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Genetic Influences on the Overlap Between Low IQ and Antisocial Behavior in Young Children

Karestan C. Koenen
Harvard School of Public Health and
Boston University School of Medicine

Avshalom Caspi and Terrie E. Moffitt
Institute of Psychiatry, King's College, London and
University of Wisconsin—Madison

Fruhling Rijsdijk and Alan Taylor
Institute of Psychiatry, King's College, London

The well-documented relation between the phenotypes of low IQ and childhood antisocial behavior could be explained by either common genetic influences or environmental influences. These competing explanations were examined through use of the Environmental Risk Longitudinal Twin Study 1994–1995 cohort (Moffitt & the E-Risk Study Team, 2002) of 1,116 twin pairs and their families. Children's IQ was assessed via individual testing at age 5 years. Mothers and teachers reported on children's antisocial behavior at ages 5 and 7 years. Low IQ was related to antisocial behavior at age 5 years and predicted relatively higher antisocial behavior scores at age 7 years when antisocial behavior at age 5 years was controlled. This association was significantly stronger among boys than among girls. Genetic influences common to both phenotypes explained 100% of the low IQ–antisocial behavior relation in boys. Findings suggest that specific candidate genes and neurobiological processes should be tested in relation to both phenotypes.

Keywords: IQ, antisocial behavior, young children, behavior genetics

Early-onset antisocial behavior is a strong risk factor for poor mental health, criminality, unemployment, and a host of other adjustment problems in adult life (Moffitt, Caspi, Harrington, & Milne, 2002; Robins, 1966). Understanding the etiology of antisocial behavior in young children is necessary for informing prevention efforts and, therefore, remains an important public health goal. Low IQ is a consistent risk factor for emergence and continuity of antisocial behavior across the life course in both prospective and cross-sectional studies, even when other relevant risk factors are statistically controlled (Hinshaw, 1992; Moffitt & Lynam, 1994; Nigg & Huang-Pollock, 2003; Simonoff et al., 2004). The effect size of the association between low IQ and antisocial behavior ranges from small to medium, with correlations ranging from .20 to .30 across studies, and comparisons of antisocial groups and control groups show half a standard deviation (approximately 8 points) difference in their IQ scores. These findings have been replicated in cohorts throughout the Western world (Hinshaw, 1992; Nigg & Huang-Pollock, 2003). Although the bulk of studies involve adolescents, the association has been reported for preschool children (Moffitt, Caspi, Harrington, & Milne, 2002; Raine, Yaralian, Reynolds, Venables, & Mednick, 2002). The association between IQ and antisocial behavior is, therefore, robust. It is not an artifact of differential detection, socioeconomic status, race, or children's poor effort on the tests (Lynam, Moffitt, & Stouthamer-Loeber, 1993). This article examines the etiology of the relation between low IQ and the development of antisocial behavior in young children.

Many explanations have been proposed for the correlation between low IQ and antisocial behavior. Lynam and Henry (2001) noted that cognitive deficits might promote antisocial behavior if children with low IQs misunderstand rules, find it too difficult to negotiate conflict with words, find school frustrating, or become tracked with antisocial peers. Nigg and Huang-Pollock (2003) have offered a model that highlights the underlying role of early deficits in self-regulation capacities, specifically emotion regulation and attentional control. Others have focused on the role of early adverse environments (Aguilar, Sroufe, Egeland, & Carlson,

Karestan C. Koenen, Department of Society, Human Development, and Health and Epidemiology, Harvard School of Public Health, and Department of Psychiatry, Boston University School of Medicine; Avshalom Caspi and Terrie E. Moffitt, Social Genetic and Developmental Psychiatry Research Centre, Institute of Psychiatry, King's College, London, and Department of Psychology, University of Wisconsin—Madison; Fruhling Rijsdijk and Alan Taylor, Social Genetic and Developmental Psychiatry Research Centre, Institute of Psychiatry, King's College, London.

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Correspondence concerning this article should be addressed to Karestan C. Koenen, Department of Society, Human Development, and Health, Harvard School of Public Health, 677 Huntington Avenue, Kresge 613, Boston, MA 02115. E-mail: kkoenen@hsph.harvard.edu

2000). As far as we know, no previous study has used the twin method to test competing explanations for the relation between IQ and antisocial behavior.

In the twin method, researchers exploit the different level of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twin pairs to estimate the contribution of genetic and environmental factors to individual differences in an outcome of interest (Plomin, DeFries, McClearn, & McGuffin, 2001). Population covariance between IQ and antisocial behavior may be partitioned into an additive genetic component and two types of environmental components. The first is a shared or family-wide environmental effect that is correlated between twins and has made children in the same family similar to each other. The second is a nonshared or child-specific environmental effect that is uncorrelated between twins and includes measurement error.

To inform both research and practice, researchers must establish the etiology of the low IQ–antisocial behavior association. One possibility is that genetic factors account for the association. Antisocial behavior is partly heritable throughout the lifespan (Rhee & Waldman, 2002), including in early childhood, particularly if it is pervasive across home and school settings (Arseneault et al., 2003). Genetic influences explain about one third of the variation in IQ in young children, and the amount of variance in IQ explained by genetic influences increases through adulthood (Plomin et al., 2001). Information on genetic etiology is valuable in the search for neurobiological systems and specific genes involved in these phenotypes. A genetically mediated association between low IQ and antisocial behavior would be consistent with a common neurodevelopmental etiology, as suggested in the model proposed by Nigg and Huang-Pollock (2003). A common genetic etiology also would suggest that some of the genes influencing IQ contribute to variation in antisocial behavior or vice versa.

A second explanation is that environmental factors influencing both IQ and antisocial behavior account for their association. If environmental factors influence the covariance between IQ and antisocial behavior, then prevention efforts targeting either of these factors would be expected to have positive effects on both outcomes. Twin studies document that environmental influences account for approximately 50% of the variance in antisocial behavior (Rhee & Waldman, 2002) and about two thirds of the variance in young children's IQ (Plomin et al., 2001). Low IQ and antisocial behavior share many putative environmental risk factors, such as low family socioeconomic status (SES; Bradley & Corwyn, 2002). Twin studies that include measured environmental variables have also found consistent effects for factors such as child maltreatment, parent–child conflict, neighborhood deprivation, and domestic violence on antisocial behavior (Burt, Krueger, McGue, & Iacono, 2001; Caspi, Taylor, Moffitt, & Plomin, 2000; Jaffee, Caspi, Moffitt, & Taylor, 2004; Jaffee, Moffitt, Caspi, Taylor, & Arseneault, 2002). Some of these same factors, such as domestic violence and child maltreatment, have also been shown in twin research to have negative associations with young children's IQ (Koenen, Moffitt, Caspi, Taylor, & Purcell, 2003).

A further question is whether the etiology of the association between IQ and antisocial behavior differs between males and females. Childhood antisocial behavior is more prevalent among males. One cohort study found that compromised intelligence had a stronger association with antisocial behavior in males than in females (Moffitt, Caspi, Rutter, & Silva, 2001) although this finding was not replicated in another cohort (Fergusson & Horwood, 2002). A possible gender difference in the relation between IQ and antisocial behavior remains unresolved, and thus we tested it here.

In this study, we used data from the 1,116 twin pairs in the Environmental Risk (E-Risk) Longitudinal Twin Study (Moffitt & the E-Risk Study Team, 2002) to accomplish four goals. The first goal was to replicate the negative relation between IQ and antisocial behavior and to examine whether low IQ predicted relatively higher antisocial behavior over time in this sample. The second goal was to examine whether the relation between IQ and antisocial behavior differed between boys and girls. The third goal was to test competing explanations for the etiology of the relation. Finally, because antisocial behavior is highly comorbid with attention-deficit/hyperactivity disorder (ADHD; Mannuzza, Klein, Abikoff, & Moulton, 2004; Patterson, DeGarmo, & Knutson, 2000) and because ADHD also is associated with low IQ (Kuntsi et al., 2004), the fourth goal was to test whether our findings persisted after the exclusion of children with ADHD diagnoses.

Method

Participants

Participants were members of the E-Risk Longitudinal Twin Study, which investigated how genetic and environmental factors shape children's development. The sampling frame from which the E-Risk families were drawn was two consecutive birth cohorts (1994 and 1995) in a birth register of twins born in England and Wales (Trouton, Spinath, & Plomin, 2002). Of the 15,906 twin pairs born in these 2 years, 71% joined the register.

Bias from nonjoining was corrected as follows. The E-Risk Study probability sample was drawn through use of a high-risk stratification strategy. 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 to replace high-risk families who were selectively lost to the register via nonresponse and to ensure sufficient base rates of environmental risk. Age at first childbearing was used as the risk-stratification variable because it was recorded for virtually all families in the register, it is relatively free of measurement error, and early childbearing is a known risk factor for children's problem behaviors (Maynard, 1997; Moffitt & the E-Risk Study Team, 2002). The sampling strategy resulted in a final sample in which one third of study mothers constituted a 160% oversample of mothers who were at high risk on the basis of their young age at first birth (13–20 years), whereas the other two thirds of study mothers accurately represented all mothers in the general population (aged 13–48 years) in England and Wales in 1994–1995 (estimates were derived from the General Household Survey; Bennett, Jarvis, Rowlands, Singleton, & Haselden, 1996). For the provision of unbiased statistical estimates from the whole sample that can be generalized to the population of British families with children born in the 1990's, the data and analyses reported in this article were corrected with weighting to represent the proportion of maternal ages in that population.

In the study, we sought a sample size of 1,100 families to allow for attrition in future years of the longitudinal study while retaining statistical power. An initial list of families who had same-gender twins was drawn from the register as targets for home visits, with a 10% oversample to allow for nonparticipation. Of the families from the initial list who were eligible for inclusion, 1,116 (93%) participated in home-visit assessments when the twins were age 5 years, forming the base sample for the study. With parents' permission, questionnaires were posted to the children's teachers (with a 94% response rate). A follow-up home visit was conducted 18 months after the twins' age-5 assessment, when the children were, on average, 6½ years old (range was 6–7 years). Follow-up data were col-

lected for 98% of the 1,116 E-Risk Study families, and teacher questionnaires were obtained for 91% of the 2,232 E-Risk Study twins (93% of those taking part in the follow-up).

Zygosity was determined via genotyping. The sample included 54% MZ twin pairs and 46% same-gender DZ twin pairs. Gender was evenly distributed within zygosity (49% male; 51% female).

Written informed consent was obtained from mothers. The E-Risk Study received ethical approval from the Maudsley Hospital Ethics Committee. Data analyses for this article received approval from the Harvard School of Public Health.

Measures

Children's IQ. This measure was individually tested at age 5 years using a short form of the Wechsler Preschool and Primary Scale of Intelligence—Revised (WPPSI–R; Wechsler, 1990). Using two subtests (Vocabulary and Block Design), we prorated children's IQs following procedures described by Sattler (1992). The prorated IQ score correlated highly (above .86) with the full-scale IQ over a wide age range and is a good measure of "g" (Sattler, 1992, p. 137). Scores ranged from 52 to 145 (raw $M = 95.79$, $SD = 14.46$; weighted $M = 97.83$, $SD = 14.40$).

Children's antisocial behavior. This measure was assessed via mother and teacher reports at ages 5 and 7 years using the Aggression and Delinquency subscales of the Child Behavior Checklist (Achenbach, 1991a, 1991b) supplemented with *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*; American Psychiatric Association, 1994) items assessing conduct and oppositional defiant disorder. Each item was scored 0 "(*not true*)," 1 "(*somewhat or sometimes true*)," or 2 "(*very true or often true*)." We followed a previously recommended strategy in which we aggregated measurements, where possible, across parent and teacher reporters (Achenbach, McConaughy, & Howell, 1987; Bird, Gould, & Staghezza, 1992; Piacentini, 1993; Piacentini, Cohen, & Cohen, 1992; van der Ende, 1999). Thus, mother and teacher reports of children's behavior problems were totaled (items were scored from 0, *not true,* to 2, *very true or often true*) for creation of a measure that reflects pervasive antisocial behavior across settings.[1] Very few children had any missing data; 86% of cases had data for all time points/raters, and 96.3% had data for three or more of the time points/raters. We addressed the small amount of missing data at each informant/age level with mean imputation, whereby we used means within gender and risk group to impute scores for a missing age/rater. Mother and teacher reports of antisocial behaviour correlated at .29, $p < .001$, which is typical of interrater agreement about behavioral problems (Achenbach et al., 1987). At age 5 years, scores ranged from 0 to 130 ($M = 21.17$, $SD = 16.27$), and at age 7 years, scores ranged from 0 to 132 ($M = 18.48$, $SD = 15.80$). The scale reliability of the combined score was .94 at age 5 years and .95 at age 7 years as calculated according to the formula by Nunnally (1967). Given the young age of the sample and concerns about reliable measurement of antisocial behavior in young children, we created a composite variable of children's antisocial behavior in early childhood by totaling the two age-specific measures, which were correlated at .69, $p < .001$.

ADHD. We examined this measure to ascertain whether IQ and antisocial behavior were associated among children who did not have the ADHD diagnosis. This diagnosis was ascertained on the basis of mother and teacher reports at ages 5 and 7 years (the years 1999–2002), as described in Kuntsi et al. (2004). In the mother interview, children's symptomatology was assessed with 18 items concerning hyperactivity, impulsivity, and inattention representing symptom criteria for ADHD specified by the *DSM-IV* (American Psychiatric Association, 1994; e.g., "very restless, has difficulty staying seated for long," "impulsive, acts without thinking, "inattentive, easily distracted"). Symptoms were reported for the preceding 6 months. Teachers rated the same set of items. A research diagnosis of ADHD was made following *DSM-IV* (American Psychiatric Association, 1994) criteria: Children received the diagnosis if they had six or more of the hyperactivity or impulsivity symptoms or if they had six or more of the inattentiveness symptoms according to either mother or teacher report. In addition, the other rater had to indicate two or more symptoms to ensure pervasiveness across home and school. Onset before age 7 years was required. The prevalence of this research diagnosis of ADHD was 8% (70% boys; 30% girls).

Results

Is There an Association Between Children's IQ and Their Antisocial Behavior?

We first tested whether the well-documented association between IQ and antisocial behavior would replicate in our sample. IQ at age 5 years was significantly correlated with antisocial behavior at age 5 years, $r = -.18$, $p < .001$; with antisocial behavior at age 7 years, $r = -.17$, $p < .001$; and with the early childhood composite measure of antisocial behavior, $r = -.19$, $p < .001$.

Does Children's Low IQ Predict Relatively Higher Antisocial Behavior at Age 7 Years When Researchers Control for Age-5 Antisocial Behavior?

We used ordinary least squares (OLS) regression models[2,3] to test the hypothesis that low IQ predicts relatively higher antisocial behavior at age 7 years when age-5 antisocial behavior is controlled. The effect of IQ on antisocial behavior at age 7 years remained significant after we controlled for age-5 antisocial behavior, $b = -0.05$, $SE = 0.02$, $p < .01$. Thus, low IQ predicted relatively higher antisocial behavior at age 7 years when age-5 antisocial behavior was controlled.

Is the Association Between Children's IQ and Children's Antisocial Behavior Similar for Boys and Girls?

Using moderated OLS regression analyses, we tested whether the association between IQ and antisocial behavior differed significantly between boys and girls. We regressed antisocial behavior on IQ, gender (1 = *boys*, 0 = *girls*), and the interaction between IQ and gender. IQ was centered for interaction models. A significant interaction term would support the hypothesis that the association between IQ and antisocial behavior is stronger for one gender.

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[1] Specific measures of antisocial behavior have been analyzed separately and have been reported to yield highly similar parameters in twin ACE models (Arseneault et al., 2003).

[2] Because these analyses included two children from each family, significance tests were based on the sandwich or Huber/White variance estimator, a method available from STATA 7.0 (Statacorp, 2001), which adjusts estimated standard errors to account for the nonindependence of data from children in the same family.

[3] Given concerns about the nonnormal distribution of antisocial behavior in this sample, data were reanalyzed through the use of negative binomial regression, which is appropriate for skewed count data characterized by overdispersion. The pattern of results was largely identical to those presented. The one exception was the model testing for whether there is a gender difference in IQ predicting the change in antisocial behavior over time. In the OLS regression, the interaction term for this test is statistically significant ($p = .01$), and in the negative binomial regression, the interaction term is not quite significant ($p = .06$).

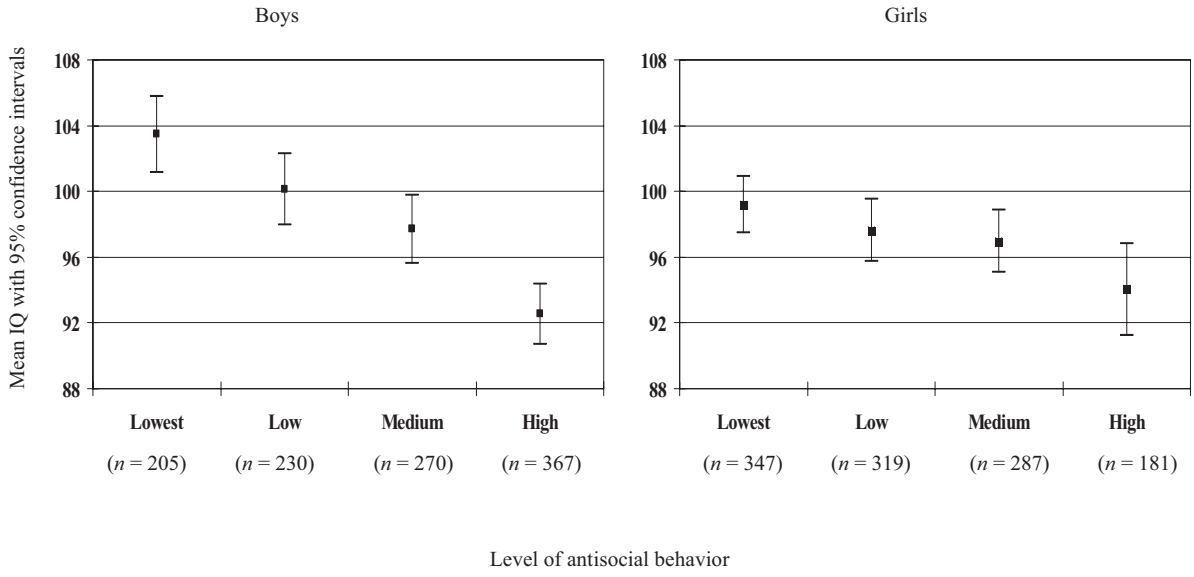

Figure 1. Mean IQ with 95% confidence intervals as a function of antisocial behavior level for boys and girls. IQ was tested at age 5 years, and antisocial behavior was a composite of four ratings by mothers and teachers of children at ages 5 and 7 years. Error bars represent 95% confidence intervals around the mean.

Boys and girls did not differ on mean IQ, $F(1, 1112) = 0.43$, $p = .51$. However, boys showed more antisocial behavior than did girls at age 5 years, $F(1, 1115) = 51.49$, $p < .001$, as well as at age 7 years, $F(1, 1088) = 54.75$, $p < .001$. In addition, the association between IQ and antisocial behavior among boys (rs ranged from $-.23$ to $-.26$, $p < .001$) was larger than the same association among girls (rs ranged from $-.09$, $p < .05$, to $-.12$, $p < .01$). The interaction effect showed that this association was indeed significantly stronger among boys than among girls, whether measured at age 5 years ($b = -0.17$, $SE = 0.06$, $p < .01$) or at age 7 years ($b = -0.20$, $SE = 0.05$, $p < .001$) or as a composite early childhood outcome ($b = -0.38$, $SE = 0.10$, $p < .001$).

We then created three dummy variables that categorized children into quartiles according to the composite measure of antisocial behavior. The lowest quartile was the reference group ($n = 552$ children) of the least antisocial children. The three comparison groups were medium-low antisocial children ($n = 549$), medium-high antisocial children ($n = 557$), or high antisocial children ($n = 548$). These dummy variables were entered simultaneously into two separate OLS regression models (one for boys, one for girls) predicting IQ.

For boys, the regression coefficients increased linearly with level of antisocial behavior as follows: low antisocial behavior ($b = -3.36$, $SE = 1.50$, $p < .05$), medium antisocial behavior ($b = -5.76$, $SE = 1.52$, $p < .001$), and high antisocial behavior ($b = -10.93$, $SE = 1.50$, $p < .001$). The regression coefficients demonstrate that boys with the highest levels of antisocial behavior had, on average, IQs that were 11 points lower than those of boys with the lowest levels of antisocial behavior. For girls, the linear association was not as strong, with level of antisocial behavior as follows: low antisocial behavior ($b = -1.57$, $SE = 1.24$, $p = .20$), medium antisocial behavior ($b = -2.23$, $SE = 1.26$, $p = .08$), and high ($b = -5.16$, $SE = 1.65$, $p = .002$). The effect sizes and gender pattern are illustrated in Figure 1, which presents mean IQ and 95% confidence intervals by quartiles of antisocial behavior for boys and girls separately.

Further moderated OLS regression analyses showed that the interaction term for IQ and gender significantly predicted age-7 antisocial behavior after controlling for age-5 antisocial behavior ($b = -0.10$, $SE = 0.04$, $p = .013$). We then stratified the sample by gender and found that after controlling for age-5 antisocial behavior, the effect of IQ on age-7 antisocial behavior remained significant for boys ($b = -0.10$, $SE = 0.03$, $p = .002$) but not for girls ($b = -.01$, $SE = 0.02$, $p = .58$). These results confirm that low IQ predicted relatively higher antisocial behavior at age 7 years more strongly in boys than in girls.

What Is the Genetic and Environmental Architecture of IQ and Antisocial Behavior?

To test hypotheses about how individual differences in IQ and antisocial behavior are affected by genetic and environmental factors, we used the statistical package Mplus 3.11 and used the robust maximum-likelihood estimator (Muthén & Muthén, 2004).

Tables 1 and 2[4] report the descriptive statistics and correlations for IQ and antisocial behavior among the twin pairs in the sample for boys and girls, respectively. The MZ and DZ within-pair correlations provide rough estimates of the extent to which genetic factors, shared environmental factors, and child-specific environmental factors contribute to IQ and antisocial behavior in childhood. For example, the greater-MZ-than-DZ correlations for boys' IQ (MZ = .73 vs. DZ = .55) and for composite antisocial behavior

[4] Means and correlation matrices were modeled through use of Mplus 3.11 (Muthén & Muthén, 2004), in which full information maximum likelihood was used in concert with both sampling weights and the Satorra–Bentler correction for nonnormal data.

Table 1
Descriptive Statistics and Correlations for IQ at Age 5 and Antisocial Behavior Composite Scores for MZ and DZ Male Twins

Variable	1	2	3	4
Male MZ twins (n = 291 pairs)				
1. Twin 1—IQ age 5	—			
2. Twin 1—Antisocial behavior composite	−.26*	—		
3. Twin 2—IQ age 5	.73*	−.24*	—	
4. Twin 2—Antisocial behavior composite	−.28*	.79*	−.27*	—
M	97.84	46.09	97.56	44.78
SD	14.37	33.67	14.20	33.41
Male DZ twins (n = 255 pairs)				
1. Twin 1—IQ age 5	—			
2. Twin 1—Antisocial behavior composite	−.28*	—		
3. Twin 2—IQ age 5	.55*	−.13*	—	
4. Twin 2—Antisocial behavior composite	−.19*	.37*	−.22*	—
M	99.01	48.90	98.38	43.34
SD	15.45	34.48	15.65	30.03

Note. Antisocial behavior composite scores represent the sum of age 5 and age 7 antisocial behavior scores. MZ = monozygotic; DZ = dizygotic.
* $p < .05$.

(MZ = .78 vs. DZ = .35) indicate substantial genetic influences on each phenotype. The pattern of cross-twin within-trait correlations was similar for girls.

In the simple univariate twin model, the variance for IQ or antisocial behavior is partitioned into the variance explained by additive genetic influences (denoted by the letter A), shared environmental influences (C), and nonshared environmental influences including error (E). The goal of fitting different structural equations to twin data is to account for the observed covariance structure using the most parsimonious number of parameters. We evaluated the fit of the ACE versus CE and AE models for each phenotype using three model-selection statistics. The first was the chi-square goodness-of-fit statistic. When models are nested (i.e., identical with the exception of constraints), the difference in fit among models is tested by the difference in the chi-square values ($\Delta\chi^2$) using as its degrees of freedom the degrees-of-freedom difference of the two models. If the chi-square difference is not statistically significant, then the more parsimonious model is selected, as the test indicates that the constrained model fits equally well to the data. Because of our use of robust maximum-likelihood estimation with sampling weights, chi-square difference tests must be adjusted because the differences between two robust chi-square goodness-of-fit statistics do not have a chi-square distribution (Satorra, 2000). Thus, we use an adjusted chi-square difference test (Muthén, 1998–2004; Satorra & Bentler, 1999). The second model-selection statistic was the comparative fit index (CFI), in

Table 2
Descriptive Statistics and Correlations for IQ at Age 5 and Antisocial Behavior Composite Scores for MZ and DZ Female Twins

Variable	1	2	3	4
Female MZ twins (n = 311 pairs)				
1. Twin 1—IQ age 5	—			
2. Twin 1—Antisocial behavior composite	−.09	—		
3. Twin 2—IQ age 5	.70*	−.14	—	
4. Twin 2—Antisocial behavior composite	−.02	.76*	−.12	—
M	96.54	33.75	96.99	33.56
SD	12.98	23.92	14.21	23.60
Female DZ twins (n = 259 pairs)				
1. Twin 1—IQ age 5	—			
2. Twin 1—Antisocial behavior composite	−.03	—		
3. Twin 2—IQ age 5	.50*	−.13	—	
4. Twin 2—Antisocial behavior composite	−.07	.36*	−.19*	—
M	98.41	34.30	98.58	32.03
SD	13.61	22.99	14.77	21.63

Note. Antisocial behavior composite scores represent the sum of age 5 and age 7 antisocial behavior scores. MZ = monozygotic; DZ = dizygotic.
* $p < .05$.

which values greater than .95 are indicative of good-fitting models (Hu & Bentler, 1999). The third model-selection statistic was the root-mean-square error of approximation (RMSEA), which is an index of the model discrepancy, per degree of freedom, from the observed covariance structure (MacCallum, Browne, & Sugawara, 1996). Values less then .05 indicate close fit to the data, and values less than .08 indicate fair fit to the data (Browne & Cudeck, 1993).

Tables 3 and 4 present the univariate model fitting results for IQ and for composite antisocial behavior in male and female twins, respectively. (The results for the univariate analyses were similar if antisocial behavior at age 5 years or age 7 years was used instead of the composite variable. These analyses are available from Karestan C. Koenen on request.) For IQ, the ACE model provided the best fit to the data for boys and girls. For boys, the proportion of variance in IQ that was accounted for by additive genetic effects was 44% ($b = 0.66$, 95% CI = .49, .84), by shared environmental factors was 31% ($b = 0.55$, 95% CI = .36, .74), and by child-specific environmental factors was 25% ($b = 0.50$, 95% CI = .45, .56). For girls, the proportion of variance in IQ that was accounted for by additive genetic effects was 45% ($b = 0.67$, 95% CI = .49, .85), by shared-environmental factors was 26% ($b = 0.51$, 95% CI = .30, .71), and by child-specific environmental factors was 29% ($b = 0.54$, 95% CI = .48, .59).

For antisocial behavior, the AE model provided the best fit. For boys, the proportion of variance that was accounted for by additive genetic effects was 78% ($b = 0.82$, 95% CI = .75, .89) and by child-specific environmental factors was 22% ($b = 0.43$, 95% CI = .39, .47). For girls, the proportion of variance that was accounted for by additive genetic effects was 77% ($b = 0.81$, 95% CI = .73, .89) and by child-specific environmental factors was 23% ($b = 0.44$, 95% CI = .40, .49).

What Is the Genetic and Environmental Architecture of the Association Between IQ and Antisocial Behavior?

In the bivariate case, we examined the genetic and environmental architecture for the covariation of children's IQ and antisocial behavior. In the bivariate twin analysis, MZ and DZ correlations are compared across traits—that is, one twin's IQ score is correlated with the cotwin's antisocial behavior score. If the cross-trait twin correlations are greater for MZ twins than for DZ twins, the implication is that genetic factors contribute to the phenotypic correlation between the two traits. A significant path from additive genetic influences (A) on IQ to antisocial behavior indicates the extent to which genetic influences on IQ also influence variation in antisocial behavior. A significant path from shared environmental influences (C) on IQ to antisocial behavior indicates the extent to which family-wide environmental influences on IQ also influence antisocial behavior. A significant path from nonshared environmental influences (E) on IQ to antisocial behavior indicates the extent to which child-specific environmental influences on IQ also influence antisocial behavior. The most parsimonious model was selected through use of the model-selection statistics described in the previous paragraph.

The cross-twin cross-trait correlations for boys were higher for MZ twins than for DZ twins (MZ = −.24 and −.28; DZ = −.13 and −.19), suggesting a role for genetic influences in the low IQ–antisocial behavior relation. For boys, model fit did not deteriorate significantly if the shared environmental pathway specific to antisocial behavior and the shared environmental and nonshared environmental pathways from IQ to antisocial behavior were fixed to zero, $\Delta\chi^2(3, N = 546) = 1.94$, $p = .59$. However, model fit deteriorated significantly if additive genetic influences on IQ were hypothesized to have no effect on antisocial behavior, $\Delta\chi^2(1, N = 546) = 52.34$, $p = .001$. The bivariate model that provided the best fit to the data consisted, therefore, of the ACE model for IQ, the AE model for composite antisocial behavior, and a significant genetic pathway from IQ to antisocial behavior. This model (Figure 2) provided excellent fit to the data, CFI = 1.00, RMSEA = .00, $\chi^2(20, N = 546) = 17.07$, $p = .65$.

Figure 2 illustrates that for the best-fitting model for boys, the only path between IQ and antisocial behavior is that from genetic influences on IQ to antisocial behavior. This path indicates that

Table 3
Fit of Univariate Quantitative Genetic Models Examining the Etiology of IQ and Antisocial Behavior in Boys (n = 546 pairs)

Variable	CFI	RMSEA	χ^2	*df*	*p*	Comparison model	$\Delta\chi^2$	Δdf	*p*
				Boys' IQ					
ACE[a]	1	.00	3.00	6	.81				
AE	.97	.05	12.18	7	.09	1	7.03	1	.008
CE	.88	.10	27.23	7	<.001	1	18.48	1	<.001
				Boys' composite antisocial behavior					
ACE	.99	.03	7.42	6	.28				
AE[a]	.99	.03	8.65	7	.28	1	0.00	1	*ns*
CE	.67	.20	81.03	7	<.001	1	57.55	1	<.001

Note. $\Delta\chi^2$ is a mean-adjusted robust chi-square difference test and, therefore, is not equal to the simple difference between the chi-square values for the models shown in Column 3 (Muthen, 1998-2004). CFI = comparative fit index; RMSEA = root-mean-square error of approximation; A, C, and E refer to additive genetic influences, shared environmental influences, and nonshared environmental (including error) influences.
[a] Best-fitting model.

Table 4
Fit of Univariate Quantitative Genetic Models Examining the Etiology of IQ and Antisocial Behavior in Girls (n = 570 pairs)

Variable	CFI	RMSEA	χ^2	*df*	*p*	Comparison model	$\Delta\chi^2$	Δdf	*p*
				Girls' IQ					
ACE[a]	.98	.04	9.02	6	.17				
AE	.96	.06	14.50	7	.04	1	5.15	1	.02
CE	.89	.10	25.50	7	<.001	1	18.19	1	<.001
				Girls' composite antisocial behavior					
ACE	1.00	.00	2.93	6	.82				
AE[a]	1.00	.00	3.42	7	.84	1	0.04	1	*ns*
CE	.72	.14	48.56	7	<.001	1	44.87	1	<.001

Note. $\Delta\chi^2$ is a mean-adjusted robust chi-square difference test and, therefore, is not equal to the simple difference between the chi-square values for the models shown in Column 3 (Muthen, 1998-2004). CFI = comparative fit index; RMSEA = root-mean-square error of approximation; A, C, and E refer to additive genetic influences, shared environmental influences, and nonshared environmental (including error) influences.
[a] Best-fitting model.

100% of the low IQ–antisocial behavior relation in boys is explained by genetic influences common to both phenotypes. When parameter estimates were calculated as proportions of the variance, genetic influences on IQ accounted for 13% of the variance in antisocial behavior. The remaining variance in antisocial behavior was accounted for by genetic influences (66%) and nonshared environmental influences (22%) unique to antisocial behavior.

For girls, the cross-twin cross-trait correlations were similar for MZ and DZ twins (MZ = −.14 and −.02; DZ = −.13 and −.07), suggesting a role for shared environmental influences in the low IQ–antisocial behavior relation. Model fit did not deteriorate significantly if the shared environmental pathway specific to antisocial behavior and the shared genetic and nonshared environmental pathways from IQ to antisocial behavior were fixed to zero, $\Delta\chi^2(3, N = 570) = 5.57$, $p = .13$. However, model fit deteriorated significantly if shared environmental influences on IQ were hypothesized to have no effect on antisocial behavior, $\Delta\chi^2(1, N = 570) = 6.55$, $p = .01$. The bivariate model that provided the best fit to the data consisted, therefore, of the ACE model for IQ, the AE model for composite antisocial behavior, and a significant

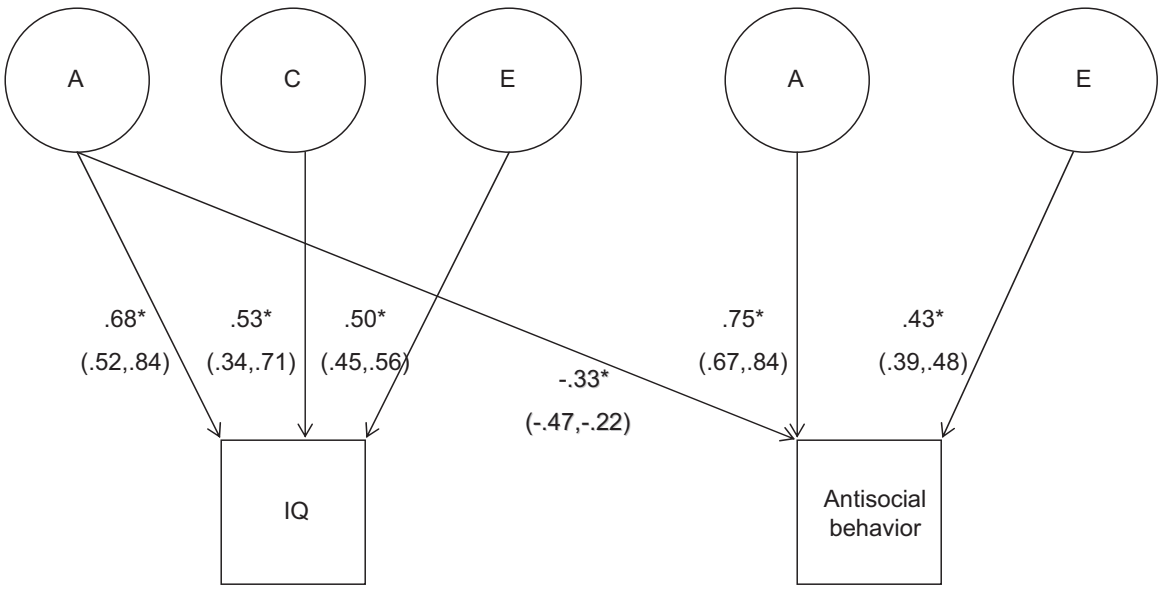

Figure 2. Parameter estimates with 95% confidence intervals for bivariate model of association between IQ and composite antisocial behavior in male twins. The letters A, C, and E refer to additive genetic influences, shared environmental influences, and nonshared environmental (including error) influences, respectively. The model is displayed for Twin 1 only; the model for Twin 2 would look identical. The variances of the latent variables are fixed at 1. All parameter estimates are statistically significant at $p < .05$.

shared environmental pathway from IQ to antisocial behavior. This model (see Figure 3) provides good fit to the data, CFI = .99, RMSEA = .02; $\chi^2(20, N = 570) = 21.71$, $p = .36$.

Figure 3 illustrates that for the best-fitting model for girls, the only path between IQ and antisocial behavior is that from shared environmental influences on IQ to antisocial behavior. This indicates that 100% of the low IQ–antisocial behavior relation in girls is explained by shared environmental influences common to both phenotypes. When parameter estimates were calculated as proportions of the variance, shared environmental influences on IQ accounted for only 2% of the variance in antisocial behavior. The remaining variance in antisocial behavior was accounted for by genetic influences (75%) and nonshared environmental influences (23%) unique to antisocial behavior.

Does the Relation Between Low IQ and Antisocial Behavior Persist After Excluding Children With ADHD Diagnoses?

To ensure that our findings were not an artifact of children with diagnosable ADHD, we reanalyzed our data, excluding children who received diagnoses of ADHD. We found that the strength of the relation between low IQ and antisocial behavior was slightly attenuated but the pattern of findings remained very consistent. IQ at age 5 years was significantly correlated with antisocial behavior at age 5 years ($r = -.13$, $p < .001$) and at age 7 years ($r = -.11$, $p < .001$) and with the early childhood composite measure of antisocial behavior (r = −.14, $p < .001$). The effect size of IQ on antisocial behavior at age 7 years after controlling for age-5 antisocial behavior remained similar, but its significance level was reduced to a trend ($b = -0.03$, $SE = 0.02$, $p = .11$).

The exclusion of children who received a diagnosis of ADHD had no effect on our findings regarding the gender difference in the strength of the association between low IQ and antisocial behavior. The association between IQ and antisocial behavior among boys (*r*s ranged from −.19 to −.22, $p < .001$) remained larger than the same association among girls (*r*s ranged from −.04, $p = .22$, to −.06, $p = .19$). The interaction effect confirmed that this association was indeed significantly stronger among boys than among girls, whether measured at age 5 years ($b = -0.17$, $SE = 0.05$, $p < .01$), at age 7 years ($b = -0.20$, $SE = 0.05$, $p < .01$), or as a composite early childhood outcome ($b = -0.38$, $SE = 0.10$, $p = .001$). Moreover, we found that after controlling for age-5 antisocial behavior, the effect of IQ on age-7 antisocial behavior was significant for boys ($b = -0.06$, $SE = 0.04$, $p < .05$) but not for girls ($b = -0.005$, $SE = 0.02$, $p = .84$).

Finally, the exclusion of children who received a diagnosis of ADHD had almost no effect on our findings regarding the genetic and environmental architecture of the relation between IQ and antisocial behavior in MZ boys ($r = -.19$, $p < .01$, and $r = -.20$, $p < .01$) versus DZ boys ($r = -.16$, $p < .05$, and $r = -.18$, $p < .05$). However, the relation between IQ and antisocial behavior in girls was reduced to nonsignificance for MZ twins ($r = -.02$, $p = .77$, and $r = -.06$, $p = .32$) and for DZ twins ($r = -.01$, $p = .84$, and $r = -.08$, $p = .24$). The best-fitting univariate models for IQ and antisocial behavior were the ACE and AE models for both boys and girls. Parameter estimates were almost identical to those presented for the complete sample.

Once children with the ADHD diagnosis were excluded from the sample, the correlations for girls were too small to be decomposed further in a bivariate twin model. We, therefore, focused on

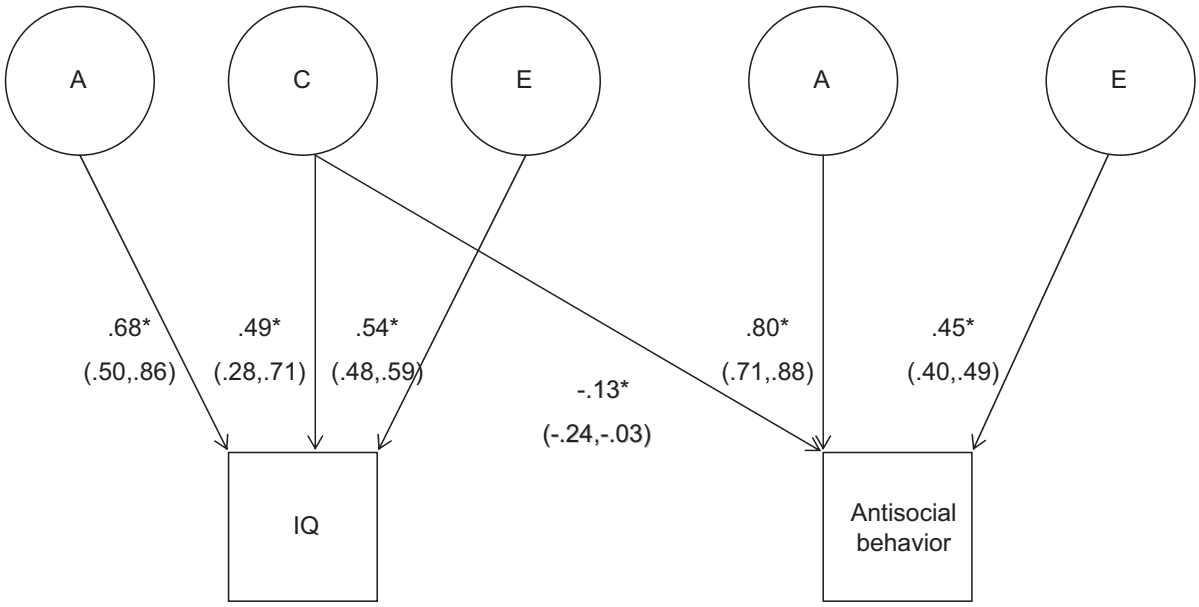

Figure 3. Parameter estimates with 95% confidence intervals for bivariate model of association between IQ and composite antisocial behavior in female twins. The letters A, C, and E refer to additive genetic influences, shared environmental influences, and nonshared environmental (including error) influences, respectively. The model is displayed for Twin 1 only; the model for Twin 2 would look identical. The variances of the latent variables are fixed at 1. All parameter estimates are statistically significant at $p < .05$.

boys for the bivariate analysis. The bivariate model that provided the best fit to the data for boys corresponded to that presented in Figure 2, CFI = 1.00, RMSEA = .00; $\chi^2(20, N = 425) = 17.67$, $p = .61$. Parameter estimates differed only very slightly. Genetic influences in common with IQ accounted for 12% of the variance in antisocial behavior. The remaining variance in antisocial behavior was accounted for by genetic influences (61%) and nonshared environmental influences (27%) unique to antisocial behavior. These results indicate that the etiology of the low IQ–antisocial behavior relation in boys cannot be explained by the ADHD diagnosis. However, the exclusion of children with the ADHD diagnosis further attenuated the already small IQ–antisocial behavior relation in girls.

Discussion

The relation between low IQ and the development of antisocial behavior has been well documented. Our data are consistent with other studies in demonstrating that, for boys, low IQ is associated with antisocial behavior in a dose–response fashion; boys with high levels of antisocial behavior had IQ's that were, on average, 11 points ($SD = 0.73$) lower than those of nonantisocial boys. For boys, low IQ also prospectively predicted relatively higher antisocial behavior over time. Although low IQ was also associated with antisocial behavior in a dose–response fashion for girls, the association was only significant for the girls with the highest levels of antisocial behavior. Such girls had IQ's that were, on average, 5 points ($SD = 0.33$) lower than those of nonantisocial girls, an effect size less than half of that found for boys. Moreover, for girls, low IQ did not significantly predict relatively higher antisocial behavior at age 7 years when age-5 antisocial behavior was controlled. Thus, low IQ had a significantly stronger, cross-sectional, and prospective relation with antisocial behavior in boys than in girls, a finding that replicates the results from at least one other epidemiologic cohort (Moffitt et al., 2001).

For boys, the relation between low IQ and antisocial behavior was due to a shared genetic etiology: 100% of the phenotypic correlation between IQ and a composite of antisocial behavior scores at ages 5 years and 7 years was accounted for by genetic factors that influence both IQ and antisocial behavior. Although environmental experiences such as domestic violence have been associated with low IQ (Koenen et al., 2003) and increased antisocial behavior (Jaffee et al., 2002), these data indicate that the low IQ–antisocial behavior covariation arises from a genetically transmitted process. This process could include genotype–environment correlation or interaction; such effects are included within the proportion of variance explained by genetic influences in most twin studies (Purcell, 2002). This finding means that even when genetic influences explain 100% of the phenotypic correlation, such as they do for low IQ and antisocial behavior in boys, environmental influences may still be important in mediating or moderating genetic effects. For example, low IQ is associated with verbal deficits that may result in children with low IQs misunderstanding rules or having difficulty negotiating conflict with words (Lynam & Henry, 2001). The genetic overlap between IQ and antisocial behavior does not rule out a model whereby the low IQ–antisocial behavior relation is mediated by such deficits.

The genetically mediated association between low IQ and antisocial behavior may indicate that some of the same genes influence variation in both phenotypes in boys. Our results are also consistent with a common neurodevelopmental etiology. Such a process has been proposed by Nigg and Huang-Pollock (2003) in their early-onset model of the role of executive functions and intelligence in conduct disorder. However, the relatively small magnitude of the low IQ–antisocial behavior correlation suggests that most of the neurodevelopmental and genetic influences on variation in each phenotype are not shared.

The results of our bivariate analyses suggest that there are gender differences in the etiology of the overlap between low IQ and antisocial behavior. In boys, the overlap was best accounted for by genetic influences common to both phenotypes. In girls, shared environmental influences explained the overlap. However, we did not have opposite-gender twin pairs in our sample, and, therefore, we could not formally test the gender difference using a gender-limitation model. Furthermore, it is important to note that once children with ADHD diagnoses were excluded from the sample, the low IQ–antisocial behavior correlation in girls was attenuated to almost nonsignificance. These findings suggest that the low IQ–antisocial behavior relation in girls is largely an artifact of comorbid ADHD. Further research is needed to address the question of why IQ and antisocial behavior appear to be linked more strongly and robustly in boys than in girls. Epidemiologic research suggests that early-onset persistent antisocial behavior and a range of early-onset neurodevelopmental disorders are more prevalent in boys than in girls (Rutter, Caspi, & Moffitt, 2003), and neurodevelopmental difficulties such as undercontrolled temperament, inattention, hyperactivity, impulsivity, and low IQ are more strongly associated with antisocial behavior among boys than among girls (Moffitt et al., 2001). Measured genes with connections to antisocial behavior (Caspi et al., 2002; Foley et al., 2004) and some forms of mental retardation (Brunner et al., 1993; Skuse, 2005) are located on the X chromosome. Although it is purely speculative given the present state of the knowledge, perhaps the genetic underpinnings of the low IQ–antisocial behavior relation will be found to be gender linked.

This study's first limitation concerns whether findings from twins can generalize to nontwin populations. The distribution of IQ in our sample of twins ($M = 98$, $SD = 14.4$) is similar to that of singletons ($M = 100$, $SD = 15$; Wechsler, 1990). Furthermore, twins and singletons do not differ in mean levels of behavior problems (Gjone & Novik, 1995; Moilanen et al., 2002). In addition, the correlation between IQ and antisocial behavior in our twin sample ($r = -.19$) is virtually identical to that from representative cohorts throughout the Western world (Moffitt et al., 2001). A second, and related, limitation is that we do not know whether the findings will replicate in other age groups. We focused on young children because this is the developmental period during which antisocial behavior is first manifest. These findings apply only to early-onset antisocial behavior. The association between IQ and antisocial behavior emerging in adolescence may be accounted for by nongenetic factors. A third limitation is that we did not administer full IQ tests, and our results rely on prorated scores that are based on a subset of the full battery. As a result, we are unable to examine whether antisocial behavior is more strongly associated with verbal versus nonverbal components of IQ. Prior studies suggest a stronger association between IQ and verbal intelligence scores (Nigg & Huang-Pollock, 2003), although there are exceptions (Raine et al., 2002). Finally, our study focused solely on the

etiology of the low IQ–antisocial behavior relation. Both low IQ and antisocial behavior are significantly correlated with ADHD (Burt et al., 2001; Kuntsi et al., 2004; Mannuzza et al., 2004; Patterson et al., 2000). The next step for future research will be to examine more broadly the role of low IQ in the relation between antisocial behavior and ADHD and in the etiology of externalizing spectrum disorders (Krueger, Markon, Patrick, & Iacono, 2005; Krueger, Watson, & Barlow, 2005).

The population prevalence of early-onset antisocial behavior that is life-course persistent is low (5% among men, less than 1% among women); however, these individuals account for more than their share of crime (Robins, 1966). Low IQ predicts the chronicity of antisocial behavior (Lahey et al., 1995); therefore, the children in our study who are boys, have low IQs, and have high levels of early antisocial behavior are at high risk for becoming life-course persistent antisocial individuals. This antisocial subtype is at the highest risk for myriad negative outcomes in adulthood, including mental health problems, substance dependence, financial problems, drug-related violent crime, and violence against women and children (Moffitt, Caspi, Harrington, & Milne, 2002). Genetic influences on IQ and antisocial behavior suggest that the parents of these vulnerable children are also likely to have low IQ and to be antisocial. Such parents are at risk for creating family environments that aggravate rather than ameliorate their children's vulnerabilities. Thus, the families of young boys with low IQ who exhibit high levels of antisocial behavior should be targeted for early intervention.

Our finding of shared genetic influences on the relation between IQ and antisocial behavior in boys in no way argues that the developmental course for these children is immutable. Developmental theories of life-course-persistent antisocial behavior propose that trait vulnerability interacts with environmental risk factors to produce antisocial continuity. Thus, reducing environmentally mediated risk factors for low IQ and antisocial behavior is potentially important for altering the developmental course of these vulnerable children.

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