

Co-occurrence of Depressive Symptoms and Antisocial Behavior in Adolescence: A Common Genetic Liability

Thomas G. O'Connor
Institute of Psychiatry

Shirley McGuire
University of California, San Diego

David Reiss
George Washington University Medical Center

E. Mavis Hetherington
University of Virginia

Robert Plomin
Institute of Psychiatry

Recent reviews of research on child and adolescent psychopathology have highlighted the consistently high rates of co-occurring dimensions of psychopathology, particularly between internalizing and externalizing disorders, and have suggested that further research examining the causes of co-occurring syndromes is needed. The authors examined this question in a national sample of 720 same-sex adolescent siblings between 10 and 18 years of age consisting of monozygotic and dizygotic twins, full siblings, half siblings, and unrelated siblings. Composite measures of adolescent and parent reports and observational measures of depressive symptoms and antisocial behavior were subjected to behavioral genetic models that examine the genetic and environmental influences on individual differences in each dimension as well as in the co-occurrence between dimensions. Results indicated that approximately half of the variability in depressive symptoms and antisocial behavior is attributed to genetic factors; shared and nonshared environmental influences were also significant. The co-occurrence of depressive and antisocial symptoms was explained by genetic and shared and nonshared environmental influences. Specifically, approximately 45% of the observed covariation between depressive and antisocial symptoms could be explained by a common genetic liability. Results are interpreted in light of contribution of genetic studies to debates on child and adolescent psychopathology.

Developmental and epidemiological studies from the past decade consistently report that clinically significant rates of psychopathology first appear in adolescence (Costello et al., 1988; Feehan, McGee, & Williams, 1993; Fergusson, Horwood, & Lynskey, 1993; Kashani et al., 1987; Rutter, 1989; Whitaker et al., 1990). These findings have stimulated numerous research efforts to examine the familial, extrafamilial, hormonal, and genetic risk factors for adolescent psychiatric disorders and more broad-band measures of maladjustment (Cicchetti & Toth, 1992a; McCauley et al., 1993; Puig-Antich, 1982). The data

provided by these reports have influenced developmental theories of psychopathology and have suggested important guidelines for developing primary, secondary, and tertiary intervention efforts for at-risk children and adolescents (Cicchetti & Toth, 1992b; Rolf, Masten, Cicchetti, Nuechterlein, & Weintraub, 1992).

More recently, the focus of empirical studies and literature reviews on child and adolescent psychopathology has moved beyond documenting the prevalence of, and risk factors for, individual disorders (for these data are now reasonably well-accepted) and has instead emphasized the need for further study of the patterns and associated features of co-occurring disorders (Angold & Costello, 1993; Caron & Rutter, 1991; Compas & Hammen, 1992; Verhulst & van der Ende, 1993). The recent focus on patterns of co-occurring disorders or correlated symptom clusters stems from two important findings. First, higher than expected rates of comorbidity or covariation among different dimensions of psychopathology have been repeatedly found in both clinical and epidemiological samples of adolescents from the United States (Kashani, Orvaschel, Rosenberg, & Reid, 1989) and abroad (Fergusson et al., 1993; Verhulst & van der Ende, 1993). Indeed, significantly correlated dimensions of psychopathology in adolescence appear to be the rule and not the exception. Second, available data suggest that individuals who meet diagnostic criteria for only one disorder or who exhibit

Thomas G. O'Connor and Robert Plomin, Social, Genetic and Developmental Psychiatry Research Centre, Institute of Psychiatry, London, England; Shirley McGuire, Department of Psychology, University of California, San Diego; David Reiss, Center for Family Research, George Washington University Medical Center; E. Mavis Hetherington, Department of Psychology, University of Virginia.

This article is based on the project titled "Nonshared Environment and Adolescent Development." The project is supported by the National Institute of Mental Health (1R01MH43373) and the William T. Grant Foundation.

Correspondence concerning this article should be addressed to Thomas G. O'Connor, Social, Genetic and Developmental Psychiatry Research Center, Institute of Psychiatry, 113 Denmark Hill, London SE5 8AF, England. Electronic mail may be sent to spjwtoe@iop.bpmf.ac.uk.

elevated levels of symptoms in only one dimension of maladjustment appear to have different (e.g., less severe) prognoses, treatment responses, and environmental and biological risk factors compared with adolescents who have multiple problems (Angold & Costello, 1993; Capaldi, 1992; Dadds, Sanders, Morrison, & Rebgetz, 1992; Harrington, Fudge, Rutter, Pickles, & Hill, 1991; Puig-Antich et al., 1989). Thus, the importance of previous research on individuals exhibiting only one disorder may be questioned to the extent that the etiology, prognosis, and maintaining factors of a single disorder are qualitatively different in the presence of co-occurring disorders (Caron & Rutter, 1991).

Perhaps because of its high prevalence rate in adolescence, depression has received significant attention in recent reviews of child psychiatric comorbidity (Angold & Costello, 1993; Compas & Hammen, 1992). In particular, the correlation between depressive symptoms and antisocial behavior, or more generally internalizing and externalizing behaviors, has garnered attention because of the high rates of symptom co-occurrence and because adolescents who exhibit both forms of maladjustment appear to be distinguishable from individuals who exhibit only depressive or antisocial symptoms (Capaldi, 1992; Harrington, Fudge, Rutter, Pickles, & Hill, 1990; Kovacs, Paulauskas, Gatsonis, & Richards, 1988; Puig-Antich et al., 1989; Zoccolillo, 1992).

Several explanations have been proposed to explain the high rates of co-occurring depressive symptoms and antisocial behaviors in adolescence. First, higher than expected rates of depression and antisocial behavior may be related to diagnostic overlap or inaccuracies, in which the same symptom (e.g., irritability) may be part of both dimensions. Second, antisocial behavior may be a risk factor for depression (Capaldi, 1992) or vice versa (Geller, Chestnut, Miller, Price, & Yates, 1985; Kovacs et al., 1988; Puig-Antich, 1982). Third, the same risk factor or co-occurring risk factors (e.g., maternal depression and parental hostility) may make adolescents vulnerable to both forms of maladjustment (Caron & Rutter, 1991; Downey & Coyne, 1990).

One hypothesis that has received little empirical attention proposes that depressive symptoms and antisocial behavior may share a genetic diathesis. That is, depression and antisocial behaviors may co-occur because a common genetic liability increases the vulnerability to both dimensions. A common genetic diathesis underlying two (or more) syndromes is possible if each syndrome is genetically influenced and the syndromes are correlated. Interestingly, despite evidence supporting a genetic influence on child and adolescent depression (Puig-Antich et al., 1989; Rutter et al., 1990; Silberg et al., 1994; Weissman, Warner, Wickramaratne, & Prusoff, 1988) and antisocial behavior or conduct problems (Cadoret & Cain, 1980; Eaves et al., 1993; Gottesman & Goldsmith, 1994; Plomin, Nitz, & Rowe, 1990; Rowe, 1983; Rutter et al., 1990; Silberg et al., 1994), the hypothesis that these dimensions may co-occur because of a common genetic diathesis has not been directly examined.

To date, the method used most often to test whether a genetic diathesis is common to two or more disorders is the family history method. With the family history method, a genetic liability to co-occurring syndromes is implied if depressed persons have a higher than normal rate of first degree relatives with

conduct problems, antisocial behavior, or related disruptive behavior disorders and vice versa. In one of the only family history studies to examine a genetic link between depression and antisocial behavior, Puig-Antich and his colleagues (Puig-Antich et al., 1989) reported that first degree relatives of prepubertal depressed children had higher rates of alcoholism, but were no more likely to be diagnosed with antisocial personality than were first degree relatives of psychiatric controls or nonclinic children. These null findings should be interpreted with caution because of the relatively small sample size and because family history methods confound genetic influences with familial (shared) environmental influences.

A better method for examining whether there is a genetic risk for two (or more) syndromes is to examine family members (e.g., siblings) who vary in genetic relatedness and assess whether the correlation between, for example, depressive symptoms in sibling A and antisocial behavior in sibling B (i.e., the cross-correlation) decreases with decreasing genetic relatedness (Kendler, Heath, Neale, Kessler, & Eaves, 1993). This is the standard quantitative genetic multivariate analysis that decomposes the covariance between traits rather than the variance of each trait by itself. Although multivariate genetic analyses of the co-occurrence of depressive symptoms and antisocial behavior in adolescence are unavailable, this method has been used in a series of studies by Kendler and his colleagues on a large sample of adult female twins. They report that a significant genetic correlation exists between depression and generalized anxiety disorder (Kendler, Neale, Kessler, Heath, & Eaves, 1992), alcoholism (Kendler, Heath, et al., 1993), and even smoking (Kendler, Neale, et al., 1993). Interestingly, in the case of major depression and generalized anxiety disorder, Kendler et al.'s findings suggest that the genes that make an individual vulnerable to major depression are the same genes underlying a vulnerability to generalized anxiety disorder. In other words, the co-occurrence of depression and anxiety is genetic in origin, whereas the lack of a co-occurrence is environmentally mediated.

An additional benefit of the twin design or, as in this study, a twin-sibling design, is that it examines two environmental sources of symptom covariation, shared and nonshared environment, in addition to genetic influence. Shared environmental experiences are common to both siblings and therefore tend to make them similar. If shared environment were important for two traits, it could also account for covariation between the traits. For example, siblings who are exposed to equally elevated levels of parental depression or hostility, or both, may, as a consequence, display both depressive symptoms and antisocial behavior (Downey & Coyne, 1990).

Nonshared environmental experiences are those experiences that siblings do not share and function to make them dissimilar. A surprising but consistent finding from genetic research during the past decade is that environmental influences that affect personality and psychopathology are largely of the nonshared variety, with the exception of antisocial behavior in adolescence (Plomin, 1994; Rowe, Rodgers, & Meseck-Bushey, 1992). Nonshared environmental experiences may also explain covariation between dimensions of maladjustment if, for example, the unique experiences of one sibling lead to his or her development of both depression and antisocial behavior. In this case, the

affected adolescent's sibling who is protected from similar environmental stressors does not develop depressive or antisocial symptoms.

Research findings consistently document gender differences in the prevalence of depressive and antisocial symptoms beginning in adolescence and continuing through adulthood. Females display higher rates of depressive symptoms and disorders than males whereas the reverse is true for conduct problems and antisocial behavior (Angold & Rutter, 1992; Robins & Regier, 1991; Weissmann et al., 1988). Such mean differences, however, do not speak to the issue of genetic and environmental components of variance. Gender differences in the genetic and environmental components of depressive and antisocial symptoms are relatively unexamined and available data are mixed. Some preliminary findings suggest that genetic risk may be greater for females whereas shared environmental risks may be greater for males (Cadoret & Cain, 1980; Rowe et al., 1992; Rutter et al., 1990), although other studies have found the reverse (Silberg et al., 1994). Gender differences in the genetic-environmental components of the covariation of depressive symptoms and antisocial behaviors has received even less attention.

This is the first bivariate behavioral genetic study of the co-occurrence of depressive and antisocial symptoms in adolescence. On the basis of existing research on adults, we hypothesized that the covariation between depression and antisocial behaviors would be largely due to a common genetic liability. Shared environmental risk factors that predisposed siblings to both depression and antisocial behavior were also hypothesized. Finally, given the research findings on the importance of non-shared environment, we hypothesized that the co-occurrence of depression and antisocial behavior would be influenced, in part, by nonshared environmental risks.

Method

The data in this study are part of the Nonshared Environment and Adolescent Development (NEAD) project. The goals of the NEAD study are to identify sources of nonshared environment, to estimate its impact on social and personality development in adolescence, and to integrate genetic and environmental models of adolescent development (Reiss et al., 1994).

Sample

Two target same-sex adolescent siblings from 720 families from 47 states were included. The average ages of the target children were 14.8 years and 12.6 years for the older and younger adolescents, respectively; siblings were no more than 4 years apart in age. Nondivorced families consisted of 93 monozygotic (MZ) twin pairs, 99 dizygotic (DZ) twin pairs, and 95 full sibling pairs. Remarried families included 182 full sibling pairs, 109 half-sibling pairs, and 130 unrelated sibling pairs. Thus, the six groups of siblings differ in genetic relatedness from 100% (MZ twins) to 50% (DZ and full siblings) to 25% (half siblings) and 0% (unrelated siblings). All remarried families were together for a minimum of 5 years.

Families in this study were drawn from several sources. The nondivorced families with nontwin siblings and a subset of the stepfamilies were recruited from random digit dialing. However, because random digit dialing procedures would be prohibitively expensive to identify the rare sibling and family types, the remainder of the families were identified from two large national market surveys of over 675,000 families

that spanned a wide range of geographic and income characteristics (Reiss et al., 1994).

The sample is generally middle class but contains a wide range of educational and income levels. Fathers averaged 14 years of education and mothers averaged 13.6 years. Thirty-five percent of the fathers and 41% of the mothers had a high school education or less; 31% of the fathers and 22% of the mothers completed college. The average family income was \$25,000 to \$35,000. Ninety-three percent of the fathers and 94% of the mothers were Caucasian. The percentage of Caucasians in the sample is above national norms but is expected given the large number of long-remarried stepfamilies that the study design required. The average age of mothers was 38.1 years, and for fathers, 41 years.

Procedure

Families were visited in their homes by trained interviewers on two occasions about 2 weeks apart; family members also completed a set of measures between visits. Each visit lasted approximately 2 hr. Mothers, fathers, and the two adolescent siblings completed an extensive battery of questionnaires during the home visits. In addition, during the visits the four dyadic parent-child interactions were videotaped. Each dyad was given two issues to discuss and resolve; topics were previously identified as areas of continuing difficulty in their relationship.

Measures

Twin zygosity was determined by using parent and interviewer reports on a questionnaire (Nichols & Bilbro, 1966). When compared with blood tests of single gene markers, this questionnaire correctly identifies greater than 90% of twins. Consistent with other studies, 6% of the twins could not be definitively diagnosed in this study; these twin pairs are excluded from all analyses.

As detailed elsewhere (Reiss et al., 1995), this study used a dimensional model of psychopathology based on symptom checklists rather than a categorical "affected-unaffected" distinction. A dimensional model was adopted for five reasons. First, symptom checklists have a long history in clinical, developmental, and epidemiological research (e.g., Kandel & Davies, 1982). Second, available findings indicate that symptom checklists with children and adolescents tend to replicate research using diagnostic classifications with respect to concurrent and longitudinal validity (e.g., Kandel & Davies, 1986; Stiffman, Cheuh, & Earls, 1992) and rates of co-occurring symptoms (Fergusson et al., 1993; Verhulst & van der Ende, 1993). Symptom checklists also discriminate clinic from nonclinic populations (e.g., Achenbach & Edelbrock, 1981). Third, a sample size considerably larger than 720 families would be required to collect a reasonable sample of clinically diagnosed individuals in each of the six groups in a normal risk population (cf. Fergusson et al., 1993). Fourth, available data suggest that the genetic-environment pattern of influence is similar for clinical diagnoses and dimensional models of psychopathology (Livesley, Jang, Jackson, & Vernon, 1993; Nigg & Goldsmith, 1994). Fifth, it would have been prohibitively expensive to train interviewers and collect diagnostic data on both adolescents and obtain similar reports from parents.

This study focused on the two dimensions of psychopathology that were indexed in the current study by multiple measures and multiple raters. Depressive and antisocial symptoms were measured with symptom checklists and an observational coding system, all of which have been widely used in other studies of adolescence and demonstrate very good validity and reliability. The symptoms assessed in the checklists are comparable with other symptom lists reported in previous articles (Kandel & Davies, 1986). In the present report, we sought to minimize rater bias by compositing across reporters and across different measures. Thus, although these findings may not directly translate to research using diagnoses, the present strategy is analogous to research using diagnostic

classifications in which adolescent self-report and parental reports are combined.

The Child Depression Inventory (CDI; Kovacs, 1981) is a widely used 27-item inventory of depressive symptoms (e.g., depressed affect, poor appetite, poor sleep). The CDI has been previously reported to demonstrate good reliability, validity, and correspondence with clinical diagnoses (Kovacs, 1981). Adolescents completed the CDI; mothers and fathers completed the parent version of the CDI.

The Behavior Problem Index (BPI; Zill, 1985) is a 32-item questionnaire that assesses the frequency of specific behaviors that discriminate clinic from nonclinic persons; items on the BPI were adapted from the widely used Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1981). The BPI has been used in a national sample of over 15,000 children age 4 to 17 years (Zill, 1985). The BPI has been used in previous research and demonstrates satisfactory reliability and validity (Zill, 1988). The two 6-item Depression/Internalizing (e.g., sudden changes in mood; felt sad or depressed) and Antisocial (e.g., got into trouble at school, bully/mean to others) factors were included in these analyses. Mother, father, and adolescent self-reports were included.

The Behavior Events Inventory (BEI; Hetherington & Clingempeel, 1992) was originally developed by Patterson (1982) and modified by Hetherington and Clingempeel (1992). This is a 32-item true/false inventory of common behavioral problems and prosocial behavior that the adolescent engaged in during the preceding 24 hr. To increase the reliability, we administered the BEI twice and summed the scores from the two assessments. Mother, father, and adolescent self-reports of the 5-item Depression factor (e.g., felt withdrawn, depressed, lonely) and 7-item Antisocial/Coercive factor (e.g., was verbally aggressive, lied, or cheated) were included.

In addition to the questionnaire assessments, observational ratings of depressed mood and antisocial behavior were made by trained raters using a previously validated coding system (Hetherington & Clingempeel, 1992). Following the completion of a 10-min dyadic videotaped interaction, coders rated each dimension on a 5-point Likert scale. A total observational score of depressed mood and antisocial behavior was averaged across the adolescent's behavior in three dyadic interactions (i.e., with mother, father, and sibling). Coder agreement (intraclass correlation) for the depressed mood scale was .70 and for antisocial behavior, .86.

A composite score for depressive symptoms was created to compensate for the well-known rater differences in depressive symptoms reported in previous research (Andrews, Garrison, Jackson, Addy, & McKnown, 1993; Angold, Weissman, John, Wickramaratne, & Prusoff, 1991). All measures were standardized before compositing. The depressive composite included mother, father, and self-reports of the total score from the CDI, the depression factor from both the BPI and BEI, and observers' reports of depressed mood based on the observational data. The internal consistency (Cronbach's alpha) for this composite was .86 for the older adolescent and .84 for the younger adolescent. The CDI contains three oppositional/antisocial items. These items did not inflate the correlation between dimensions, however, because there was no difference in the correlation between the composites when these items were removed from the CDI scales. Accordingly, the full CDI is included in the depression composite.

The antisocial composite consisted of mother, father, and self-report of the antisocial factor from the BPI and BEI and observers' reports of the adolescent's observed antisocial behavior. The internal consistency of this seven-item composite is .85 for the older adolescent and .85 for the younger adolescent.

Data Analysis

A twin-sibling design was used in this study to decompose observed (phenotypic) variance into genetic and environmental components

(Plomin, DeFries, & McClearn, 1990). Genetic influence is implied if the following pattern exists among the correlations: MZ twins > DZ twins and full siblings > half siblings > unrelated siblings. Shared environment is suggested if the correlations among the different sibling pairs are significant and similar and is most clearly observed in the correlation between unrelated siblings. Nonshared environmental influence is suggested if the correlations among sibling pairs are small and nonsignificant and is most clearly observed as the dissimilarity of MZ twins. An advantage of the twin-sibling design is that the derived estimates are not limited to twins, but are based on an array of sibling constellations. Estimates of genetic and environmental influences were obtained using model-fitting procedures using LISREL VII, 2nd edition (Joreskog & Sorbom, 1993) and Mx (Neale, 1994). In this design, there was adequate (.79) power to detect a moderate genetic effect ($h^2 = 32\%$) assuming, for example, a 10% shared environmental effect.

The modeling procedures for the univariate and bivariate models are explained in detail elsewhere (Neale & Cardon, 1992) and are therefore only briefly mentioned here. The aim of univariate and bivariate behavioral genetic analyses is to decompose sibling similarity in a dimension (univariate) or in the covariation between dimensions (bivariate) into three components: (a) additive genetic (G_a), (b) shared environment (E_s), and (c) nonshared environment (E_n). In the univariate case, sibling similarity is decomposed into similarity due to genetics and similarity due to shared environmental experiences. Sibling dissimilarity is attributed to nonshared environmental experiences that serve to make siblings dissimilar. The bivariate model is an extension of the univariate model in that the covariance between two dimensions is decomposed into similarity due to genes predisposing to both dimensions, shared environmental experiences that create sibling similarity in both dimensions, and nonshared environmental experiences that make only one sibling vulnerable to both dimensions of psychopathology.

In these analyses, we examined only additive genetic variance and not nonadditive (dominant) genetic variance because we had little power to discriminate these two genetic influences. Moreover, the estimates obtained with only additive genetic variance estimated were essentially identical to the models in which both additive and nonadditive genetic variance were estimated.

On the basis of quantitative genetic theory, MZ twins are genetically identical and are therefore correlated 1.0 for additive genetic variance. Full siblings and DZ twins are correlated .5, half siblings are correlated .25, and genetically unrelated siblings are correlated 0 (Plomin et al., 1990). By definition, shared environment is correlated 1.0 for siblings who live in the same house. Also by definition, the nonshared environment correlation for all siblings pairs is 0 because it represents the influences that are unique to each sibling (plus measurement error).

Several methodological conditions should be noted. First, consistent with most studies, age, sex (except when testing for gender differences—see below), and age differences were partialled from the covariances prior to genetic modeling because these variables artificially inflate sibling similarity, especially when twins and nontwins are examined. Second, all behavior genetic models analyzed intraclass covariances based on double-entered data, a widely used method in behavioral genetic research (e.g., Neale & Cardon, 1992). Covariances were analyzed because they are sensitive to, and allow tests of, variance differences among groups.

Both univariate and bivariate behavioral genetic models make certain assumptions about the nature of the data and the processes being estimated. Specifically, the models assume no gene-environment interaction, assortative mating, or epistasis. A more detailed discussion of the validity of these assumptions can be found in Loehlin (1992). In the current study, it was possible to assess possible assortative mating effects for depressive symptoms based on parent reports on the Center for Epidemiological Studies Depression Scale (see Reiss et al., 1994). Across the sibling groups, the correlation for mothers' and fathers' self-report of depression was minimal ($r = .16$).

Results

Univariate Analyses

The means and variances for the composite measures of depression and antisocial behavior for each sibling group are displayed in Table 1. Mean differences were observed for depressive symptoms, as the mean level of MZ twins was lower than that found in full siblings in nondivorced families and all sibling types in stepfamilies. Additionally, the level of depressive symptoms in the DZ group was lower than that observed in the unrelated sibling group. The mean level of antisocial symptoms in the MZ group was significantly lower than all siblings in remarried families, and the mean level of antisocial symptoms in the DZ group was significantly lower than full and half siblings in stepfamilies. There was also a tendency for the variances in the twin groups to be smaller than the other sibling groups. The implications of these findings for the central behavioral genetic analyses are discussed later.

The twin-sibling correlations for depressive symptoms and antisocial behavior by sibling type are displayed in Table 1. For the antisocial composite measure, the pattern of twin and sibling correlations corresponds well to the pattern indicative of genetic influence. The greater DZ correlation as compared with the full sibling correlation suggests the possibility of shared environmental influences specific to twins. For depression, the pattern of twin and sibling correlations is not so clear. The comparison of MZ and DZ twin correlations suggests substantial genetic influence; however, the correlations for the other sibling groups are similar, suggesting minimal genetic influence. Nonetheless, the best estimate for genetic influence for these data comes from the model-fitting that analyzes all of the data simultaneously. The cross-sibling correlations are discussed later in relation to bivariate analyses.

The results of the univariate behavior genetic model-fitting analyses for depressive symptoms and antisocial behavior are displayed in Table 2. In the first model for both depressive and antisocial symptoms, Ga, Es, and En were estimated and the variances across sibling groups were constrained to be equal.

The parameters in this model produced a poor fit to the data for both dimensions, $\chi^2(9, N = 708) = 51, p < .01$ for depressive symptoms; $\chi^2(9, N = 708) = 37, p < .01$ for antisocial behavior.

Significant variance and mean differences were observed among the different sibling types on both dimensions; less variability and lower mean scores were observed among MZ and DZ twins compared with siblings in remarried families (Table 1). These differences exist despite using the same protocol to recruit twins and siblings in remarried families. Although groups of individuals who experience more stress—particularly stress associated with a marital transition—are predicted to be more variable and less well-adjusted on average than those persons who do not experience high levels of stress (Hetherington, 1989), for the present purposes these differences create a poor fit to the data.

As an alternative to Model 1, a second model was run in which variance differences were included. In this alternative model, the variance in the MZ twins was fixed to 1 and the variances in the other five groups were allowed to vary. In this model the observed variances are scaled with respect to the MZ twins. Testing the difference in fit of the two models can be calculated by comparing the difference in chi-square values with the difference in the degrees of freedom of the two models. Table 2 shows that the second model that freed the variances among non-MZ sibling groups improved the fit of the model significantly. The nonsignificant chi-square value of Model 2 further suggests that the parameters provide a good fit to the data. More important, the values obtained with the variances freed and constrained across groups are similar for depression and essentially identical for antisocial symptoms, suggesting that variance differences do not alter the substantive conclusions that can be made from the estimates. To examine further possible selection biases that may influence the obtained parameters, we included the means for the groups in a third model, that is, models were run using the moment matrices. When the means were included in the models and allowed to vary there was essentially no observed change in the parameter values (there

Table 1
Means (With Standard Deviations) and Correlations for Outcome Composites by Sibling Type

Measure	Sibling type					
	MZ (93)	DZ (99)	FI (95)	FS (182)	HS (109)	UN (130)
Means						
Depression	-.26 (.45) _a	-.08 (.49) _{a,b}	.02 (.57) _{b,c}	.06 (.54) _{b,c}	.08 (.64) _{b,c}	.12 (.64) _c
Antisocial	-.21 (.53) _a	-.13 (.51) _{a,b}	-.06 (.53) _{a,b}	.12 (.63) _c	.15 (.66) _c	.01 (.62) _{b,c}
Within-sibling correlation						
Depression	.66	.19	.20	.20	.28	.21
Antisocial	.81	.68	.49	.46	.38	.27
Cross-sibling, cross-dimension correlation	.35	.11	.28	.23	.22	.23
Within-sibling, cross-dimension correlation	.45	.23	.48	.50	.58	.50

Note. Means and standard deviations for composites are reported; correlations given are those for the double-entered data used in the models (see text). MZ = monozygotic twins; DZ = dizygotic twins; FI = full siblings in nondivorced families; FS = full siblings in stepfamilies; HS = half siblings in stepfamilies, UN = unrelated siblings in stepfamilies. Significant mean difference contrasts are reported at the $p < .05$ level; sibling groups without common subscripts are significantly different. Overall correlation between depression and antisocial behaviors $r = .47$. The number of sibling pairs is given in parentheses below each sibling type.

This document is copyrighted by the American Psychological Association or one of its allied publishers. This article is intended solely for the personal use of the individual user and is not to be disseminated broadly.

Table 2
Model-Fitting Results of Univariate Behavior Genetic Model

Variable	h^2	Es^2	En^2	Ga	Es	En	χ^2	df
Depression								
Variances fixed	.70*	.03	.29*	.84 (.05)	.19 (.16)	.54 (.04)	51*	9
Variances free	.48*	.07*	.45*	.53 (.08)	.21 (.09)	.51 (.04)	12	4
Antisocial								
Variances fixed	.63*	.22*	.16*	.80 (.04)	.47 (.06)	.39 (.03)	37*	9
Variances free	.56*	.25*	.19*	.66 (.07)	.45 (.05)	.39 (.03)	7	4

Note. Univariate behavioral genetic models were run with variance differences among sibling groups free and fixed. In both cases, the model that allowed variances to differ across groups fit better. For depression the increase in fit: $\chi^2(5, N = 708) = 39, p < .01$. For antisocial behavior the increase in fit: $\chi^2(5, N = 708) = 30, p < .01$. Models that included the means were also examined (i.e., based on the moment matrix). However, for neither depression nor antisocial behavior did freeing or fixing the means across sibling groups make any difference in the fit of the model. Unstandardized estimates (SDs in parentheses) are given for genetic (Ga), shared environment (Es), and nonshared environment (En); the corresponding variance components are h^2 , Es^2 , and En^2 , respectively.

* $p < .05$.

was no more than a 1-point change in the variance estimates). The results from these alternative models indicate that the unequal variances and means across sibling groups in depressive and antisocial symptoms do not influence the genetic-environmental pattern.

The significance of the genetic and environmental parameters for both depressive symptoms and antisocial behavior was examined by comparing the difference in chi-square values for the model that included the parameter with an alternative model that set the parameter equal to 0 (Neale & Cardon, 1992). Table 2 displays the model-fitting results for depressive symptoms and antisocial behavior and the variance explained by each component. For both dimensions, genetic similarity was the most important influence, accounting for one-half of the observed individual differences. Environmental influences were also important, with shared influences playing an especially important role for antisocial behavior and nonshared influences playing an especially important role for depressive symptoms.

Next, gender differences in the genetic and shared environmental components of depressive symptoms and antisocial behavior were examined. Within the model-fitting procedures, a significant gender difference is defined as a significant change in the fit of the model in which the genetic parameter is held invariant across gender compared with the model in which the genetic parameter is allowed to vary. Because the models are nested, the fit of the two models can be compared directly by subtracting the difference in chi-squares with, in this case, 1 degree of freedom (Loehlin, 1992). For both depressive symptoms and antisocial behavior, there was no significant gender difference in influence of genetics or shared environment (i.e., chi-square change with 1 degree of freedom was <4 for all comparisons). It is important to consider that the present sample size had power to detect only relatively large effects. Specifically, we had 80% power to detect heritability differences between genders of about 40%.

Bivariate Analyses

The observed (phenotypic) correlation between depressive and antisocial symptoms on the composite measure was .47, which is consistent with numerous other studies of psychopa-

thology in adolescence (Verhulst & van der Ende, 1993). In multivariate genetic analyses, genders were combined because no differences in the univariate analyses were detected and because the correlation between depressive symptoms and antisocial behavior was similar for boys ($r = .48$) and girls ($r = .47$).

The goal of bivariate behavioral genetic analyses is to decompose the correlation between two constructs into genetic and environmental components. This is possible by analyzing the cross-sibling correlations, that is, one adolescent's score on dimension A with his or her sibling's score on dimension B. A genetic correlation for the liability to both constructs is implied if the cross-sibling correlations decrease with decreasing genetic relatedness.

Table 1 displays the cross-sibling correlations. Genetic influence is suggested because the MZ cross-sibling correlation of .35 is greater than the DZ cross-sibling correlation of .11 and the correlation for the other sibling groups. However, the cross-sibling correlations for the other groups do not correspond to their genetic relatedness, a finding that suggests some shared environmental contribution to the correlation. Finally, a nonshared environmental contribution to the correlation is suggested because the cross-sibling correlation for MZ twins (.35) is less than the phenotypic correlation (.47). The results of the bivariate model-fitting analyses confirm these impressions, as shown in Figure 1.

To estimate the genetic and environmental estimates that are unique to depression and antisocial behavior in Figure 1 (i.e., Ga, Es, and En), we fixed the two loadings on the latent common factors for Ga, Es, and En to be equal. As in the univariate case, the significance of each path was determined by the difference in chi-square between the model that estimated the parameter and a model that fixed the parameter equal to zero. Only the shared environmental influence unique to depression was not significant at $p < .05$ (see Figure 1). The model provides an adequate fit to the data. Although the chi-square of 49 with a degree of freedom of 18 is significant, model-fitting criteria such as the goodness-of-fit index (GFI = .99) and the root mean square error of approximation (.05) suggest that the model is an adequate fit (see Neale & Cardon, 1992).

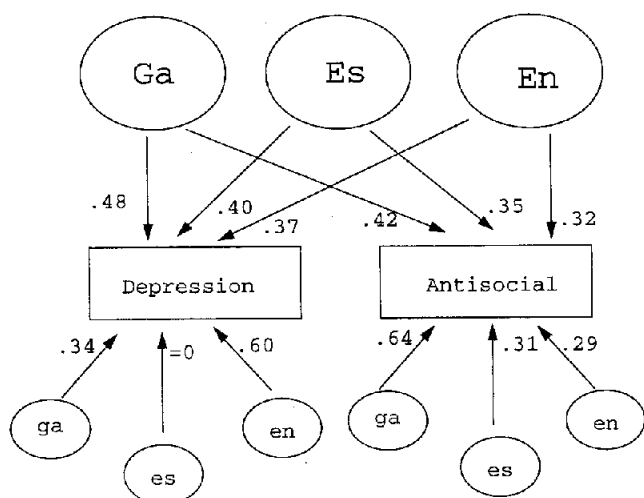


Figure 1. Bivariate behavioral genetic model indicating the shared genetic (Ga), shared environmental (Es), and nonshared environmental (En) sources of covariance between depressive symptoms and antisocial behavior as well as the shared genetic (ga), shared environmental (es), and nonshared environmental (en) influences specific to depression and antisocial behavior. Estimates are equivalent for siblings; data for only one sibling are shown: $\chi^2(18, N = 708) = 49, p < .05$; goodness-of-fit index = .99. The two paths from the latent variables Ga, Es, and En to the antisocial and depression composites are set to be equal; the values differ because the variances of the antisocial and depression composites are not identical. The model is shown without the scalar latent term used to examine whether modeling variance differences across groups made a significant improvement in the fit (see text).

Multiplying and summing the paths for the genetic and environmental latent factors common to depression and antisocial behavior, that is $(.48 * .42) + (.40 * .35) + (.37 * .32)$, produces, within rounding error, the phenotypic correlation of .47. The genetic and environmental decomposition of the phenotypic correlation can be estimated from the obtained values in Figure 1. Slightly less than 45% of the correlation between depressive symptoms and antisocial behavior can be attributed to genetic influences, $(.48 * .42)/.47$; about 30%, $(.40 * .35)/.47$, to shared environmental influences; and the remaining approximately 25%, $(.37 * .32)/.47$, to nonshared or individual-specific experiences.

Significant specific genetic (Ga) and shared (Es) and nonshared environmental (En) components of antisocial behavior were observed. In other words, there is genetic influence on antisocial behavior that is independent of the genetic influence on depressive symptoms (Ga = .64) and there are shared and nonshared environmental influences that are unique to antisocial behavior (Es = .31 and En = .29, see Figure 1). The percentage of genetic variance unique to antisocial behavior is slightly less than 70%, that is, $.64^2 / (.64^2 + .42^2)$; the corresponding figures for shared and nonshared environmental variance are, respectively, 45% and 45%. Additionally, there are significant genetic (Ga = .34; 35% unique variance) and nonshared environmental (En = .60; 72% unique variance) influences on depression that are independent of antisocial behavior.

Discussion

Using a behavioral genetic methodology that combined several types of nontwin siblings, we examined genetic and environmental influences on depressive symptoms and antisocial behavior in adolescence. Genetic and environmental explanations for the covariation between depressive symptoms and antisocial behavior were also examined. These results suggest that genetic influences underlie the tendency for developing not only a specific disorder or dimension, but also for the co-occurrence of maladjustment. In addition, these findings underscore the important role that environmentally mediated risks play in the development of depressive symptoms and antisocial behavior and in the co-occurrence of these dimensions in adolescence. The implications of these findings for research on the environmental and biological risk factors for psychopathology and nosological debates in child and adolescent psychopathology research are discussed later.

Genetic Risks for Individual and Co-occurring Dimensions of Psychopathology

Using a twin-sibling design, we found significant genetic influence on both depressive symptoms and antisocial behavior. Approximately 50% of the variance in depressive symptoms could be explained by genetics. These data complement other studies using different methods to examine genetic influences on depression and depressive symptoms in children and adolescents (Silberg et al., 1994; Wierzbicki, 1987; Weissman et al., 1988) and the already well-established findings of genetic influence on depression in adults (Tsuang & Faraone, 1992).

The corresponding genetic estimate for antisocial behavior was 56%, which falls near the higher end of the relatively wide range of estimates reported by other investigations (Gottesman & Goldsmith, 1993; Silberg et al., 1994). The diverse ways in which antisocial behavior is defined by different symptoms, severity, and duration, from serious criminality and recidivism to dimensional measures of aggression, have undoubtedly led to the disparate reports (Edelbrock, Rende, Plomin, & Thompson, 1995; Gottesman & Goldsmith, 1994). Indeed, findings reported by Lyons et al. (1995) suggest that certain specific conduct disorder symptoms show marked genetic influence whereas other symptoms of the disorder showed no genetic influence. Research on the genetic contribution to antisocial behavior in adolescence is further complicated by age-based changes that may obfuscate genetic influence (DiLalla & Gottesman, 1989). The definition of antisocial behavior used in this report—sub-clinical aggressiveness, coerciveness, and oppositionality—may show greater genetic influence than more clinical and severe forms of antisocial behavior. Although the specific percentage of variance in depressive symptoms or antisocial behavior that is due to genetic influence is likely to vary with study measure, design, and sample, the overarching conclusion from these and related analyses is that genetic factors play a significant role in the development of psychopathology in adolescence.

The absence of gender differences in the current study is consistent with some but not other analyses of genetic influence on antisocial behavior (e.g., Cadoret & Cain, 1980; Rowe, 1983; Silberg et al., 1994). It is interesting to note that Silberg et al.

found a larger genetic influence on male antisocial or externalizing behaviors in younger (8–11 years) but not older (12–16 years) children. The mixed gender findings to date and current debates regarding different expressions of antisocial behavior in males and females (Zahn-Waxler, 1993) underscore the need for future study of this issue.

The novel finding reported in these analyses is that the covariation between depressive symptoms and antisocial behavior is genetically mediated. Approximately half of the observed correlation of .47 could be attributed to a genetic liability common to both depressive and antisocial symptoms. These results contrast with data reported by Puig-Antich et al. (1989), who found that prepubertal depressed probands did not have an elevated percentage of first-degree relatives with antisocial personality. However, the differences in age, measurement, sample characteristics, and study design make direct comparisons between studies difficult. Clearly, additional family history studies as well as twin studies of clinically affected children are important to complement the field of psychiatric genetic research. Further research is also needed to specify which genetically influenced processes may underlie both depressive symptoms and antisocial behavior (e.g., neurotransmitter systems) and whether the genetic risk common to antisocial and depressive behaviors is also shared by other forms of psychopathology (e.g., anxiety, poor attention, hyperactivity). Recent molecular genetic studies of adults provide evidence that genes influence particular personality traits through their influence on the hypothesized underlying neurotransmitter systems, for example, the dopaminergic and serotonergic systems (e.g., Benjamin, Patterson, Greenberg, Murphy, & Hamer, 1996; Lesch et al., 1996). The current data suggest that depressive and antisocial symptoms may be correlated because genes influence a neurotransmitter system associated with both behavioral syndromes.

It is important to note that genetic correlation between depressive and antisocial symptoms was not unity. That is, there were some genetic influences on the liability to depressive symptoms that were independent of the genetic influences on the liability to antisocial behaviors and vice versa. These analyses thus suggest that despite a remarkably high and consistent rate of covariation between depressive and antisocial symptoms, these two syndromes can be differentiated genetically.

Environmentally Mediated Risks

Shared environmental experiences explained approximately one quarter of the variance for antisocial behavior but were less important for depressive symptoms. A significant shared environmental component to adolescent antisocial behavior is noteworthy. The finding that antisocial behavior is affected by shared environmental risks independent of genetic influence is consistent with the hypothesis that the sibling relationship may be a training ground for conduct problems (Patterson, DeBaryshe, & Ramsey, 1989; Rowe & Gulley, 1992). Furthermore, compared with depressive symptoms, antisocial behavior may be more "contagious" among siblings in the same family. Many studies implicate shared environmental factors in juvenile antisocial behavior, but a recent study suggests that shared environmental influences are not salient for antisocial behavior in adulthood (Lyons et al., 1995). These inferred developmental

differences underscore the need for long-term longitudinal research that follows adolescents through the transition to adulthood.

Although the effects were relatively small, shared (familial) environmental factors also contributed to the correlation between depressive and antisocial symptoms independent of genetic factors. That is, by virtue of sharing the same household, siblings share familial experiences that function to make them similar to each other in their levels of both depressive symptoms and antisocial behavior. It is important to note that because environmental risks were not directly examined in these analyses, the particular shared environmental factors that underlie the correlation between depressive symptoms and antisocial behaviors cannot be deciphered. However, other data from this project reported elsewhere indicate that parental behavior, especially hostility and warmth, are strongly related to depressive symptoms and to antisocial behavior (Reiss et al., 1995). These important parenting experiences that are moderately to highly correlated for siblings in the same family may buffer both siblings from, or put both siblings at risk for, experiencing both depression and antisocial behavior. This finding suggests that the environmental risk factors for depression and antisocial behavior are not completely distinct (Downey & Coyne, 1990).

The significant shared environmental parameter in explaining the covariation of depressive and antisocial symptoms conflicts with the results reported by Kendler and colleagues (Kendler et al., 1993) on the genetic and environmental correlation between depression and other disorders in a sample of adult female twins—although they did not report on externalizing or antisocial symptoms. Differences between our results and those reported by Kendler et al. may be due to shared environmental processes specific to the development of both depression and antisocial behavior or the inclusion of males in the current study. A more likely explanation for the contrasting findings is that shared environment is a more salient feature of adjustment in adolescence than it is in adulthood. That is, unlike adult siblings, adolescent siblings share much more of their environment—particularly familial environment—and the same persons provide the environments for adolescent siblings, that is, their parents. Consistent with this hypothesis, findings from a recent meta-analysis suggest that shared environmental influence on a wide variety of behavioral measures decreases with age (McCartney, Harris, & Bernieri, 1990).

The similarity of siblings notwithstanding, it is clear from these analyses that even genetically identical twins tend to be quite different from one another. For example, the correlation between genetically identical twins for depression was .66. These data therefore also highlight the importance of nonshared experiences in the development of psychopathology, because nonshared experiences (plus measurement error) explain differences in adjustment between these genetically identical siblings. Similarly, individual-specific environmental risk factors also contribute to the covariation between depressive symptoms and antisocial behavior. That is, siblings who are differentially exposed to environmental risk factors differ on both depressive symptoms and antisocial behavior. Relative to genetic influences, however, the contribution of individual-specific environmental risk factors to the covariation of depressive and antisocial symptoms is small. Nonetheless, these findings buttress previous

calls for increased attention directed toward understanding the environmental risks and protective factors that siblings who share the same home do not have in common (Plomin, 1994). It is important to note that because composite measures of adjustment variables were used, the nonshared parameter is not likely to be elevated because of shared rater bias, as it would be if only a single reporter's information were used.

Several limitations of the present study should be noted. First, this was a normal to moderate risk sample; single-mother families were excluded but there was a disproportionate percentage of stepfamilies. These results may not extend to high-risk samples and severely disturbed adolescents. Further research is required to examine whether genetic influences that influence depressive and antisocial symptoms within the normal range of adolescent psychopathology are of the same magnitude as the genetic influences that shape extreme levels of psychopathology (Plomin, 1991). In addition, the mean and variance differences among the six groups of sibling pairs are not surprising: Higher mean levels of psychopathology and variability in adjustment have been reported in studies of children's adjustment in diverse family forms (Hetherington, 1989; Hetherington & Clingempeel, 1992). Nonetheless, these findings are inconsistent with assumptions based on genetic theory. Concern about this statistical issue is largely offset by the finding that the mean and variance differences among the groups did not significantly alter the genetic-environmental profile of these measures. Third, the relatively small sample size across the six groups precluded analyses of how age and other indices of development (e.g., puberty) may influence these findings. Age and, in particular, age by gender interactions are repeatedly found to influence mean levels of psychopathology and should be pursued in further studies of genetic and environmental sources of co-occurring syndromes. Fourth, because the risk period for both dimensions of psychopathology extends well beyond adolescence, the results reported may pertain only to adolescent psychopathology. Finally, the importance of nonshared environment in the preceding models is likely to be overestimated because the nonshared variance estimate includes error of measurement.

Expanding this research methodology to other co-occurring dimensions, to younger samples, and to categorical rather than dimensional views of psychopathology are three important directions for future research. Additionally, research using longitudinal methods to examine genetic influences on change and stability of depressive and antisocial symptoms may offer further evidence for genetic risks for co-occurring dimensions of psychopathology. For example, longitudinal behavioral genetic methods may be used to examine whether genetic influences associated with antisocial behavior may be correlated with the emergence (or change) of later depression. That is, genetic factors may explain why one disorder may be a risk factor for subsequent disorders (Capaldi, 1992). Given the remarkably high rates of co-occurring dimensions of psychopathology throughout the life span (Fergusson et al., 1993; Kessler et al., 1994), researchers may benefit from examining genetic and environmental liabilities that make a person vulnerable for a pattern of adjustment problems.

References

Achenbach, T. M., & Edelbrock, C. (1981). Behavioral problems and competencies reported by parents of normal and disturbed children

aged four to sixteen. *Monographs of the Society for Research in Child Development*, 46(1, Serial No. 188).

- Andrews, V. C., Garrison, C. Z., Jackson, K. L., Addy, C. L., & McKnown, R. E. (1993). Mother-adolescent agreement on the symptoms and diagnoses of adolescent depression and conduct disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 731-738.
- Angold, A., & Costello, E. J. (1993). Depressive comorbidity in children and adolescents: Empirical, theoretical, and methodological issues. *American Journal of Psychiatry*, 150, 1779-1791.
- Angold, A., & Rutter, M. (1992). Effects of age and pubertal status on depression in a large clinical sample. *Development and Psychopathology*, 4, 5-28.
- Angold, A., Weissman, M. M., John, K., Wickramaratne, P., & Prusoff, B. (1991). The effects of age and sex on depression ratings in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30, 67-74.
- Benjamin, J., Li, L., Patterson, C., Greenberg, B. D., Murphy, D. L., & Hamer, D. H. (1996). Population and familial association between D4 dopamine receptor gene and measures of novelty seeking. *Nature Genetics*, 12, 81-84.
- Cadore, R. J., & Cain, C. (1980). Sex differences in predictors of antisocial behavior in adoptees. *Archives of General Psychiatry*, 37, 1171-1175.
- Capaldi, D. M. (1992). Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: II. A 2-year follow-up at Grade 8. *Development and Psychopathology*, 4, 125-144.
- Caron, C., & Rutter, M. (1991). Comorbidity in child psychopathology: Concepts, issues and research strategies. *Journal of Child Psychology and Psychiatry*, 32, 1063-1080.
- Cicchetti, D., & Toth, S. L. (1992a). *Rochester symposium on developmental psychopathology: Vol. 4. Developmental perspectives on depression*. Rochester, NY: University of Rochester Press.
- Cicchetti, D., & Toth, S. L. (1992b). The role of developmental theory in prevention and intervention. *Development and Psychopathology*, 4, 489-493.
- Compas, B. E., & Hammen, C. L. (1992, April). *Child and adolescent depression: Covariation and comorbidity in development*. Paper presented at the W. T. Grant Foundation Conference on Risk, Resiliency, and Development: Research and Interventions, Kiawah Island, SC.
- Costello, E. J., Costello, A. J., Edelbrock, C., Burns, B. J., Dulcan, M. K., Brent, D., & Janiszewski, S. (1988). Psychiatric disorders in pediatric primary care: Prevalence and risk factors. *Archives of General Psychiatry*, 45, 1107-1116.
- Dadds, M. R., Sanders, M., Morrison, M., & Rebgetz, M. (1992). Childhood depression and conduct disorder: II. An analysis of family interaction patterns in the home. *Journal of Abnormal Psychology*, 101, 505-513.
- DiLalla, L. F., & Gottesman, I. I. (1989). Heterogeneity of causes for delinquency and criminality: Lifespan perspectives. *Development and Psychopathology*, 1, 339-350.
- Downey, G., & Coyne, J. C. (1990). Children of depressed parents: An integrative review. *Psychological Bulletin*, 108, 50-76.
- Eaves, L. J., Silberg, J. L., Hewitt, J. K., Rutter, M., Meyer, J. M., Neale, M. C., & Pickles, A. (1993). Analyzing twin resemblance in multi-symptom data: Genetic applications of a latent class model for symptoms of conduct disorder in juvenile boys. *Behavior Genetics*, 23, 5-19.
- Edelbrock, C., Ronde, R., Plomin, R., & Thompson, L. A. (1995). Genetic and environmental effects on competence and problem behaviors in childhood and early adolescence. *Journal of Child Psychology and Psychiatry*, 36, 775-785.
- Feehan, M., McGee, R., & Williams, S. M. (1993). Mental health disorder

- ders from age 15 to age 18 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 1118–1126.
- Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1993). Prevalence and comorbidity of DSM-III-R diagnoses in a birth cohort of 15 year olds. *Journal of the American Academy of Child Adolescent Psychiatry*, 32, 1127–1134.
- Geller, B., Chestnut, E. C., Miller, M. D., Price, D. T., & Yates, E. (1985). Preliminary data on DSM-III associated features of major depressive disorder in children and adolescents. *American Journal of Psychiatry*, 142, 643–644.
- Gottesman, I. I., & Goldsmith, H. H. (1994). Developmental psychopathology of antisocial behavior: Inserting genes into its ontogenesis and epigenesis. In C. A. Nelson (Ed.), *Threats to optimal development: Minnesota symposium on child psychology* (Vol. 27, pp. 69–104). Hillsdale, NJ: Erlbaum.
- Harrington, R., Fudge, H., Rutter, M., Pickles, A., & Hill, J. (1990). Adult outcomes of childhood and adolescent depression. *Archives of General Psychiatry*, 47, 465–473.
- Harrington, R., Fudge, H., Rutter, M., Pickles, A., & Hill, J. (1991). Adult outcomes of childhood and adolescent depression: II. Links with antisocial disorders. *Journal of the American Academy of Child Adolescent Psychiatry*, 30, 434–439.
- Hetherington, E. M. (1989). Coping with family transitions: Winners, losers, and survivors. *Child Development*, 60, 1–14.
- Hetherington, E. M., & Clingempeel, W. G. (1992). Coping with marital transitions: A family systems perspective. *Monographs of the Society for Research on Child Development*, 57(2–3, Serial No. 227).
- Joreskog, K. G., & Sorbom, D. (1993). *LISREL* (8th ed.). Chicago: Scientific Software International.
- Kandel, D. B., & Davies, M. (1982). Epidemiology of depressed mood in adolescents: An empirical study. *Archives of General Psychiatry*, 39, 1205–1212.
- Kandel, D. B., & Davies, M. (1986). Adult sequelae of adolescent depressive symptoms. *Archives of General Psychiatry*, 43, 255–262.
- Kashani, J. H., Beck, N. C., Hooper, E. W., Fallahi, C., Corocran, C. M., McAllister, J. A., Rosenberg, T. K., & Reid, J. C. (1987). Psychiatric disorders in a community sample of adolescents. *American Journal of Psychiatry*, 144, 584–589.
- Kashani, J. D., Orvaschel, H., Rosenberg, T. K., & Reid, J. C. (1989). Psychopathology in a community sample of children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28, 701–706.
- Kendler, K. S., Heath, A. C., Neale, M. C., Kessler, R. C., & Eaves, L. J. (1993). Alcoholism and major depression in women: A twin study of the causes of comorbidity. *Archives of General Psychiatry*, 50, 690–698.
- Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). Major depression and generalized anxiety disorder: Same genes, (partly) different environments? *Archives of General Psychiatry*, 49, 716–722.
- Kendler, K. S., Neale, M. C., MacLean, C. J., Heath, A. C., Eaves, L. J., & Kessler, R. C. (1993). Smoking and major depression: A causal analysis. *Archives of General Psychiatry*, 50, 36–43.
- Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., Wittchen, H.-U., & Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. *Archives of General Psychiatry*, 51, 8–19.
- Kovacs, M. (1981). The Children Depression Inventory (CDI). *Psychopharmacology Bulletin*, 21, 995–998.
- Kovacs, M., Paulauskas, S., Gatsonis, C., & Richards, C. (1988). Depressive disorders in childhood: III. A longitudinal study of the comorbidity with and risk for conduct disorder. *Journal of Affective Disorders*, 15, 205–217.
- Lesch, K. P., Bengel, D., Heils, A., Sabol, S., Greenberg, B. D., Petri, S., Benjamin, J., Muller-Reible, C. R., Hamer, D. H., & Murphy, D. L. (1996). A gene regulatory region polymorphism alters serotonin transporter expression and is associated with anxiety-related personality traits. *Science*, 274, 1527–1530.
- Livesley, W. J., Jang, K. L., Jackson, D. N., & Vernon, P. A. (1993). Genetic and environmental contributions to dimensions of personality disorder. *American Journal of Psychiatry*, 150, 1826–1831.
- Loehlin, J. C. (1992). *Genes and environment in personality development*. Newbury Park, CA: Sage.
- Lyons, M. J., True, W. R., Eisen, S. A., Goldberg, J., Meyer, J. M., Faraone, S. V., Eaves, L. J., & Tsuang, M. T. (1995). Differential heritability of adult and juvenile antisocial traits. *Archives of General Psychiatry*, 52, 906–915.
- McCartney, K., Harris, M. J., & Bernieri, F. (1990). Growing up and growing apart: A developmental meta-analysis of genetic studies. *Psychological Bulletin*, 107, 220–237.
- McCaughey, E., Myers, K., Mitchell, J., Calderon, R., Schloredt, K., & Treder, R. (1993). Depression in young people: Initial presentation and clinical course. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 714–722.
- Neale, M. C. (1994). *Mx: Statistical modeling* (2nd ed.). (Available from Virginia Commonwealth University, Department of Psychiatry, Box 710 MCV, Richmond, VA 23298)
- Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Norwell, MA: Kluwer Academic.
- Nichols, R. C., & Bilbro, W. C. (1966). The diagnosis of twin zygosity. *Acta Genetica*, 16, 265–275.
- Nigg, J. T., & Goldsmith, H. H. (1994). Genetics of personality disorders: Perspectives from personality and psychopathology research. *Psychological Bulletin*, 115, 346–380.
- Patterson, G. R. (1982). *Coercive family process: Vol. 3. A social learning approach*. Eugene, OR: Castalia.
- Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist*, 44, 329–335.
- Plomin, R. (1991). Genetic risk and psychosocial disorders: Links between the normal and the abnormal. In M. Rutter & P. Caesar (Eds.), *Biological risk factors for psychosocial disorders* (pp. 101–138). Cambridge, England: Cambridge University Press.
- Plomin, R. (1994). *Genetics and experience*. Thousand Oaks, CA: Sage.
- Plomin, R., DeFries, J. C., & McClearn, G. E. (1990). *Behavioral genetics: A primer* (2nd ed.). New York: Freeman.
- Plomin, R., Nitz, K., & Rowe, D. C. (1990). Behavioral genetics and aggressive behavior in childhood. In M. Lewis & S. M. Miller (Eds.), *Handbook of developmental psychopathology* (pp. 119–133). New York: Plenum Press.
- Puig-Antich, J. (1982). Major depression and conduct disorder in prepuberty. *Journal of the American Academy of Child Psychiatry*, 21, 118–128.
- Puig-Antich, J., Goetz, D., Davies, M., Kaplan, T., Davies, S., Ostrow, L., Asnis, L., Twomey, J., Iyengar, S., & Ryan, N. D. (1989). A controlled family history study of prepubertal major depressive disorder. *Archives of General Psychiatry*, 46, 406–418.
- Reiss, D., Hetherington, E. M., Plomin, R., Howe, G. W., Simmens, S. J., Henderson, S. H., O'Connor, T. G., Bussell, D. A., Anderson, E. R., & Law, T. C. (1995). Genetic questions for environmental studies: Differential parenting of siblings and its association with depression and antisocial behavior in adolescence. *Archives of General Psychiatry*, 52, 925–936.
- Reiss, D., Plomin, R., Hetherington, E. M., Howe, G., Rovine, M., Tryon, A., & Hagan, M. S. (1994). The separate worlds of teenage siblings. In E. M. Hetherington, D. Reiss, & R. Plomin (Eds.), *Separate social worlds of siblings: Impact of nonshared environment on development* (pp. 63–109). Hillsdale, NJ: Erlbaum.

- Robins, L., & Regier, D. A. (1991). *Psychiatric disorders in America*. New York: Free Press.
- Rolf, J., Masten, A. S., Cicchetti, D., Nuechterlein, K. H., & Weintraub, S. (1992). *Risk and protective factors in the development of psychopathology*. New York: Cambridge University Press.
- Rowe, D. C. (1983). Biometric genetic models of self-reported delinquent behavior: A twin study. *Behavior Genetics*, *13*, 473-489.
- Rowe, D. C., & Gulley, B. L. (1992). Sibling effects on substance use and delinquency. *Criminology*, *30*, 217-233.
- Rowe, D. C., Rodgers, J. L., & Meseck-Bushey, S. (1992). Sibling delinquency and the family environment: Shared and unshared influences. *Child Development*, *63*, 59-67.
- Rutter, M. (1989). Isle of Wight revisited: Twenty-five years of child psychiatric epidemiology. *Journal of the American Academy of Child and Adolescent Psychiatry*, *28*, 633-653.
- Rutter, M., MacDonald, H., Le Couteur, A., Harrington, R., Bolton, P., & Bailey, A. (1990). Genetic factors in child psychiatric disorders: II. Empirical findings. *Journal of Child Psychology and Psychiatry*, *31*, 39-83.
- Silberg, J. L., Erickson, M. T., Meyer, J. M., Eaves, L. J., Rutter, M. L., & Hewitt, J. K. (1994). The application of structural equation modeling to maternal ratings of twins' behavioral and emotional problems. *Journal of Consulting and Clinical Psychology*, *62*, 510-521.
- Stiffman, A. R., Cheuh, H.-J., & Earls, F. (1992). Predictive modeling of change in depressive disorder and counts of depressive symptoms in urban youths. *Journal of Research on Adolescence*, *2*, 295-316.
- Tsuang, M. T., & Faraone, S. V. (1992). *The genetics of mood disorders*. Baltimore: Johns Hopkins University Press.
- Verhulst, F. C., & van der Ende, J. (1993). "Comorbidity" in an epidemiological sample: A longitudinal perspective. *Journal of Child Psychology and Psychiatry*, *34*, 767-783.
- Weissman, M. M., Warner, V., Wickramaratne, P., & Prusoff, B. A. (1988). Onset of major depression in adolescence and early adulthood: Findings from a family study of children. *Journal of Affective Disorders*, *15*, 269-277.
- Whitaker, A., Johnson, J., Shaffer, D., Rapoport, J. L., Kalikow, K., Walsh, B. T., Davies, M., Braiman, S., & Dolinsky, A. (1990). Uncommon troubles in young people: Prevalence estimates of selected psychiatric disorders in a referred adolescent population. *Archives of General Psychiatry*, *47*, 487-496.
- Wierzbicki, M. (1987). Similarity of monozygotic and dizygotic child twins in level and lability to subclinically depressed mood. *American Journal of Orthopsychiatry*, *57*, 33-40.
- Zahn-Waxler, C. (1993). Warriors and worriers: Gender and psychopathology. *Development and Psychopathology*, *5*, 79-90.
- Zill, N. (1985). *Behavior problems scale developed for the 1981 Child Health supplement to the National Health Interview Survey*. Washington, DC: Child Trends, Inc.
- Zill, N. (1988). Behavior, achievement, and health problems among children in stepfamilies: Findings from a national survey of child health. In E. M. Hetherington & J. D. Arasteh (Eds.), *Impact of divorce, single parenting, and stepparenting on children* (pp. 325-368). Hillsdale, NJ: Erlbaum.
- Zoccolillo, M. (1992). Co-occurrence of conduct disorder and its adult outcomes with depressive and anxiety disorders: A review. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 547-556.

Received August 15, 1995

Revision received October 7, 1996

Accepted February 17, 1997 ■