely, with only one exception, shared environmental influences (both common and specific) were larger for male than for female adolescents. The proportion of variance due to each factor can be obtained by squaring each parameter estimate (e.g., the proportion of variance in depressed mood due to genetic influences that are specific to depression among female adolescents is $.52^2 = .270$, or 27%).

Table 8 contains the proportions of variance that are due to total genetic and environmental influences. Similar to other studies of family environment, total genetic influences (i.e., common plus specific) explained a considerable percentage of variation in adolescent reports of family connectedness, although total genetic influences were stronger among female than among male adolescents (58% vs. 26%). Genetic factors also contributed to variation in school connectedness, albeit somewhat more weakly. Similar sex differences in the heritability of school connectedness were also observed, with genetic influences explaining 45% of the variation in school connectedness for female adolescents, compared with 17% for male adolescents. The largest sex difference appeared for the heritability of depressed mood. Among female adolescents, total genetic influences explained 45% of the variation in depressed mood. In contrast, for male adolescents, genetic influences were negligible (3%).

The reverse pattern was seen for shared environmental influences. Among female adolescents, total shared environmental influences explained none (i.e., 0%) of the variation in family

Table 7
Standardized Parameter Estimates (and Their 95% Confidence Intervals [CI]) From the Best-Fitting Model (Model 4)

	Depressed mood		Family connectedness		School connectedness	
Factor	Female	Male	Female	Male	Female	Male
			Common			
A	.42	.04	.49	.25	.48	.37
95% CI	.29–.54	.00–.25	.37–.66	.00–.55	.34–.62	.07–.57
C	.01	.26	.00	.17	.07	.48
95% CI	.00–.19	.11–.56	.00–.13	.00–.30	.00–.27	.10–.59
E	.49	.45	.34	.47	.33	.44
95% CI	.34–.69	.35–.57	.22–.46	.37–.58	.21–.46	.34–.54
			Specific			
A	.52	.15	.58	.45	.47	.19
95% CI	.29–.63	.00–.45	.39–.67	.05–.60	.11–.62	.00–.44
C	.21	.47	.00	.41	.26	.09
95% CI	.00–.37	.00–.55	.00–.19	.26–.53	.00–.43	.00–.46
E	.52	.69	.55	.57	.61	.62
95% CI	.21–.64	.60–.76	.46–.63	.47–.66	.52–.68	.55–.69

Note. A = genetic factors; C = shared environmental factors; E = nonshared environmental factors.

Table 8
Proportions of Variance and Covariance Explained by Genetic and Environmental Factors

Variable	Genetic		Shared environmental		Nonshared environmental	
	Female	Male	Female	Male	Female	Male
	Proportion of variance[a]					
Depressed mood	.445	.025	.046	.289	.509	.686
95% CI	.27–.57	.00–.21	.00–.14	.17–.36	.42–.62	.58–.76
Family connectedness	.581	.264	.000	.196	.419	.540
95% CI	.48–.66	.07–.42	.00–.04	.08–.32	.34–.51	.45–.65
School connectedness	.451	.174	.072	.240	.477	.585
95% CI	.27–.60	.03–.36	.00–.19	.11–.35	.39–.58	.49–.69
	Proportion of covariance					
Family connectedness–depressed mood	.555	.041	.000	.166	.445	.792
95% CI	.36–.73	.00–.34	.00–.05	.00–.34	.27–.64	.58–.97
School connectedness–depressed mood	.556	.048	.002	.366	.442	.586
95% CI	.30–.74	.00–.37	.00–.11	.14–.53	.26–.65	.39–.75

Note. CI = confidence interval.
[a] Refers to the total genetic and environmental influences (i.e., common + specific).

connectedness and only 7% of the variation in school connectedness. Among male adolescents, total shared environmental influences accounted for 20% of the variation in family connectedness and 24% of the variation in school connectedness. Again, the largest sex difference was found for depressed mood. Shared environmental influences explained less than 5% of the variation among female adolescents but 29% of the variation among male adolescents.

Finally, by using the parameters shown in Table 7, it was possible to determine the proportion of the correlations between environment and depressed mood that were due to genetic, shared environmental, and nonshared environmental influences. These results are also shown in Table 8. Again, substantial sex differences were found. Genetic influences accounted for 56% of the relationship between family connectedness and depressed mood among female adolescents. In contrast, genetic influences explained only 4% of the correlation between family connectedness and depressed mood among male adolescents. Shared environmental influences explained none of the relationship between family connectedness and depressed mood among female adolescents (0%) and 17% of the relationship among male adolescents. Virtually identical results were found for the relationship between school connectedness and depressed mood (although shared environmental influences were somewhat stronger among male adolescents): Genetic factors explained 56% (female adolescents) and 5% (male adolescents) of the correlation, and shared environmental factors accounted for less than 1% (female adolescents) and 37% (male adolescents). Finally, common nonshared environmental influences contributed substantially to the correlations between environment and depressed mood for both sexes. Among female adolescents, nonshared environmental influences accounted for 45% of the relationship between family connectedness and depressed mood and 44% of the relationship between school connectedness and depressed mood. Among male adolescents, these estimates were 79% and 59%, respectively.

Discussion

One of the goals of this study was to examine sex differences in the genetic and environmental influences on variation in adolescent family connectedness, school connectedness, and depressed mood. Similar to other studies of family environments (e.g., Neale et al., 1994; O'Connor et al., 1995; Pike et al., 1996; Rowe, 1981, 1983), we found that genetic factors explained between 26% and 58% of the variance in family connectedness. As predicted, genetic influences on variation in school connectedness were somewhat weaker, although still substantial (17%–45%). Shared environmental factors explained less of the variation in the two measures: between 0% and 20% of the variance in family connectedness and between 7% and 24% of the variance in school connectedness. Overall, these results are consistent with a growing body of literature that has found moderate genetic influences on variation in "environmental" measures and relatively weak shared environmental influences (see Plomin, 1995; Plomin & Bergeman, 1991; Plomin et al., 1985; Rowe, 1994).

However, this study revealed a number of important sex differences. In general, genetic influences were stronger for female adolescents than for male adolescents. This effect was particularly true for variation in adolescent depressed mood. Although the heritability of depressed mood was estimated at .45 for female adolescents, it was significantly lower among male adolescents (.03). This finding is consistent with other studies that have examined sex differences in the relative contributions of genetic and environmental factors to variation in depression (e.g., Tambs et al., 1995), and our heritability estimates for female adolescents fall within the range of estimates that have been reported using only female adolescent or same-sex samples (Kendler et al., 1994; Neale et al., 1994; Reiss et al., 1995; Wierzbicki, 1987). Nonetheless, it is interesting that genetic factors explained a greater proportion of variation in depressed mood for female adolescents than for male adolescents.

Although other researchers have hypothesized that the heritability of depression would be higher for female adolescents and women because of sex-specific genes that are "activated" during puberty (e.g., Weissman & Klerman, 1977), our study does not support this hypothesis. Specifically, the coefficient of genetic relatedness for male–female pairs could be constrained to equal .50, indicating that the same genes influence variation in the three variables for male and female adolescents. What the higher heritability for depressed mood among female adolescents represents, then, is a difference in the magnitude of genetic influences on depressed mood. In genetic terminology, the genes are more penetrant, that is, have more visible phenotypic effects in female adolescents than in male adolescents. This effect might occur if hormonal differences between the sexes lead to greater physiological consequence, or behavioral expression, of the relevant genes in female adolescents than in male adolescents. There is some evidence that hormonal differences, but not pubertal status, predict differences in depression among female adolescents (Brooks-Gunn, 1992).

Interestingly, the total variance in depression did not differ for male adolescents (.88) and female adolescents (.85), $F = 1.04, p > .10$. Thus, the "loss" of genetic effect among male adolescents must be compensated for by an increase in another source of variation: Namely, shared and nonshared environmental influences explained a larger proportion of variance in depression for male adolescents than for female adolescents (29% vs. 5%, respectively, for shared environment; 69% vs. 51%, respectively, for nonshared environment). Accordingly, future research should attempt to identify the environmental influences that may be more strongly related to depressed mood for male adolescents than for female adolescents. Likewise, the identification of specific genes that are associated with depression might reveal why these genes are more penetrant in female adolescents.

We also found sex differences in the genetic and environmental influences on variation in our two environmental measures. Similar to the sex differences for depressed mood, we found that the heritability of both measures was higher for female adolescents than for male adolescents (.58 vs. .26, respectively, for family connectedness; .45 vs. .17, respectively, for school connectedness). This effect makes sense if the more penetrant genes in depression found among female adolescents color their perceptions of their environment, making them perceive their environment more negatively.

In contrast, shared environmental influences were substantially greater among male adolescents than female adolescents (20% vs. 0% for family connectedness; 24% vs. 7% for school connectedness). It is unlikely that brothers receive more similar (i.e., shared) treatments from families and schools than do sisters. In fact, nonshared environmental factors, which include differential treatment, were also slightly greater among male adolescents than among female adolescents (54% vs. 42% for family connectedness; 59% vs. 48% for school connectedness). Our only explanation for this somewhat surprising result is that factors that are truly shared among siblings, regardless of sex (e.g., family structure, socioeconomic status), may be more strongly related to perceptions of family and school environments for male adolescents than for female adolescents.

The second major goal of this study was to determine the relative contribution of genes and environment to the correlations between our three variables. Similar to results obtained from the core Add Health sample (Resnick et al., 1997), depressed mood was significantly correlated with both family and school connectedness ($r = .27$–$.38$). Results from the sibling-pairs sample indicate that for female adolescents, genetic factors accounted for more than one half of the correlations between family connectedness and depressed mood and between school connectedness and depressed mood (approximately 56%). In contrast, for male adolescents, genetic factors explained less than 5% of these correlations. Undoubtedly, the greater genetic influence on the relationship between family connectedness and depressed mood for female adolescents is due, at least in part, to the higher heritabilities of depressed mood and family and school connectedness for female adolescents.

To date, only a handful of studies have examined the genetic influences on the relationship between environment and depressed mood (e.g., Bergeman et al., 1991; Neale et al., 1994; Pike et al., 1996). Although our estimate of genetic mediation of the correlation between perception of family environment and depression for female adolescents is similar to the one reported in the study of female–female adult twins (Neale et al., 1994), it is substantially lower than the 70% reported by Pike et al. for the correlation between maternal negativity and adolescent depression. However, Pike et al. also reported that genetic factors accounted for only 55% of the correlation between paternal negativity and adolescent depression; thus, it is possible that our more global measure of family environment is responsible for the lower estimate found in the present study. Nevertheless, the estimate of genetic mediation for the male adolescents in the present study was significantly smaller than the estimates for either maternal negativity or paternal negativity that Pike et al. reported. Because Pike et al.'s study did not examine sex differences and because Neale et al.'s study was conducted on a sample of female–female twins, we cannot make any direct comparisons at this time. It is intriguing, however, that, similar to the findings of other studies, shared environmental influences did not explain very much of the family environment–depression correlation for female or male adolescents (0% vs. 17%, respectively). The largest environmental influence was found for the common nonshared environmental factors: For female adolescents, these factors explained almost one half of the family environment–depression correlation (45%); for male adolescents, these factors explained more than three quarters of the relationship (79%). This finding suggests that factors that are not shared by siblings, such as differential parental treatment, are the strongest environmental influences on the relationship between family connectedness and depressed mood.

Shared environmental influences on the correlation between school connectedness and depressed mood were more substantial, but only for male adolescents (37% for male adolescents and 0.2% for female adolescents). However, the increase in the shared environmental influence on the relationship between school connectedness and depressed mood among male adolescents comes at the cost of a decrease in nonshared environmental influences. Thus, these results suggest that, for boys, schools may provide more similar treatment than families. Nevertheless, again, nonshared environmental influences on the correlation between school connectedness and depressed mood were substantial for both male and female adolescents (59% and 44%, respectively), indicating that

different experiences in school may be related to differences in depressed mood.

An important implication of the finding that genetic factors accounted for more than one half of the correlations between family and school connectedness and depressed mood among female adolescents is that it calls into question the implicit assumption found in many studies of environmental influences on depression: namely, that differences in adolescent and child outcomes are entirely caused by differences in their rearing environments. What our results suggest is that at least part of the relationship between the environment and depression represents some type of gene → environment correlation. Future studies should try to determine the mechanisms responsible for this correlation. Likely candidates include personality attributes, such as sociability and anxiety, which might influence both depressed mood and the quality of family and school relationships.

Given the evidence for sex differences provided by this study, it would also be useful to uncover the mechanisms underlying the greater genetic influences on variation and covariation among female adolescents. For example, the hypothesis that hormonal changes associated with puberty are responsible for the greater magnitude of genetic influence on variation in depressed mood among female adolescents could be tested by obtaining a sample of female–female pairs who differ in terms of their pubertal status.

Finally, our results concerning the genetic and environmental influences on the relationship between environmental measures and depressed mood do not negate the importance of the environment. Specifically, total environmental influences (both shared and nonshared) accounted for almost one half of the correlations between environment and depression for female adolescents and approximately 95% of the correlations for male adolescents. Similar to other research, however, was our finding that the primary nature of the environmental influences on the relationships between environment and depressed mood were nonshared by siblings in the same family (see Reiss et al., 1994). To the extent that siblings in a family do not experience similar relationships with their environment, such as family connectedness and school connectedness, then these nonshared environmental influences could be responsible for differences in depressed mood. The fact that the MZ twin correlations for both family connectedness and school connectedness were substantially less than 1.00 (approximately .50) suggests that siblings in the same family do experience different environments. Thus, the goal of this research was not to discredit the years of research that have concluded that family and school environments cause some of the differences in adjustment. In fact, data from this study may support such a conclusion. Instead, the goal of this study was to determine whether genetic differences could also account for some of the correlation between environment and adjustment and whether the magnitude of genetic influence differed for male and female adolescents.

Strengths and Limitations

One of the strengths of this study is the data set from which the sample was drawn. Specifically, the Add Health sibling-pairs sample consists of multiple sibling groups, making it easier to detect small but significant shared environmental influences. Moreover, relatively large numbers of both same-sex and opposite-sex pairs are included, making it easier to isolate potential sex differences. The fact that this study did find significant sex differences suggests that studies that include only same-sex siblings may overestimate genetic influences for one sex (i.e., male) and underestimate genetic influences for the other sex (i.e., female). Likewise, if shared environmental influences on depressed mood are more important for male adolescents, these influences will not appear in studies of female–female siblings. An additional strength is that data for this study come from a large, population-based study of adolescents. The sample encompasses a wide variety of ethnic and socioeconomic groups; therefore, results from the Add Health data set should be more generalizable than results from twin and adoption studies that often use more restricted samples.

Nonetheless, the present study does have some limitations. First, it is postulated that the genetic mediation of the correlation between environmental measures and depressed mood among female adolescents represents a gene → environment correlation. However, it should be emphasized that both perceptions of environment and depressed mood were ascertained vis-à-vis adolescent self-report. It is possible that adolescent depressed mood actually influences perceptions of the environment. Specifically, depressed adolescents may be more likely to perceive their family and school environments as less positive. Thus, observational measures of the environment should also be used. Nevertheless, other studies have examined the genetic influences on objective measures of parenting (e.g., Lytton, 1977; O'Connor et al., 1995), and results from these studies also confirm substantial genetic influences on objective measures of parenting.

Relatedly, as with all cross-sectional research, this study cannot disentangle cause and effect. It is unclear whether lower family and school connectedness leads to greater depressed mood, whether greater depressed mood leads to lower feelings of connectedness, or whether other factors (including genetic factors) lead to perceptions of family and school environment and depressed mood simultaneously. Longitudinal data are needed to definitively answer this question.

This study also suggests that there are significant sex differences; however, it should be noted that many of the confidence intervals for male and female adolescents overlap considerably. This is particularly true of the parameters related to school connectedness. Moreover, the model that constrained the school parameters to be equal was almost as parsimonious as the model that allowed the parameters to vary, $\Delta \chi^2(6, N = 2{,}302) = 15.00, p < .05$. Thus, it is especially important that these sex differences be replicated because they may reflect sampling variation. However, the confidence intervals surrounding the common genetic influence on depressed mood did not overlap for male and female adolescents, indicating that genetic influences on depressed mood do differ substantially among male and female adolescents. Furthermore, other studies (e.g., Tambs et al., 1995) have found that the heritability of depressed mood is higher among women than men, lending support to our results. Because the genetic influences on the covariation between two variables are limited by the genetic influences on the variations of the individual variables, it is not surprising that genetic factors contribute more strongly to the relationship between environment and depressed mood among female adolescents. Moreover, neither the confidence intervals surrounding the total genetic influences nor the total shared envi-

ronmental influences on depressed mood and family connectedness (but not school connectedness) overlapped, suggesting that the sex differences in this sample are real. Finally, the finding that virtually all of the sibling correlations for male–female pairs were substantially lower than those for their same-sex counterparts also implies that the etiology of these three variables differs for male and female adolescents.

Finally, as with most behavioral genetic studies, this study operated under a number of assumptions. One assumption is the equal-environment assumption, which states that twins do not experience more similar environments than nontwins. Although this assumption has been validated in studies of personality and psychopathology (Hettema, Neale, & Kendler, 1995), it has not been examined in studies of perceptions of parenting. For example, the proportion of time spent living in the same household also increases with the degree of genetic relatedness (see Table 2). Thus, it is possible that the greater resemblance among siblings who are more genetically related is simply an artifact of the greater proportion of time they have lived together. However, data from the present study suggest that this is not the case. Specifically, except among the opposite-sex pairs, the full-sibling correlations were similar in magnitude to those for DZ twins, even though full siblings had lived a smaller proportion of their lives together than DZ twins. Likewise, proportion of life together cannot explain the substantial difference in correlations between MZ and DZ twins, because both types of twins have lived together 100% of their lives. Moreover, the parameter estimates obtained in this study are from an analysis of all sibling groups, not just twins or nontwins; thus, potential violations of the equal-environment assumption are less likely to bias our results.

A second assumption is that of no assortative mating among spouses and no selective placement among adoptive families. Both selective placement and assortative mating would increase estimates of shared environment and might account for the significant sibling correlations that were found for the half siblings and the unrelated siblings. However, violations of this assumption would not explain why the full-sibling and DZ twin correlations were more than one half the MZ twin correlations; nor would they explain why shared environmental influences were more important among male adolescents. Nonetheless, this assumption should be tested in future research. Finally, this study assumed that there are no gene by environment interactions. Yet, Bronfenbrenner and Ceci (1994) hypothesized that the relationship between proximal processes (e.g., family and school connectedness) and adjustment would be moderated by environmental context and that heritabilities may differ among adolescents in different ecological niches. Although they are beyond the scope of the present article, future research should examine these kinds of questions by using behavioral genetic designs within an ecological framework.

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