

Maternal Depression and Children's Antisocial Behavior

Nature and Nurture Effects

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Background: Children of depressed mothers have elevated conduct problems, presumably because maternal depression disrupts the caregiving environment. Alternatively, the association between maternal depression and children's antisocial behavior (ASB) may come about because (1) depressed women are likely to have comorbid antisocial personality traits, (2) depressed women are likely to mate and bear children with antisocial men, or (3) children of depressed mothers inherit a genetic liability for psychopathology.

Method: We used data from the E-Risk Study, a representative British cohort of 1116 twin pairs assessed at 5 and 7 years of age. We tested for environmental mediation of the association between maternal depression during the children's first 5 years of life and children's ASB at age 7 years, free from familial liability for ASB.

Results: Maternal depression occurring after, but not before, the twins' birth was associated with child ASB and showed a significant dose-response relationship with child

ASB at 7 years of age. Parental history of ASPD symptoms accounted for approximately one third of the observed association between maternal depression and children's ASB, but maternal depression continued to significantly predict children's ASB. Intraindividual change analyses indicated that children exposed to their mother's depression between ages 5 and 7 years showed a subsequent increase in ASB by age 7 years. The combination of depression and ASPD symptoms in mothers posed the greatest risk for children's ASB.

Conclusions: Studies ignoring genetic transmission overestimate social transmission effects because both genetic and environmental processes are involved in creating risk for ASB in children of depressed mothers. Interventions for depressed mothers aiming to reduce conduct problems in their children should address parents' antisocial personality, as well as mothers' depression.

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CHILDREN OF DEPRESSED mothers are at increased risk of developing emotional and behavioral problems,¹⁻⁶ including antisocial behavior (ASB).⁷⁻¹² Psychosocial theories posit that the link between maternal depression and children's ASB represents environmental causation via "nurture." That is, depressed mothers provide inadequate parenting,¹³ poor-quality interactions,⁸ and stressful family contexts^{7,14} that promote behavioral problems in their children. If this psychosocial account is correct, it would imply that treating maternal depression should improve parenting and family functioning and, consequently, improve children's behavior. However, there are at least 3 alternative explanations for the association between maternal depression and children's ASB: (1) maternal comorbidity for antisocial traits, (2) cross-trait assortative mat-

ing with antisocial fathers, and (3) genetic transmission of risk. Although these alternative hypotheses pose a considerable challenge to the psychosocial account that maternal depression causes children's ASB, to our knowledge, they have not been tested. Using data from a longitudinal, population-based sample of 7-year-old twins and their mothers, we tested whether each of these alternative hypotheses could be ruled out to show that the effect of maternal depression on children's ASB is transmitted environmentally.

The first alternative hypothesis is that the association between maternal depression and children's ASB comes about because depressed women are significantly more likely than nondepressed women to have comorbid conduct disorder and/or antisocial personality traits.¹⁵⁻²¹ Furthermore, maternal ASB is associated with poor parenting^{22,23} and children's conduct prob-

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lems.²⁴ According to this comorbidity hypothesis, it may be depressed mothers' antisocial history and not their depression per se that explains their children's elevated ASB, implying that treating mothers for depression would not necessarily reduce their children's ASB. Instead, treatment should target antisocial mothers and unskilled parenting.

The second alternative explanation is that the association between mothers' depression and their children's ASB results because depressed women are likely to mate and bear children with antisocial men at rates greater than expected by chance.²⁵⁻²⁷ According to this cross-trait assortative mating hypothesis, the association between maternal depression and children's ASB may come about because dually affected couples, regardless of specific psychiatric diagnoses, create more dysfunctional family environments for their children.^{27,28} Moreover, antisocial fathers often provide conflictual, abusive, and socioeconomically disadvantaged family environments that increase the likelihood of children's antisocial outcomes.^{29,30} If children of depressed mothers behave antisocially because of the influence of antisocial fathers, this would imply that treating mothers for depression alone would not improve their children's antisocial problems. Treatment should focus on the dysfunctional family milieu that results from an antisocial father.

The association between mothers' and fathers' antisocial personality and maternal depression raises the third alternative hypothesis that children of depressed mothers behave antisocially because they inherit a genetic liability for ASB (ie, via "nature"). We know of no studies that have tested this hypothesis but raise it for several reasons. First, broad-spectrum internalizing and externalizing disorders, in general,³¹ and depressive and ASB disorders, in particular,^{32,33} share some common genetic liability, suggesting that depressed women may transmit this common genetic liability to their children. Second, research shows that antisocial fathers transmit a genetic liability for ASB to their children even when they are absent from their children's lives³⁴ and independently of maternal parenting.³⁵ Third, adoption studies have shown that genetic risk on the basis of antisocial personality in biological mothers predicts ASB in children adopted and reared by prosocial parents.^{36,37} If children of depressed mothers have ASB problems because they inherit a genetic liability, then this would discredit the psychosocial hypothesis that children of depressed mothers behave antisocially because of the poor caregiving they receive.

In the present study, we applied a rigorous test of environmental mediation by using a prospective, longitudinal design of twins and their mothers, who were interviewed for *DSM-IV*³⁸ diagnosis of major depression. If mothers' depression is found to significantly predict children's ASB, after controlling for mothers' and fathers' antisocial traits, this would validate efforts to treat depressed mothers to reduce their children's conduct problems and provide renewed support for investigating which environmental processes can most effectively be modified.

Participants were members of the Environmental Risk (E-Risk) Longitudinal Twin Study, which investigates how genetic and environmental factors shape children's development. The E-Risk sampling frame was 2 consecutive birth cohorts (1994 and 1995) in a birth register of twins born in England and Wales.³⁹ Of the 15 906 twin pairs born in these 2 years, 71% joined the register.

The E-Risk Study probability sample was drawn using a high-risk stratification sampling procedure. High-risk families were those in which the mother had her first birth when she was 20 years of age or younger. We used this sampling (1) to replace high-risk families who were selectively lost to the register via nonresponse and (2) to ensure sufficient base rates of families at risk. Age at first childbearing was used because it was present for virtually all families in the register, is relatively free of measurement error, and is a known risk factor for children's problem behaviors.^{40,41} The sampling strategy resulted in a final sample in which one third of study mothers (younger only) constituted a 160% oversample of mothers who were at high risk based on their young age at first birth (15-20 years). The other two thirds of study mothers accurately represent all mothers in the general population (aged 15-48 years) in England and Wales in 1994 and 1995 (estimates derived from the General Household Survey⁴²). To provide unbiased statistical estimates that can be generalized to the population of British families with children born in the 1990s, the data reported in this article were corrected with weighting to represent the proportion of young mothers in that population.

The E-Risk Study sought a sample size of 1100 families to allow for attrition in future years of the longitudinal study while retaining statistical power. An initial list of families who had same-sex twins was drawn from the register to target for home visits, with a 10% oversample to allow for nonparticipation. Of the families from the initial list, 1116 (93%) participated in home-visit assessments when the twins were aged 5 years, forming the base sample for the study; 4% of families refused, and 3% could not be reached after many attempts. Written informed consent was obtained from mothers. With parents' permission, questionnaires were posted to the children's teachers, and teachers returned questionnaires for 94% of cohort children. A follow-up home visit was conducted 18 to 24 months after the children's age-5 assessment (hereafter called the age-7 follow-up). Follow-up data were collected for 98% of the 1116 E-Risk Study families, and teacher questionnaires were obtained for 91% of the 2232 E-Risk Study children (93% of those participating in the follow-up). The E-Risk Study has received ethical approval from the Maudsley Hospital ethics committee, London, England.

Zygosity was determined using a standard zygosity questionnaire, which has been shown to have 95% accuracy.⁴³ Ambiguous cases were zygosity-typed using DNA. The sample included 56% monozygotic and 44% dizygotic twin pairs. All twin pairs were same-sex, and sex was evenly distributed within zygosity (49% male).

MEASURES

Maternal Depression

At the age-5 assessment, mothers' major depressive disorder (MDD) was assessed using the Diagnostic Interview Schedule⁴⁴ according to *DSM-IV*³⁸ criteria. Unweighted, the prevalence of maternal lifetime MDD was 35% (n=390; weighted to

represent the population, it was 33%). Using a life-event calendar to aid recall,⁴⁵ mothers who met criteria for MDD specified whether they experienced episodes of depression at any time before the twins' birth, during the twins' first year of life, between the twins' first and fourth birthdays, and in the past year since the twins' fourth birthday. Unweighted, 317 (28%) mothers had MDD during the twins' first 5 years of life (26%, weighted to represent the population). The timing of mothers' depression was classified as never depressed (n=728), depressed only before twins' birth (n=68), depressed only after twins' birth (n=193), and depressed both before and after twins' birth (n=124). The number of periods (ranging from 0-3) during which mothers were depressed since the twins' birth was a count of developmental periods (ie, during first year, between ages 1-4 years, and between ages 4-5 years) that mothers reported having had a depressive episode. Mothers were, on average, aged 33 years at the age-5 assessment (SD=5 years, 10 months). In a separate 1-month test-retest study of 10-year retrospective reporting of psychiatric symptoms, we found that the reliability for accurate recall of the timing of depressive episodes, using the Life History Calendar method,⁴⁵ was 92.5%. Mothers' 12-month MDD was assessed at the age-7 assessment using the same method. Unweighted, 13% (n=139) of mothers had interim MDD (10% weighted to represent the population).

Mothers' and Fathers' Antisocial Personality Disorder Symptoms

At the age-5 assessment, mothers reported on their own and on the twins' biological fathers' antisocial history. Questions were derived from the Diagnostic Interview Schedule for DSM-IV⁴⁴ and assessed the lifetime presence of antisocial personality disorder (ASPD) symptoms, supplemented by items from the Young Adult Behavior Checklist,⁴⁶ to total 25 questions. The DSM-IV³⁸ symptom criteria for ASPD covered illegal behavior (5 items), deceitfulness (4 items), impulsivity (4 items), aggressiveness (4 items), recklessness (2 items), and irresponsibility (5 items). The seventh symptom, "lack of remorse," was asked only about fathers (1 item) and not of mothers about themselves because valid self-reports of remorse are difficult to obtain. A symptom was considered to be present if the mother reported any 1 behavioral item representing a symptom as being "very true or often true." Symptom counts ranged from 0 to 6 for mothers (mean±SD, 0.57±1.05) and from 0 to 7 (mean±SD, 1.16±1.83) for fathers. Although fathers were not interviewed, a methodological study of mother-father agreement about men's ASPD in a representative subset of this study sample showed that women provide reliable information about their children's fathers' antisocial personality. The latent correlation between men's and women's reports about the men's number of ASPD symptoms was 0.76 (95% confidence interval, 0.55-0.97).⁴⁷ (Of the fathers, 4.5% had never resided with their children, and 74% had always resided with their children.³⁴)

Children's ASB

Children's ASB at ages 5 and 7 years was assessed using the Achenbach family of instruments. Items from the Delinquent Behavior (eg, lying, swearing, stealing) and Aggressive Behavior (eg, physically attacks people, hot temper) scales of the Child Behavior Checklist⁴⁸ and the Teacher's Report Form⁴⁹ were supplemented with DSM-IV³⁸ items assessing conduct disorder.

Some researchers have cautioned that depressed mothers may overreport their children's problems, which artificially inflates the statistical association between their depression and their children's behavior problems.⁵⁰⁻⁵⁵ We used 2 strategies to address this potential problem: (1) we measured children's ASB

at the age-7 assessment, 2 years after maternal depression was assessed, and (2) we report findings using teacher reports of children's ASB to corroborate mothers' reports. The ASB scale was derived by (1) summing items from mother and teacher reports, and (2) using teacher reports only. For 5% of children with missing teacher reports, their mother's score was used to impute the combined mother/teacher report.

Mother and teacher reports of ASB correlated 0.30 and 0.38 ($P<.001$) at ages 5 and 7 years, respectively, which is typical of interrater agreement about children's behavioral problems.⁵⁶ Antisocial behavior at ages 5 and 7 years were correlated 0.69 and 0.52 ($P<.001$) for mother/teacher and teacher reports, respectively. The internal consistencies of the mother/teacher and teacher report scores ranged from 0.94 to 0.95. Means are reported as z scores.

STATISTICAL ANALYSIS

First, we used ordinary least squares regression to test the association between maternal depression and children's ASB, adjusted for sex. We compared children of mothers who did and did not meet diagnostic criteria for MDD during the twins' first 5 years of life. The baseline model was estimated as

$$\text{Model I: } CHILDASB = \beta_0 + \beta_1(MATDEP) + e,$$

where *CHILDASB* refers to the child's ASB, β_0 refers to the intercept, *MATDEP* refers to the mother's MDD diagnosis (yes or no), and *e* refers to error. Regression results are based on the sandwich or Huber/White variance estimator,^{57,58} a method available in Stata 8.0,⁵⁹ which adjusts estimated standard errors to account for the dependence in the data due to analyzing sets of twins and provides results that are robust to model assumptions.⁶⁰

Second, planned contrast analyses⁶¹ were used to test the association between children's ASB and (1) the timing and (2) the number of periods of maternal depression.

Third, we tested the maternal comorbidity hypothesis by reestimating model I after entering a measure indexing mothers' ASPD symptoms,

$$\text{Model II: } CHILDASB = \beta_0 + \beta_1(MATDEP) + \beta_2(\text{Mother's ASPD}) + e.$$

Fourth, we tested the cross-trait assortative mating model by reestimating model I after entering a measure of biological fathers' ASPD symptoms,

$$\text{Model III: } CHILDASB = \beta_0 + \beta_1(MATDEP) + \beta_2(\text{Father's ASPD}) + e.$$

Fifth, we tested the combination of maternal comorbidity and cross-trait assortative mating by reestimating model I after entering measures of both mothers' and fathers' ASPD symptoms.

$$\text{Model IV: } CHILDASB = \beta_0 + \beta_1(MATDEP) + \beta_2(\text{Mother's ASPD}) + \beta_3(\text{Father's ASPD}) + e.$$

Sixth, we used DeFries-Fulker (DF) regression analyses to estimate the heritability of children's ASB at age 7 years. DeFries-Fulker analysis uses kinship-pair data (eg, twin data) to separate genetic and environmental influences in a regression framework.⁶² The sandwich variance estimator was used to correct for the nonindependence of twin observations.⁶³ The equation for the basic DF regression model is:

$$ASB_{\text{twin1}} = \beta + \beta_1(R) + \beta_2(ASB_{\text{twin2}}) + \beta_3(R \times ASB_{\text{twin2}}) + e,$$

where ASB_{twin1} represents twin 1's ASB score, β_0 represents the intercept, *R* represents the coefficient of genetic relatedness (1.0 for monozygotic twins; 0.5 for dizygotic twins), and ASB_{twin2} represents twin 2's ASB score; β_3 represents the population heritability estimate (h^2), because when it is statistically signifi-

Table 1. Comparing Antisocial Behavior in Children of Mothers Who Were or Were Not Depressed in the Child's First 5 Years of Life

	Mother Not Depressed		Mother Depressed		Group Comparison t_{df}^{*}
	Sample Size	Mean \pm SD	Sample Size	Mean \pm SD	
Child antisocial behavior (z score) at 7 y of age†					
Mother and teacher report	1544	-0.20 \pm 0.85	620	0.17 \pm 1.04	6.23 _{1,1081} ‡
Teacher report	1452	-0.13 \pm 0.81	559	0.12 \pm 1.12	3.88 _{1,1019} ‡

*Degrees of freedom are based on number of families rather than number of children to account for the dependence in the data due to analyzing 2 children in the same family.^{57,58}

†Differences between groups can be interpreted in terms of standard deviation units (SDs), where SD=0.2 is considered a small effect size; SD=0.5, a medium effect size; and SD=0.8, a large effect size.⁵⁶

‡ $P < .001$.

Table 2. The Association Between Maternal Depression and Combined Mother and Teacher Reports of Children's Antisocial Behavior at 7 Years of Age, Controlling for Maternal Comorbidity and Cross-trait Assortative Mating*

	Model I: Baseline	Model II: Maternal Comorbidity	Model III: Cross-trait Assortative Mating	Model IV: Maternal Comorbidity and Cross-trait Assortative Mating	Model V: Heritability
Constant	-0.36 (-0.42 to -0.31)	-0.43 (-0.49 to -0.37)	-0.46 (-0.52 to -0.40)	-0.48 (-0.54 to -0.42)	-0.32 (-0.41 to -0.24)
Sex of child	0.34 (0.25 to 0.44)†	0.34 (0.24 to 0.43)†	0.34 (0.24 to 0.43)†	0.34 (0.24 to 0.43)†	0.15 (0.11 to 0.20)†
Maternal depression during child's first 5 y	0.35 (0.23 to 0.46)†	0.25 (0.13 to 0.37)†	0.27 (0.15 to 0.38)†	0.23 (0.11 to 0.34)†	0.10 (0.04 to 0.16)†
Maternal comorbidity					
Maternal ASPD symptoms		0.17 (0.11 to 0.24)†			0.06 (0.03 to 0.10)†
Assortative mating					
Biological father ASPD symptoms			0.10 (0.07 to 0.13)†		0.03 (0.01 to 0.05)†
Genetic transmission					
<i>R</i>				0.12 (0.05 to 0.19)†	0.13 (0.04 to 0.22)‡
<i>R</i> \times <i>ASB</i> (h^2)				0.07 (0.04 to 0.10)†	0.69 (0.63 to 0.76)‡
<i>ASB</i> _{twin2} (estimate of shared environment)					

Abbreviations: ASB, child antisocial behavior; *ASB*_{twin2}, ASB score for twin 2; ASPD, antisocial personality disorder; CI, confidence interval; h^2 , heritability; *R*, coefficient of genetic relatedness (1.0 for monozygotic twins; 0.5 for dizygotic twins).

*Values are expressed as β (95% CI). The 95% CIs are based on estimates of standard errors adjusted to account for the dependence in the data due to analyzing 2 children in the same family.^{57,58} When family socioeconomic disadvantage was included in the models as a covariate, maternal depression remained a significant predictor of ASB in models I through IV.

† $P < .001$.

‡ $P < .01$.

cant, it demonstrates that twin 1 and twin 2's resemblance for ASB is conditioned on their degree of genetic relatedness; and β_2 estimates shared environmental variation, because it represents the twins' resemblance for ASB independent of genetic resemblance.⁶⁴

The basic DF model can be expanded as:

$$\text{Model V: } ASB_{twin1} = \beta_0 + \beta_1(R) + \beta_2(ASB_{twin2}) + \beta_3(R \times ASB_{twin2}) + \beta_4(MATDEP) + \beta_5(\text{Mother's ASPD}) + \beta_6(\text{father's ASPD}) + e.$$

Next, longitudinal bivariate Cholesky models were fitted to the data on children's ASB (adjusted for sex of child) to assess genetic and environmental contributions to stability and change in children's ASB. We used Mx⁶⁵ to fit models to the data using maximum likelihood estimation. Regression analysis controlled for ASB at age 5 years to test the association between interim exposure to mothers' MDD and children's new ASB at age 7 years.

Finally, we classified children into 4 groups according to the combination of maternal MDD diagnosis (yes or no) and the presence of any ASPD symptoms (0 or ≥ 1) that mothers had. We used contrast analyses to compare groups of children on their ASB.

RESULTS

IS THERE AN ASSOCIATION BETWEEN MATERNAL DEPRESSION AND CHILDREN'S ASB?

Children of mothers who were depressed during the child's first 5 years of life had significantly elevated levels of ASB at age 7 years, according to combined mother/teacher and teacher-only reports (**Table 1**). We found no significant interaction of maternal depression by child sex on child ASB. Ordinary least squares regression models, controlling for child sex, showed that maternal depression significantly predicts child ASB (**Table 2**, model I). This pattern was replicated when teachers alone were the source of data about child ASB (**Table 3**, model I).

IS THERE A TIMING EFFECT OF MATERNAL DEPRESSION ON CHILDREN'S ASB?

Figure 1 shows maternal depression occurring after the children's birth was associated with children's ASB, but

Table 3. The Association Between Maternal Depression and Teacher-Only Reports of Children's Antisocial Behavior at 7 Years of Age, Controlling for Maternal Comorbidity and Cross-trait Assortative Mating*

	Model I: Baseline	Model II: Maternal Comorbidity	Model III: Cross-trait Assortative Mating	Model IV: Maternal Comorbidity and Cross-trait Assortative Mating	Model V: Heritability
Constant	-0.29 (-0.34 to -0.23)	-0.32 (-0.37 to -0.26)	-0.33 (-0.38 to -0.27)	-0.34 (-0.39 to -0.28)	-0.26 (-0.34 to -0.17)
Sex of child	0.33 (0.24 to 0.43)†	0.33 (0.23 to 0.42)†	0.33 (0.23 to 0.43)†	0.33 (0.23 to 0.42)†	0.14 (0.09 to 0.19)†
Maternal depression during child's first 5 y	0.22 (0.10 to 0.34)†	0.18 (0.05 to 0.30)‡	0.19 (0.06 to 0.31)‡	0.17 (0.04 to 0.29)‡	0.06 (0 to 0.13)§
Maternal comorbidity					
Maternal ASPD symptoms		0.08 (0.02 to 0.14)‡			0.04 (0 to 0.07)§
Assortative Mating					
Biological father ASPD symptoms			0.05 (0.02 to 0.08)‡		0.01 (-0.01 to 0.03)
Genetic transmission					
<i>R</i>				0.06 (-0.02 to 0.13)	0.13 (0.04 to 0.22)‡
<i>R</i> × <i>ASB</i> (<i>h</i> ²)				0.03 (0 to 0.07)	0.69 (0.61 to 0.77)†
<i>ASB</i> _{twin2} (estimate of shared environment)					

Abbreviations: ASB, child's antisocial behavior; *ASB*_{twin2}, ASB score for twin 2; ASPD, antisocial personality disorder; CI, confidence interval; *h*², heritability; *R*, coefficient of genetic relatedness (1.0 for monozygotic twins; 0.5 for dizygotic twins).

*Values are expressed as β (95% CI). The 95% CIs are based on estimates of standard errors adjusted to account for the dependence in the data due to analyzing 2 children in the same family.^{57,58} When family socioeconomic disadvantage was included in the models as a covariate, maternal depression remained a significant predictor of ASB in models I through IV.

†*P* < .001.

‡*P* < .01.

§*P* ≤ .05.

maternal depression before the children's birth was not. Planned contrast analyses revealed 3 notable findings. First, children whose mother was depressed only before the child's birth did not have significantly more ASB compared with children whose mother was never depressed ($\beta = -0.03$; SE = 0.09; $t_{(1,1085)} = 0.36$; *P* = .72 for mother/teacher report; $\beta = -0.11$; SE = 0.07; $t_{(1,1023)} = 1.49$; *P* = .14 for teacher report). Second, children whose mother was depressed only after the child's birth had significantly more ASB compared with children whose mother was never depressed only before the child's birth ($\beta = 0.38$; SE = 0.10; $t_{(1,1085)} = 3.62$; *P* < .001 for mother/teacher report; $\beta = 0.34$; SE = 0.10; $t_{(1,1023)} = 3.48$; *P* < .001 for teacher report). Third, children whose mother was depressed both before and after the child's birth and children whose mother was depressed only after the child's birth had similarly elevated ASB ($\beta = 0.07$; SE = 0.11; $t_{(1,1085)} = 0.62$; *P* = .54 for mother/teacher report; $\beta = 0.02$; SE = 0.12; $t_{(1,1023)} = 0.17$; *P* = .87 for teacher report).

IS THERE A DOSE-RESPONSE RELATIONSHIP BETWEEN MATERNAL DEPRESSION AND CHILDREN'S ASB?

Figure 2 shows a dose-response relationship between the number of developmental periods in the child's first 5 years of life during which mothers experienced a depressive episode and children's ASB at age 7 years. Ordinary least squares regression analysis revealed a significant increase in children's ASB according to the increasing number of periods when mothers experienced depression ($\beta = 0.21$; SE = 0.04; $t_{(1,1081)} = 5.43$; *P* < .001 for mother/teacher report; $\beta = 0.13$; SE = 0.04; $t_{(1,1019)} = 3.55$; *P* < .001 for teacher-only report).

IS THE ASSOCIATION BETWEEN MATERNAL DEPRESSION AND CHILDREN'S ASB ACCOUNTED FOR BY MATERNAL COMORBIDITY?

Maternal depression was significantly associated with maternal ASPD symptoms (dichotomized, $\chi^2_1 = 38.74$; *P* < .001), and maternal ASPD symptoms were significantly associated with children's ASB at age 7 years ($\beta = 0.21$; SE = 0.03; $t_{(1,1085)} = 6.74$; *P* < .001 for mother/teacher report; $\beta = 0.10$; SE = 0.03; $t_{(1,1023)} = 3.41$; *P* < .001 for teacher report). After controlling for maternal ASPD symptoms, maternal depression continued to predict children's ASB at age 7 years (Table 2 and Table 3, model II). Maternal comorbidity accounted for 29% and 18% of the effect of maternal depression on children's ASB, according to mother/teacher report and teacher report, respectively.

IS THE ASSOCIATION BETWEEN MATERNAL DEPRESSION AND CHILDREN'S ASB ACCOUNTED FOR BY CROSS-TRAIT ASSORTATIVE MATING?

Maternal depression was significantly associated with biological fathers' ASPD symptoms (dichotomized, $\chi^2_1 = 45.17$; *P* < .001), and fathers' ASPD symptoms were significantly associated with children's ASB at age 7 years ($\beta = 0.12$; SE = 0.01; $t_{(1,1082)} = 7.77$; *P* < .001 for mother/teacher report; $\beta = 0.06$; SE = 0.02; $t_{(1,1020)} = 3.86$; *P* < .001 for teacher report). After controlling for biological fathers' ASPD symptoms, maternal depression continued to predict children's ASB at age 7 years (Table 2 and Table 3, model III). Cross-trait assortative mating ac-

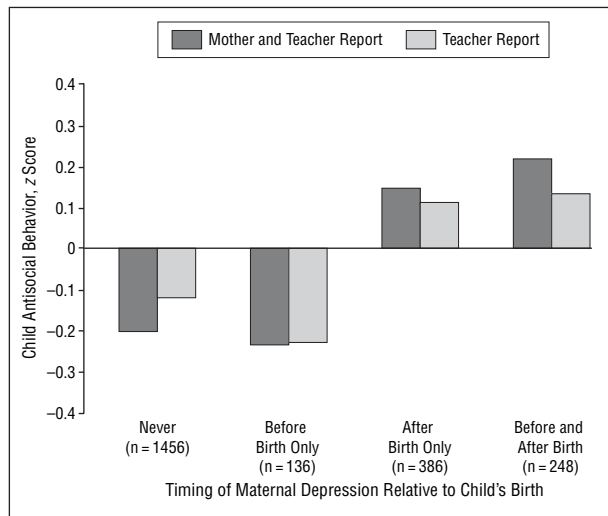


Figure 1. Mean child antisocial behavior (z score) at 7 years of age as a function of the timing of maternal depression relative to the child's birth and within the first 5 years of life. Three families had missing data on the timing calendar. Degrees of freedom are based on number of families rather than number of children to account for the dependence in the data due to analyzing 2 children in the same family.^{57,58} Degrees of freedom for mother/teacher report=3,1085; for teacher report=3,1023.

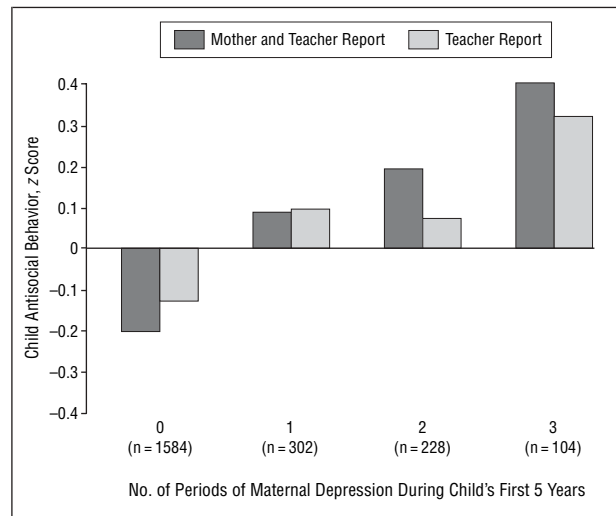


Figure 2. Dose-response relationship between children's antisocial behavior (z score) at 7 years of age and the number of developmental periods in children's first 5 years of life during which mothers were depressed. Seven families had missing data on periods of maternal depression. Degrees of freedom are based on number of families rather than number of children to account for the dependence in the data due to analyzing 2 children in the same family.^{57,58} Degrees of freedom for mother/teacher report=1,1081; for teacher report=1,1019.

counted for 23% and 14% of the effect of maternal depression on children's ASB according to mother/teacher report and teacher report, respectively.

IS THE ASSOCIATION BETWEEN MATERNAL DEPRESSION AND CHILDREN'S ASB ACCOUNTED FOR BY MATERNAL COMORBIDITY AND CROSS-TRAIT ASSORTATIVE MATING?

After controlling for mothers' and fathers' ASPD symptoms, maternal depression continued to predict children's ASB at age 7 years (Table 2 and Table 3, model IV). Maternal comorbidity and cross-trait assortative mating accounted for 34% and 23% of the effect of maternal depression on children's ASB according to mother/teacher report and teacher report, respectively.

IS CHANGE IN CHILDREN'S ASB FROM AGE 5 TO AGE 7 YEARS A FUNCTION OF EXPOSURE TO MATERNAL DEPRESSION IN THE INTERIM?

The final models in Table 2 and Table 3 show that children's ASB was highly heritable (69% for both reports). Cholesky models showed strong genetic contributions to continuity (81%-90% of the longitudinal phenotypic correlation in children's ASB). However, change in ASB from age 5 to age 7 years was accounted for by nongenetic influences (and some new genetic influences). Intraindividual analyses showed that exposure to maternal depression in the interim from age 5 to age 7 years was significantly associated with some part of this change; ASB increased more among exposed children than among nonexposed children ($\beta=0.29$; $SE=0.07$; $t_{(1,1087)}=4.03$; $P<.001$ for mother/teacher report; $\beta=0.16$; $SE=0.08$; $t_{(1,974)}=1.92$; $P=.056$ for teacher report).

HOW DOES THE ASSOCIATION BETWEEN MATERNAL DEPRESSION AND CHILDREN'S ASB VARY ACCORDING TO MATERNAL ASPD SYMPTOMS?

If maternal depression has an effect on child ASB, independently of maternal ASPD symptoms (as indicated by our model fitting), we reasoned that ASB should be greater in children of depressed mothers vs children of nondepressed mothers, whether mothers have any ASPD symptoms. We tested this association by creating 4 groups of children: (1) mother had no depression and no symptoms of ASPD ($n=1144$); (2) mother had depression but no symptoms of ASPD ($n=330$); (3) mother had no depression but had 1 or more symptoms of ASPD ($n=434$); and (4) mother had both depression and 1 or more symptoms of ASPD ($n=304$) (Table 4). The results revealed 3 notable findings. First, among children whose mothers had no symptoms of ASPD, children with a depressed mother had significantly more ASB at age 7 years compared with children with a nondepressed mother (group 2 vs group 1 in Table 4). Second, among children whose mother had 1 or more ASPD symptoms, children with a depressed mother had significantly more mother/teacher-reported ASB at age 7 years compared with children with a nondepressed mother (group 4 vs group 3). Third, children at risk because of both maternal depression and ASPD symptoms had significantly higher mother/teacher-rated ASB than children at risk because of maternal depression or maternal ASPD symptoms alone (group 4 vs groups 2 and 3; $\beta=-0.19$; $SE=0.09$; $t_{(1,1078)}=2.09$; $P<.05$ for mother/teacher report; $\beta=-0.11$; $SE=0.10$; $t_{(1,1016)}=1.12$; $P=.27$ for teacher-only report). These findings document that maternal depression in children's first 5 years of life makes a unique and additive contribution to children's risk for conduct problems over maternal ASPD symptoms.

Table 4. Children's Antisocial Behavior (Weighted z Scores) at 7 Years of Age by Maternal Depression in Child's First 5 Years and Maternal Lifetime Antisocial Personality Disorder (ASPD) Symptom Status*

Child Antisocial Behavior (z Score) at 7 y of Age	Mother Has No ASPD Symptoms			Mother Has ≥1 ASPD Symptoms			F _{df} †
	Group 1: No Depression	Group 2: Has Depression	Group 2 vs 1	Group 3: No Depression	Group 4: Has Depression	Group 4 vs 3	
Mother/teacher report							
Sample size	1114	324		424	296		
Mean ± SD	-0.29 ± 0.80	0.10 ± 1.01	4.83‡	0.07 ± 0.92	0.27 ± 1.08	2.10§	25.68 _{3,1078} ‡
Teacher report							
Sample size	1046	296		400	263		
Mean ± SD	-0.17 ± 0.77	0.09 ± 1.09	3.15	0 ± 0.87	0.16 ± 1.15	1.53	7.68 _{3,1016} ‡

*Differences between groups can be interpreted in terms of standard deviation units (SDs), where SD=0.2 is considered a small effect size; SD=0.5, a medium effect size; and SD=0.8, a large effect size.⁶⁵

†Degrees of freedom are based on number of families rather than number of children to account for the dependence in the data due to analyzing 2 children in the same family.^{57,58}

‡P<.001.

§P<.05.

||P<.01.

COMMENT

Using a population-based twin design, this study was the first, to our knowledge, to assess the relative contribution of nature and nurture in the intergenerational transmission of risk for ASB in young children of depressed mothers. We found that familial liability for ASB accounted for approximately one third of the observed association between maternal depression and children's ASB. However, our findings also suggested that children exposed to maternal depression were significantly likely to have conduct problems through a risk process that operates environmentally over any contributions of their parents' antisocial personality.

Five features of this study establish strong support for the role of nurture in the transmission of risk for ASB from depressed mothers to their children. First, maternal depression occurring after the twins' birth was linked with children's ASB, but depression occurring only before the twins' birth was not. Second, the number of developmental periods during which mothers were depressed showed a dose-response relationship with children's ASB. Third, maternal depression continued to make a unique contribution to the prediction of children's ASB, independently of parental ASPD symptoms, which is a statistical control for passive gene-environment correlation. Fourth, a longitudinal analysis controlled for prior child ASB to age 5 years that could have provoked maternal depression; children whose mothers were depressed in the interim showed a subsequent increase in ASB by age 7 years. Fifth, the results are not an artifact of maternal reporting bias, because although effect sizes were smaller when using teacher-only ratings than when using combined mother/teacher ratings of children's ASB, the findings overall remained significant either way. Although relying only on teachers as informants of child behavior eliminates potential distortion of ratings by depressed mothers, this may be applying an overly stringent control. The effect of maternal bias has been shown to account for only a small proportion of the variance in ratings of child behavior,⁵⁵ and maternal

ratings may reflect the reality that children might behave more disruptively at home with a mother who is functionally impaired by depression than they do at school.⁶⁶

Three methodological features limit the study's findings. First, research has indicated the significance of fathers' psychopathology in children's behavior problems,⁶⁷ but we could not examine risk to children of depressed fathers because fathers' depression was not assessed. Second, researchers have indicated that rates of depression may be higher in mothers of twins than of singletons,⁶⁸ and in our sample of mothers, the lifetime prevalence rate for MDD was higher than rates reported in some epidemiological samples.⁶⁹ However, a recent study found no significant difference in levels of depression between mothers of twins vs singletons.⁷⁰ Moreover, the lifetime prevalence rate we report for E-Risk Study mothers in their early 30s (33%) is comparable with that reported by Kendler et al⁷¹ in a population-based sample of women in their mid 30s (40%). Third, we cannot be certain that our findings, based on a sample of twins and their mothers in England and Wales, generalize to mothers and their singletons elsewhere. However, the association between maternal depression and children's ASB in our study (effect sizes, 0.25 to 0.37) is similar to that in studies of singletons in other countries (effect sizes, 0.25 to 0.46).^{2,8,72}

This research has implications for etiological theory, research methods, and clinical practice. First, our findings suggest that mediators of the maternal depression-child behavior problem association that have been identified in the literature, such as maladaptive parenting¹³ or disadvantaged family contexts,^{7,14} reflect more than passive gene-environment correlations.⁷³ Our research provides evidence of unique environmental mediation that is free from familial liability, and this should encourage further investigation into causal environmental processes more proximal to the child than his or her mother's depression diagnosis.

Second, our results revealed that the combination of maternal depression and ASPD symptoms had the worst

effect on children's conduct disorder. The life contexts of children whose mothers are both depressed and antisocial are likely to be highly stressful and disorganized,²² yet very little research has investigated the risks to these children, in part, because the prevalence of the ASPD diagnosis in women is low (<1%).⁷⁴ However, a lifetime history of even a single ASPD symptom in combination with depression in our study mothers resulted in a significant increase in children's ASB, relative to the risk posed to children by maternal depression alone. The developmental risks faced by children of depressed mothers who are also antisocial, as identified in the present study, warrant further investigation.

With respect to research methods, one third of the association between maternal depression and child ASB was spurious and disappeared when genetic risk for ASB was statistically controlled. The effect sizes before vs after controlling for parental antisocial traits and familial liability traits were 0.35 vs 0.23 for mother/teacher reports and 0.22 vs 0.17 for teacher reports of children's ASB. Therefore, estimates of attributable risk derived from unadjusted bivariate associations may overestimate how much children's ASB can be reduced solely by treating mothers' depression.

Regarding treatment, our findings suggest that interventions designed to reduce risk for conduct problems in children of depressed mothers are not misguided and are worthwhile. For some depressed mothers, effective treatment for depression should lead to secondary benefits for their children.⁷⁵ However, because women with an antisocial history are likely to be unskilled (and even abusive/neglectful) parents,²² medication and/or psychotherapy to lift mothers' depression may be inadequate for improving their parenting skills and decreasing their children's behavior problems. Clinicians who treat behaviorally disordered children should consider screening their mothers for depression and an antisocial history to identify treatment needs for the family.

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