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Revisiting the Association Between Reading Achievement and Antisocial Behavior: New Evidence of an Environmental Explanation From a Twin Study

Kali H. Trzesniewski, Terrie E. Moffitt, and Avshalom Caspi
Institute of Psychiatry, King's College London and University of Wisconsin-Madison

Alan Taylor and Barbara Maughan
Institute of Psychiatry, King's College London

Previous studies have reported, but not explained, the reason for a robust association between reading achievement and antisocial behavior. This association was investigated using the Environmental Risk (E-Risk) Longitudinal Twin Study, a nationally representative 1994–1995 birth cohort of 5- and 7-year-olds. Results showed that the association resulted primarily from environmental factors common to both reading and antisocial behavior and was stronger in boys. Environmental factors also explained the relation between reading disability and conduct disorder. Leading candidate environmental risk factors weakly mediated the association. For boys the best explanation was a reciprocal causation model: poor reading led to antisocial behavior, and vice versa. In contrast, the relation between reading achievement and attention deficit hyperactivity disorder was best explained by common genetic influences.

Research on the relationship between educational difficulties and antisocial behavior has had a long history. Many studies have found that children with educational difficulties are more antisocial, but the field has yet to reach a consensus about the precise cause of this relation. An authoritative review of 17 longitudinal studies concluded that the relation between educational underachievement and antisocial behavior is robust, but also lamented that the cause of the relation remained equivocal (Hinshaw, 1992) and more recent reviews agree (Dionne, 2005; Mandel, 1997). Understanding why young people's educational difficulties go hand in hand with their antisocial behavior has important implications for interventions. If, on the one hand, antisocial behavior problems were responsible for educational difficulties, then treating the behaviors themselves could improve educational difficulties. If, on the other hand, antisocial behavior problems were a response to educational difficulties, then interventions designed to increase academic success could ameliorate antisocial behavior problems. Even prospective longitudinal studies have not yet resolved the issue of which is the cause and which the outcome (Hinshaw, 1992). It is possible that the two are not actually causally related. That is, some common developmental antecedent (i.e., third variable) may cause both educational difficulties and antisocial behavior, and the two may have no direct effect on each other.

The present study offers methodological advances that were either unavailable or called for at the time of Hinshaw's (1992) authoritative review, making it possible to now revisit the question of educational difficulties and antisocial behavior. Using reading achievement as a measure of educational difficulties, we examined both common-developmental-antecedent and causal explanations and assessed empirically which was the best description of the relation between reading achievement and antisocial behavior during early childhood.

The present study extends previous research in three ways: it (1) evaluates three competing hypotheses about the source of the association between reading achievement and antisocial behavior within one study sample, (2) presents nationally representative findings from a birth cohort with data collected at two key developmental points: at the onset of reading tuition at age 5 and follow-up after 2

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Correspondence concerning this article should be addressed to Kali H. Trzesniewski, Department of Psychology, Stanford University, Stanford, CA 94305-2130. Electronic mail may be sent to kali@psych.stanford.edu.

years of tuition, and (3) uses a twin design to allow consideration of a genetic contribution to both reading achievement and antisocial behavior.

Common-Developmental-Antecedent Explanations of the Relation Between Reading Achievement and Antisocial Behavior

At least two distinct common-developmental-antecedent explanations have been advanced. The first is that the reading achievement–antisocial behavior relation is the result of underlying genetic factors common to both. That genes influence both reading achievement and antisocial behavior has been well documented (Gayan & Olson, 1999; Rhee & Waldman, 2002). However, empirical evidence about whether reading achievement and antisocial behavior stem from the same set of genetic influences is lacking. To test whether there is genetic variance common to both constructs, monozygotic (MZ) and dizygotic (DZ) twin correlations are compared across traits: i.e., one twin's reading score is correlated with his co-twin's antisocial behavior score. If the cross-trait cross-twin correlations are greater for MZ than for DZ twins, this implies that genetic factors contribute to the phenotypic correlation between the two traits. This analysis is usually conducted using structural equation modeling or regression models, and requires large sample sizes. To date, and to our knowledge, there have been no twin studies with sufficient power to disentangle genetic from environmental influences on reading achievement and antisocial behavior. However, two smaller twin studies have been conducted. Willcutt and Pennington (2000) found that co-twins of reading disabled children were at higher risk for externalizing problems, suggesting that reading disability and antisocial behavior run together in families. In contrast, Stevenson and Graham (1993) reported that spelling disability and antisocial behavior did not have any common genetic variance; however, they also found no genetic influence on antisocial behavior, a finding that is contrary to a large literature (Rhee & Waldman, 2002). Thus, it is not clear how to interpret these findings. This study will test the common genetic hypothesis within a full behavioral genetic model using a large sample.

Hypothesis 1: The relation between reading achievement and antisocial behavior is attributable to genetic influences that are common to both phenotypes. If so, when the variance that is common to reading achievement and antisocial behavior is separated into its genetic and environmental parts, we will find a significant genetic factor that is common to both.

The second common-developmental-antecedent argument is that reading achievement and antisocial behavior are related because they have common, nongenetic, developmental antecedents that exert an influence on both of them. Early familial environment is the most often proposed candidate for this third-variable hypothesis. Hinshaw (1992) implicated several familial constructs that satisfy the necessary third-variable requirement of being related (at least theoretically) to both reading achievement and antisocial behavior. Several of the developmental antecedents identified in Hinshaw's review were measured in the present study, including age of mother at first birth, family size, social deprivation, socioeconomic status (SES), maternal reading, a stimulating home environment, maternal depression, and child neglect.

This study tests whether these constructs can account for the association between reading achievement and antisocial behavior. If a common developmental antecedent explains the full association, then the partial correlation between reading achievement and antisocial behavior will be nonsignificant once the effects of the common risk factor are taken into account. It is possible that the third variable will only account for a portion of the association, in which case the partial correlation will remain significant, but will be lower than the zero-order correlation. For example, child neglect may be a common developmental antecedent for reading achievement and antisocial behavior. A neglectful environment means little or no monitoring and inconsistent parenting, both of which are related to antisocial behavior (Ary, Duncan, Duncan, & Hops, 1999). In addition, a neglectful environment suggests a lack of intellectual stimulation necessary for promoting readiness for learning to read (e.g., bedtime stories, nursery rhymes that promote phonics awareness, exposure to alphabet books; Bradley, Corwyn, Burchinal, Pipes McAdoo, & Garcia Coll, 2001; Richman, Stevenson, & Graham, 1982). Thus, it is possible that child neglect may explain the relation between reading achievement and antisocial behavior.

Hypothesis 2: The relation between reading achievement and antisocial behavior can be explained by common environmental antecedents. If so, the reading achievement–antisocial behavior relation should be eliminated or markedly reduced when variations in familial background are statistically controlled.

Causal Relations Between Reading Achievement and Antisocial Behavior

Three causal patterns are plausible (e.g., Rutter, Tizard, & Whitmore, 1970). The first is that reading problems precede and cause antisocial behavior. The mechanism of this sequence may be as follows. Reading problems lead to general school failure; school failure, in turn, results in a loss of self-esteem and frustration leading to escape from the adversity of the classroom and associating with other children who are similarly hostile toward school. Disruptive behavior may be a function of frustration, the child's association with others who are prone to delinquency, and/or attempts to regain a measure of self-esteem from peers (Kaplan, 1980). Longitudinal studies have provided some evidence to support this hypothesis, although this evidence is not unequivocal. For example, McMichael (1979) found that low reading readiness at school entry predicted an increase in antisocial behavior nine months later, but reading readiness failed to predict an increase in antisocial behavior at the 21-month follow-up. McGee, Williams, Share, Anderson, and Silva (1986) found that children with a reading disability increased in antisocial behavior from age 5 to age 7; however, the reading disability status was defined at the point of outcome. Stevenson, Richman, and Graham (1985) found that poor language abilities at age 3 predicted increases in antisocial behavior from age 3 to age 8, but reading was not examined. Finally, Bennett, Brown, Boyle, Racine, and Offord (2003) provided the most stringent test of this hypothesis by studying children without a conduct disorder at school entry. They found that children who were poor readers at school entry were more likely than good readers to develop a conduct disorder 2 years later.

The second plausible causal relation is that antisocial behavior precedes and causes reading problems. The mechanism of this sequence may be as follows. Children's externalizing behaviors interfere with their ability to learn. For instance, children who disrupt the classroom pay less attention to the lessons being taught and receive less help from the teacher (Arnold, 1997). There is circumstantial evidence for this hypothesis, because prospective studies have shown that aggression that is present at preschool ages, before children are taught to read, predicts poor reading acquisition. For example, Stott (1981) and Jorm, Share, Matthews, and Maclean (1986) found that antisocial behavior at school entry predicted reading achievement 2 years later. Sanson, Prior, and Smart (1996) found several different causal pathways demonstrating that antisocial behavior at school entry was related to reading problems for some children, but not all. However, these studies did not control for cognitive skills at school entry. McMichael (1979) also found that antisocial behavior at school entry predicted reading achievement 2 years later, but this association did not hold when reading readiness at school entry was controlled.

Alternatively, both causal relations may be true, resulting in a reciprocal relation between reading problems and antisocial behavior. That is, as poor learners become increasingly frustrated, their antisocial behavior increases, which in turn interferes with learning to read, which then creates more antisocial problems, and so on. Evidence of increasing correlations between reading achievement and antisocial behavior with age supports this reciprocal process (e.g., Arnold, 1997).

The present study includes measures of IQ—which serves as an index of general learning ability before reading tuition—and antisocial behavior at the child's entry to formal schooling, as well as measures of reading achievement and antisocial behavior after the child experienced 2 years of tuition. Thus, the three causal predictions can be tested.

Hypothesis 3: Reading achievement and antisocial behavior are causally related. If so, reading difficulties will predict increased antisocial behavior during primary school, after controlling for previous levels of antisocial behavior at school entry, and/or antisocial behavior at school entry will predict reading difficulties, after controlling for previous intellectual ability.

Before testing our three hypotheses, we need to take into account the role of gender and attention deficit hyperactivity disorder (ADHD). First, what role does gender play in the relation between reading achievement and antisocial behavior? Although several studies have shown that both reading problems (e.g., Rutter et al., 2005) and antisocial behavior (Moffitt, Caspi, Rutter, & Silva, 2001) are more common in boys, few studies have tested whether gender moderates the relation between reading achievement and antisocial behavior during the transition to formal schooling. Of those studies that did examine the role of gender, one found no difference between boys and girls (Stevenson et al., 1985) and two found that the relation was stronger for boys (Sanson et al., 1996; Willcutt & Pennington, 2000). These few, inconsistent findings do not provide sufficient evidence for making predictions about gender; therefore, we will begin our analyses

by examining the correlation between reading achievement and antisocial behavior separately for boys and girls.

Second, what is the role of ADHD in the relation between reading achievement and antisocial behavior? It has been suggested that antisocial behavior may be related to reading problems because of the high overlap between antisocial behavior and ADHD (e.g., Hinshaw, 1992). Although the relation between reading problems and ADHD is robust, there is inconsistent evidence for the role of ADHD as a mediator of the relation between reading problems and antisocial behavior. Several studies have found that ADHD completely explains the relation between reading problems and antisocial behavior (e.g., Carroll, Maughan, Goodman, & Meltzer, 2005; Rabiner & Coie, 2000; Willcutt & Pennington, 2000), but others have found that ADHD cannot completely explain the relation between reading problems and antisocial behavior (e.g., McGee et al., 1986; Stevenson & Graham, 1993). We will test whether the reason reading achievement and antisocial behavior are related is because ADHD children tend to display antisocial behavior.

If we find that ADHD does not explain the full relation between reading achievement and antisocial behavior, it will suggest that, although antisocial behavior and ADHD are strongly related, they overlap with reading achievement for different reasons. To explore these potentially independent relations, we will examine the genetic and environmental relations between reading achievement and ADHD symptoms. Specifically, is the relation between reading achievement and ADHD mediated in the same way as the relation between reading achievement and antisocial behavior? In addition, we will examine the extent that these behavioral genetic findings generalize to diagnosable disorders (i.e., to reading disability, to conduct disorder, and to ADHD). Is the comorbidity between reading disability and conduct disorder and between reading disability and ADHD best explained by genetic or environmental effects?

Methods

Participants

Participants are 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 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.

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 (Maynard, 1997; Moffitt & The E-Risk Study Team, 2002). The sampling strategy resulted in a final sample in which one third of Study mothers (younger only) constitute a 160% oversample of mothers who were at high risk based on their young age at first birth (13–20 years). The other two thirds of Study mothers accurately represent all mothers in the general population (aged 13–48) in England and Wales in 1994–1995 (estimates derived from the General Household Survey; Bennett, Jarvis, Rowlands, Singleton, & Haselden, 1996). 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 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-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, 1,116 (93%) participated in home-visit assessments within 2 months of the twins' fifth birthday, 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 parent's 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 18–24 months after the children's age-5 assessment (hereafter called the age-7 follow-up). Follow-up data were collected for 98% of the 1,116 E-Risk Study families, and teacher questionnaires were obtained for 91% of the 2,232 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.

Zygosity was determined using a standard zygosity questionnaire, which has been shown to have 95% accuracy (Price et al., 2000). Ambiguous cases were zygosity typed using DNA. The sample included 55% MZ and 45% DZ twin pairs. All twin pairs were same-sex, and sex was evenly distributed within zygosity (49% male). The sample was 90.6% Caucasian, 4.1% Asian, 1.4% Black, and 3.9% Mixed or Other. Although ethnic minority families were enrolled, recent immigrant mothers who did not speak English at home were not recruited; thus 98% of the families spoke English at home, and all mothers spoke English well enough to be interviewed.

Measures

Children's Ability Measures

Reading was individually tested at age 7 using the Test of Word Reading Efficiency (TOWRE; Torgesen, Wagner, & Rashotte, 1999). The TOWRE provides a quick assessment of sight word efficiency, the number of words on a printed list that a child can accurately identify within 45 s, and provides an index of the size of the child's reading vocabulary ($M = 105.91$, $SD = 13.96$, min = 63, max = 146). This test has been shown to have good validity (Torgesen, et al., 1999), including in the present study where it correlates .63 ($p < .05$) with teacher-reported school performance.

Intelligence was individually tested at age 5 using a short form of the *Wechsler Preschool and Primary Scale of Intelligence-Revised* (WPPSI-R; Wechsler, 1990) comprising Vocabulary and Block Design subtests. IQs were prorated (i.e., the full-scale IQ score was estimated from two subscales) following procedures described by Sattler (1992, pp. 998–1004). The children's IQs ranged from 52 to 145, normally distributed ($M = 98$, $SD = 14$).

Reading disability was defined as having a discrepant reading score in reference to IQ. We measured reading disability through the following steps: (a) fitting a regression model relating reading scores to the child's WPPSI-R IQ scores and computing for each child an expected reading score conditional on IQ and (b) classifying children whose observed reading score was more than 1 *SD* below their reading score predicted on the basis of the WPPSI-R IQ score. This method follows that recommended by Yule, Rutter, Berger, and Thompson (1974), with the exception that Yule et al. recommended a cutoff criterion of 1.5 *SD* units below the predicted scores. We used a 1 *SD* cutoff to ensure sufficient base-rates for statistical analysis; 18.8% of the boys and 10.7% of the girls were identified as reading disabled for research purposes (Rutter et al., 2004). This definition of reading disability is often referred to as specific reading retardation; however, other researchers define reading disability as 1 *SD* below the population mean on reading, regardless of cognitive status (termed general reading backwardness). Our results hold using either definition of reading disability.

Children's Behavior

Antisocial behavior was assessed at ages 5 and 7 with the Achenbach family of instruments (Achenbach, 1991a, 1991b). We combined mother interviews and teacher reports of children's behavior on the Aggression and Delinquency scales by summing the items from each rater (items scored from 0 *not true* to 2 *very or often true*). These scales were supplemented with *Diagnostic and Statistical Manual–IV* (DSM–IV; American Psychiatric Association, 1994) items assessing conduct and oppositional defiant disorder (e.g., "spiteful, tries to get revenge," "uses force to take something from another child"). Scores ranged from 0 to 130 ($M = 21.17$, $SD = 16.27$) at age 5 and from 0 to 132 ($M = 18.48$, $SD = 15.80$) at age 7. Mother and teacher reports of antisocial behavior correlated .29 ($p < .05$) at age 5 and .38 ($p < .05$) at age 7, which is typical in inter-rater studies of children's behavioral problems (Achenbach, McConaughy, & Howell, 1987). The α reliability of the combined score was .94 at age 5 and .95 at age 7.

Conduct disorder diagnosis. We derived a research diagnosis of children's conduct disorder on the basis of mothers' and teachers' reports on 14 of 15 DSM–IV symptoms of conduct disorder. Symptoms covered fighting, bullying, lying, stealing, cruelty to people or animals, vandalism, and rule violations. The "forced sexual activity" symptom was excluded as inappropriate for 5- and 7-year-olds. A child was considered to have a given symptom if either the mother or the teacher reported the symptom as being "very true or often true" of the child over the past 6 months. We counted a symptom as present if reported by either source, following evidence that this approach enhances diagnostic validity. Consistent with DSM–IV criteria, children with three or more symptoms were assigned a conduct disorder diagnosis (details in Kim-Cohen et al., 2005). The prevalence of this conduct disorder diagnosis up to age 7 was 11.5% for boys and 4.6% for girls.

ADHD symptoms were assessed at age 7. We combined mother interviews and teacher reports of children's behavior by summing the items from each

rater (items scored from 0 to 2). Children's symptomatology was measured with 18 items concerning inattention, impulsivity, and hyperactivity derived from the DSM–IV diagnostic criteria for ADHD and Rutter Child Scales (e.g., "inattentive, easily distracted," "impulsive, acts without thinking," "very restless, has difficulty staying seated for long"; Sclare, 1997). Symptoms were reported for the preceding 6 months. Scores ranged from 0 to 67 ($M = 13.70$, $SD = 11.17$). Mother and teacher reports of ADHD symptoms correlated .28 ($p < .05$). The α reliability of the combined score was .93.

ADHD diagnosis. We derived a research diagnosis of children's ADHD based on DSM–IV criteria. Children received the diagnosis if they had six or more of the hyperactivity-impulsivity symptoms and/or 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 of either inattentiveness or hyperactivity–impulsivity. Therefore, the diagnostic criteria included the presence of symptoms in more than one setting (home and school; details in Kuntsi et al., 2004). Symptoms were counted as present only if scored "very true or often true." The prevalence of ADHD diagnoses up to age 7 was 11.1% for boys and 4.6% for girls.

Control Variables

Age at first birth was collected for all mothers. Ages of mothers at the birth of the first child ranged from 13 to 42 ($M = 25.64$, $SD = 5.81$).

Family size was collected for all families. The number of siblings when the twins were age 5 ranged from 0 to 10 ($M = 1.21$, $SD = 1.21$).

Socioeconomic deprivation is a count of seven socioeconomic disadvantages, measured at age 7, which were defined as follows: (1) head of household has no educational qualifications; (2) head of household is employed in an unskilled occupation or is not in the labor force; (3) total household gross annual income is less than £10,000; (4) family receives at least one government benefit, excluding disability benefit; (5) family housing is government subsidized; (6) family has no access to a vehicle; and (7) family lives in the poorest of six neighborhood categories, in an area dominated by government-subsidized housing, low incomes, high unemployment, and single-parent families (Kim-Cohen, Moffitt, Caspi, & Taylor, 2004). Summing across these seven items yielded a composite index of socioeconomic disadvantage, ranging from 0 to 7 ($M = 1.19$, $SD = 1.71$). α reliability was .79.

SES. A standardized composite of income, education, and social class was used to measure SES at age 5. The three SES indicators were highly correlated (rs ranged from 0.57 to 0.67, all ps $< .05$) and loaded significantly onto one latent factor (factor loadings = 0.80, 0.70, and 0.83 for income, education, and social class, respectively).

Mothers' reading. Mothers were administered the reading subtest of the Wide Range Achievement Test (WRAT-3; Wilkinson, 1993) when their children were aged 5 years. This test measures single-word reading. On average, the mothers read at the high school level ($M = 47.11$, $SD = 6.44$, range 7–57, primary school to college).

Maternal depression. When the twins were 5 years, mothers' major depressive disorder was assessed using the Diagnostic Interview Schedule (Robins, Cottler, Bucholz, & Compton, 1995) according to DSM–IV criteria; 26% of mothers had major depressive disorder during the twins' first 5 years of life (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005).

Child neglect was assessed by trained research workers as part of a Coder's Inventory completed at the end of the home visit. The Coder's Inventory comprised the "Home Observation for Measurement of the Environment"—Elementary HOME Inventory (Bradley & Caldwell, 1977; Caldwell, 1984), an instrument designed specifically to measure the home environment and familial interactions of families with children between the ages of 6 and 10 years old; items from the University of Washington Parenting Clinic questionnaire (Webster-Stratton, 1998); and additional items designed by the E-Risk Study team. The child neglect scale included six items (scored from 0 to 2) and measured the extent to which each twin's physical and emotional needs appeared to be neglected. Items included "The twin is well-nourished (reverse-coded)," "The parent monitors the twin appropriately (reverse-coded)," "The twin is neglected," "The twin lacks attention to personal hygiene," "The parent is aware of the twin's needs (reverse-coded)," and "The parent is emotionally supportive of the twin (reverse-coded)." Scores ranged from 0 to 12 ($M = 0.73$, $SD = 1.51$) and high scores reflected more neglect. The internal consistency was .74 and the inter-rater reliability was .76.

Stimulating environment was assessed using the HOME Inventory. The stimulating environment scale included six items (scored from 0 to 2) and measured the extent to which the home environment of the twins was intellectually stimulating. Items included "The twins' room(s) are nicely decorated," "The twins have toys and puzzles," "The twins have

books," "The twins have a radio/tape/musical instrument," "The twins' art is displayed in the home," and "The family encourages the twins to have hobbies." Scores ranged from 0 to 12 ($M = 9.21$, $SD = 2.95$) and high scores reflected a more stimulating environment. The internal consistency was .81 and the inter-rater reliability was .89.

Results

What Is the Relation Between Reading Achievement and Antisocial Behavior?

We first tested whether the well-documented association between reading achievement and antisocial behavior would replicate in the E-Risk sample. Age 7 reading achievement correlated $-.23$ ($p < .05$) with age 7 antisocial behavior. For all analyses reported, significance levels have been adjusted using the sandwich or Huber–White variance estimator (Rogers, 1993; Williams, 2000) to correct for the interdependence of twin data. In addition, all statistics reported have been weighted to be representative of the UK population of women having children in the 1990s.

We next tested whether the association between reading achievement and antisocial behavior was the same across gender. The correlation within boys ($r = -0.27$, $p < .05$) was significantly stronger ($z = -2.23$, $p < .05$) than the correlation within girls ($r = -0.14$, $p < .05$). This finding is consistent with previous research that has found a stronger relation between reading achievement and antisocial behavior for boys (Sanson et al., 1996; Willcutt & Pennington, 2000). The significant difference in the correlations for reading achievement and antisocial behavior suggests the analyses should be conducted separately for boys and girls. Moreover, the correlation for girls is considered too small for decomposing into subparts, which could yield unstable estimates from our planned behavioral genetic and causal models. Therefore, we focused our analyses on reading achievement and antisocial behavior on boys only ($n = 1{,}092$ twins; 1,058 pairs with complete data). However, we will revisit the girls later in this report.

Explaining the Relation Between Reading Achievement and Antisocial Behavior

Previous research has suggested that reading achievement and antisocial behavior are related because ADHD children tend to display antisocial behavior (e.g., Hinshaw, 1992). We tested this by examining the correlation between reading achievement and antisocial behavior after removing the boys in the sample who have a research diagnosis of ADHD ($n = 140$ boys). We found that the correlation between reading achievement and antisocial behavior was reduced slightly when ADHD boys were removed from the analysis; however, the correlation remained significant ($r = -.21$, $p < .05$). Therefore, the relation between reading achievement and antisocial behavior cannot be entirely explained by comorbidity between antisocial behavior and ADHD.

Hypothesis 1: Reading achievement and antisocial behavior share a common genetic etiology. Hypothesis 1 states that there is no causal relation between reading achievement and antisocial behavior; instead, the relation is due to common genetic influences. Before testing for common genetic factors, we needed to check that both reading achievement and antisocial behavior are genetically influenced in this sample. Thus, this section begins with univariate behavioral genetic analyses and then moves on to bivariate behavioral genetic analyses.

Analyses overview. The logic behind quantitative genetic analyses has three parts (Neale & Cardon, 1992; Plomin, DeFries, McClearn, & McGuffin, 2001; Rijsdijk & Sham, 2002). First, MZ twins share all their genes but DZ twins, like ordinary full siblings, share on average only half of their polymorphous genes. As such, when the similarity of MZ twins is greater than the similarity of DZ twins, this indicates a genetic contribution to behavior. In model fitting, this yields a significant variance component called A (additive genetic variance). Second, MZ twins' genetic similarity is twice that of DZ twins'; therefore, if only genes were influencing their behavior, MZ twins' behavior should be twice as similar as DZ twins'. If not, this indicates that the environments the boys share in common have enhanced their similarity. In model fitting, this yields a significant variance component called C (common or shared environmental variance). Third, if MZ twins, despite sharing all their genes, are not perfectly identical in their behavior, this indicates that experiences unique to each twin, and not shared with their co-twin, have reduced the twins' behavioral similarity. In model fitting, this yields a significant variance component called E (child-specific environmental variance, which also includes measurement error). Models are fitted to the MZ and DZ covariance matrices using Mx (Neale, Boker, Xie, & Maes, 1999).

Univariate analyses. The MZ and DZ within-pair correlations (Table 1) provide rough estimates of the extent to which genetic, shared environmental, and child-specific environmental factors contribute to reading achievement and antisocial behavior in childhood. The greater MZ than DZ correlations for

Table 1
Pearson Correlations: Reading Ability and Antisocial Behavior for Boys

	Twin 1 reading	Twin 1 antisocial behavior	Twin 2 reading	Twin 2 antisocial behavior
MZ twins				
Twin 1 reading	1.00			
Twin 1 antisocial behavior	− 0.29	1.00		
Twin 2 reading	0.88	− 0.30	1.00	
Twin 2 antisocial behavior	− 0.27	0.75	− 0.29	1.00
Mean (*SD*)	103.60 (13.34)	22.60 (18.90)	103.56 (13.17)	21.68 (18.26)
DZ twins				
Twin 1 reading	1.00			
Twin 1 antisocial behavior	− 0.25	1.00		
Twin 2 reading	0.59	− 0.14	1.00	
Twin 2 antisocial behavior	− 0.29	0.38	− 0.25	1.00
Mean (*SD*)	106.27 (13.08)	22.46 (18.53)	106.11 (12.70)	20.05 (16.47)

MZ = monozygotic; DZ = dizygotic.
Note. $N = 285$ male MZ pairs and 244 male DZ pairs.

reading (MZ = 0.88 vs. *DZ* = 0.59) and antisocial behavior for boys (MZ = 0.75 vs. DZ = 0.38) indicate substantial genetic effects on both phenotypes.

The proportion of variance in reading achievement accounted for by additive genetic effects was 55% (95% confidence interval [CI] 41–71%), by shared environmental factors was 32% (CI = 16–46%), and by child-specific environmental factors was 13% (CI = 11–16%; $\chi^2 = 0.80$, $df = 3$, $p = .85$; AIC = − 5.204; RMSEA = .00). The proportion of variance in antisocial behavior accounted for by additive genetic effects was 68% (CI = 47–78%), by shared environmental factors was 6% (CI = 0–25%), and by child-specific environmental factors was 26% (CI = 22–32%; $\chi^2 = 5.53$, $df = 3$, $p = .14$; AIC = − 0.47; RMSEA = .04). These analyses showed that reading achievement and antisocial behavior were both influenced by genetic factors for boys. A bivariate model can test whether the same genetic factors are influencing both constructs.

Bivariate analyses. In the bivariate twin analysis, MZ and DZ correlations are compared across traits: i.e., one twin's reading score is correlated with his co-twin's antisocial behavior score. If the cross-trait cross-twin correlations are greater for MZ than for DZ twins, this implies that genetic factors contribute to the phenotypic correlation between the two traits. The correlation between reading achievement and antisocial behavior for MZ twins ($r = -.28$, $p < .05$) was about equal to the correlation for DZ twins ($r = -.21$, $p < .05$), suggesting little or no genetic effect common to reading achievement and antisocial behavior. Table 1 shows the twin correlations, by zygosity for both twins, that were entered into the bivariate model.

Figure 1 shows the results of the bivariate model that was fitted to the data (the data were fitted using Cholesky parametrization; however, for ease of interpretation, the results were transformed to the correlated factors models presented in Figures 1 and 4; Loehlin, 1996). The genetic correlation (r_A) indicates the extent to which genetic influences on one trait overlap with those on the second trait (regardless of the traits' individual heritabilities). On the basis of the genetic correlation and the individual heritability of each trait, the extent to which shared genetic influence generates a phenotypic correlation between two traits can be estimated. Similarly, the shared environmental correlation (r_C) and the child-specific environmental correlation (r_E) indicates the extent to which shared environmental influences and child-specific environmental influences on one trait overlap with those on the second trait.

Results from the full ACE bivariate model can be used to estimate the proportion that genetic, shared environmental, and child-specific environmental factors contribute to the phenotypic correlation between reading achievement and antisocial behavior ($r = -0.27$). Path r_A in Figure 1 is the correlation between the genetic variance for reading achievement and the genetic variance for antisocial behavior. Paths h_A and h_B are the paths for the additive genetic variance for reading achievement and antisocial behavior, respectively (squaring h_A and h_B will give the percentage of variance due to additive genes). The amount of genetic variance that is common to reading achievement and antisocial behavior can be estimated by multiplying together the additive genetic paths for reading achievement (h_A) and antisocial behavior (h_B), and the correlation

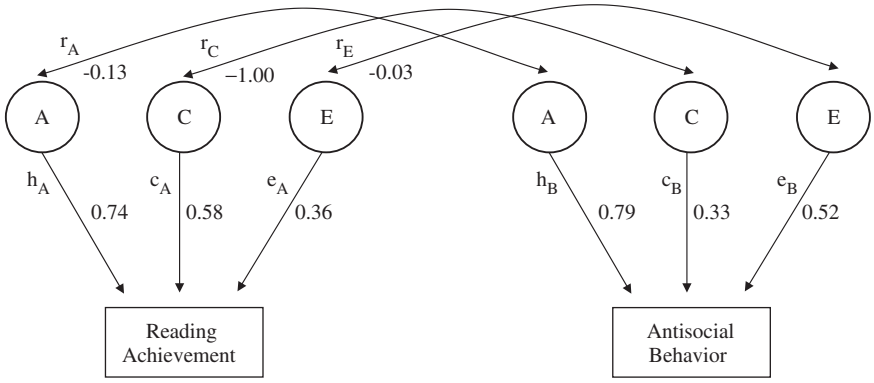

Figure 1. Correlated factors genetic model for the association between reading achievement and antisocial behavior for boys. *Note.* $\chi^2 = 11.60$ ($df = 11$), $p = .39$, RMSEA = .01. A = additive genetic, C = shared environment, E = child-specific environment, r_A = genetic correlation, r_C = shared environmental correlation, r_E = child-specific environmental correlation.

between the two (r_A). The proportion of the phenotypic correlation that is accounted for by genetic influences can be estimated by dividing the resultant score by the phenotypic correlation. The same estimates can be calculated for shared environmental and child-specific factors.

Additive genetic factors shared by reading achievement and antisocial behavior accounted for 27% of the phenotypic correlation [$(0.74 \times 0.79 \times -0.13)/-0.27$]. Environmental factors shared by reading achievement and antisocial behavior accounted for 71% of the phenotypic correlation [$(0.58 \times 0.33 \times -1.0)/-0.27$]. Child-specific factors shared by reading achievement and antisocial behavior accounted for 2% of the phenotypic correlation [$(0.36 \times 0.52 \times -0.03)/-0.27$]. Thus, the majority of the phenotypic correlation for boys was attributable to shared environmental influences that are common to reading achievement and antisocial behavior (see Figure 2).

We also conducted model fitting tests to determine the most parsimonious univariate and bivariate models. The most parsimonious univariate model for reading achievement was an ACE model and that for antisocial behavior was an AE model. For the bivariate model, the most parsimonious model was a C model for the variance that is common to both reading achievement and antisocial behavior.

Hypothesis 2: Reading achievement and antisocial behavior are related because they share a common, nongenetic, developmental antecedent. In the previous section, we found that the association between reading achievement and antisocial behavior for boys was mainly due to shared environmental influences that are common to both. Previous theoretical work suggests several constructs that may account for this shared environmental influence. Table 2 (columns 2 and 3) shows that all of the hypothesized developmental antecedents are significantly related to both reading achievement and antisocial behavior.

We tested whether these developmental antecedents could account for the association between reading achievement and antisocial behavior by comparing the zero-order correlations with the partial correlations. Table 2 (column 4) shows that none of the antecedents accounted for the full association between reading achievement and antisocial behavior. That is, the partial correlations were all significantly different from zero. Growing up in a stimulating environment accounted for the most common variance, reducing the relation between reading achievement and antisocial behavior from $-.27$ ($p < .05$) to $-.18$ ($p < .05$; $t = 6.31$, $p < .05$). Each of the developmental antecedents explained a small part of the relation between reading achievement and antisocial behavior, but a significant amount of

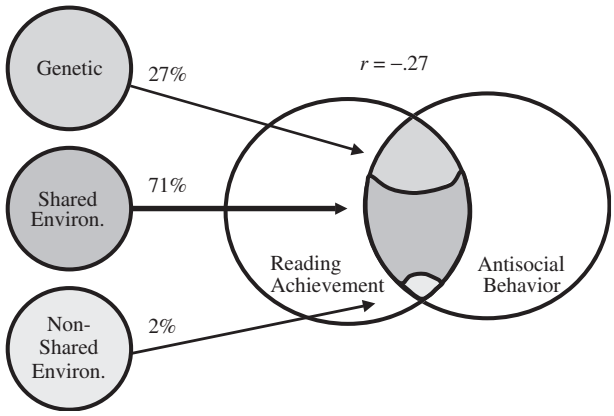

Figure 2. Pictorial representation of the association between reading achievement and antisocial behavior for boys.

Table 2
Zero-Order Correlations Between Risk Factors and Reading Achievement/Antisocial Behavior, and Partial Correlations Between Reading Achievement and Antisocial Behavior, Controlling for Risk Factor

Risk Factor	Reading achievement	Antisocial behavior	Partial correlation between reading and antisocial behavior, controlling for risk factor
Age first birth	0.27*	−0.22*	−0.22*
Family size	−0.17*	0.11*	−0.26*
Social deprivation	−0.28*	0.24*	−0.22*
Socioeconomic status	0.36*	−0.20*	−0.21*
Maternal reading	0.30*	−0.17*	−0.23*
Stimulating environment	0.36*	−0.29*	−0.18*
Maternal depression	−0.11*	0.19*	−0.26*
Child neglect	−0.28*	0.30*	−0.20*

Note. The zero-order correlation between reading achievement and antisocial behavior is −0.27 ($p < .01$).
*$p < .01$.

the association remained unexplained. Indeed, even combining all the developmental antecedents into one analysis failed to explain all of the association: the partial correlation controlling for all eight constructs was −.16 ($p < .05$).

Hypothesis 3: Reading achievement and antisocial behavior are causally related. We found only partial support for common-developmental-antecedent Hypotheses 1 and 2; therefore, much of the association between reading achievement and antisocial behavior remained unexplained. It is possible that the association can be further explained through a causal model. To test the causal hypothesis, we asked if reading achievement predicts antisocial behavior, controlling for initial levels of antisocial behavior, and if antisocial behavior predicts reading achievement controlling for initial intellectual ability.

We tested these causal models using structural equation modeling with Mplus (Muthén & Muthén, 1998; Figure 3). First, we found that the correlation between age 5 IQ and age 7 reading achievement was .42. This is similar to findings on the stability of reading achievement from age 5 to age 7 ($r = .53$; Rabiner & Coie, 2000), suggesting that at this age, IQ can serve as a proxy for the ability to learn to read. Second, we found that being antisocial predicted the acquisition of age 7 reading skill, controlling for age 5 general cognitive ability ($\beta = -.12$, $p < .05$). Thus, compared with nonantisocial boys, antisocial boys acquired less reading skill, even after controlling for general cognitive ability at the point when reading tuition began. Third, we found that IQ (our proxy for reading achievement) predicted the emergence of antisocial behavior between the ages of 5 and 7. The effect of antisocial behavior at age 7 remained significant even after controlling for age 5 antisocial behavior ($\beta = -.08$, $p < .05$). Thus, compared to boys with better reading potential, those with less potential had higher antisocial behavior scores at age 7, even after controlling for the continuity of antisocial behavior from ages 5 to 7. In sum, we found support for both hypotheses, suggesting that reading achievement and antisocial behavior are reciprocally related.

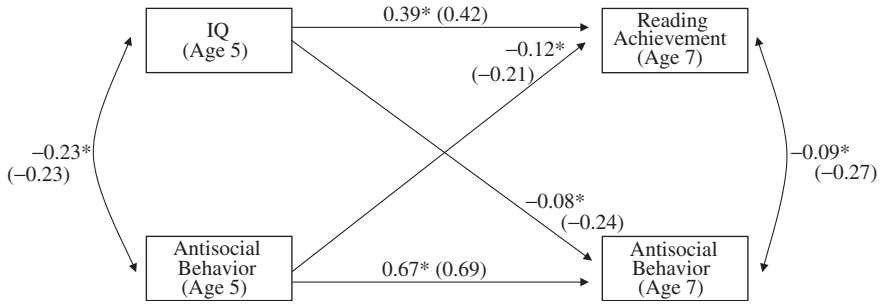

Figure 3. Cross-lag model of IQ/reading achievement and antisocial behavior for boys. *Note*. This is a saturated model with a χ^2 of 0 and zero degrees of freedom. Values in parentheses are zero-order correlations; values not in parentheses are standardized βs.

Do the Results for Boys Generalize to Girls?

We now return to the small association between reading achievement and antisocial behavior in girls to ask whether gender moderated the results for each of our hypotheses. That is, did our analyses show the same pattern for girls and boys?

For Hypothesis 1 (Is the association all genetic?), we began by examining the cross-trait cross-twin correlations for girls. Similar to the boys, we found that the MZ and DZ correlations were essentially equal ($-.18$ and $-.12$ for MZ and DZ twins, respectively), suggesting the findings would not differ for girls, i.e., common shared environmental variance would also explain the overlap between reading achievement and antisocial behavior in girls. Unpacking this variance for girls only would result in fragile findings, because the association is small. Thus, instead of repeating the analyses for girls only, we tested whether gender moderated the bivariate findings using a two-group structural equation modeling approach in Mplus (Muthén & Muthén, 1998). We found that girls showed the same general pattern of findings as boys; however, the two groups could not be equated ($\Delta\chi^2 = 149.38$, $df = 9$). The results from the model that allowed the findings to vary by gender showed that for girls, the correlation between reading achievement and antisocial behavior was explained by 36% A, 54% C, and 10% E. Thus, common environmental factors explained less of the association, and common nonshared factors explained more of the association for girls than boys (for boys the correlation was explained by 27% A, 71% C, and 2% E), but for both boys and girls, most of the correlation was explained by common environmental factors. We also tested whether gender moderated any of the findings for Hypotheses 2 and 3. For Hypothesis 2 (common environmental antecedents), we used multiple regression to test for an interaction between gender and each of the common antecedents. The gender interaction accounted for less than .5% of the variance for each of the antecedents; thus, we found no evidence that the findings for Hypothesis 2 varied by gender. For Hypothesis 3, we again used a two-group structural equation modeling approach. Again, the two groups could not be equated ($\Delta\chi^2 = 36.68$, $df = 6$). Specifically, we found that the cross-path from IQ at age 5 to antisocial behavior at age 7 varied by gender ($\beta = -.02$ for girls and $-.08$ for boys), but the cross-path from antisocial behavior at age 5 to reading achievement at age 7 did not differ by gender ($\beta = -.09$ for girls and $-.12$ for boys). Thus, the reciprocal relation found for boys did not generalize to girls. For girls, it appears that the direction of the effect is from antisocial behavior to reading achievement and not vice versa.

Does the Relation Between Reading Achievement and ADHD Have the Same Origins as the Relation Between Reading Achievement and Antisocial Behavior?

In this cohort, boys' ADHD symptoms correlated highly with antisocial behavior ($r = .70$, $p < .05$) and, like antisocial behavior, ADHD was negatively correlated with reading achievement ($r = -.38$, $p < .05$). This suggested that the reason ADHD is related to reading achievement may be similar to the reason antisocial behavior is related to reading achievement. However, the cross-trait, cross-twin correlation between reading achievement and ADHD symptoms for MZ twins was about twice as large as the correlation for DZ twins (rs $= -.36$ and $-.19$ for MZ and DZ twins, respectively). This pattern suggested that there were genetic influences common to reading achievement and ADHD.

The results of the bivariate analysis using Mx (Neale et al., 1999) are shown in Figure 4. Using the method described above to estimate the bivariate variances, we found that additive genetic factors shared by reading achievement and ADHD accounted for 90% of the phenotypic correlation [$(0.79 \times 0.87 \times -0.50)/-0.38$]. Shared environmental factors shared by reading achievement and ADHD accounted for 0% of the phenotypic correlation [$(0.50 \times 0.00 \times 0.00)/-0.38$]. Child-specific factors shared by reading achievement and ADHD accounted for 10% of the phenotypic correlation [$(0.36 \times 0.49 \times -0.21)/-0.38$]. Thus, although environmental influences primarily explained the association between reading achievement and antisocial behavior, genetic influences primarily explained the association between reading achievement and ADHD. (Further details about genetic and environmental influences on ADHD in this sample are available in Kuntsi et al., 2004.)

We tested the robustness of these independent pathways between reading achievement and antisocial behavior and reading achievement and ADHD symptoms by examining the extent to which these results generalized to diagnosable disorders. We computed the proband-wise cross-concordance rates for boys for research diagnoses of reading disability and conduct disorder and reading disability and ADHD (see McGue, 1992, for concordance rate formulas). The cross-concordance rates for reading achievement and conduct disorder were essentially equal for MZ twins (33%) and DZ twins (23%), suggesting that genetic influences do not contribute

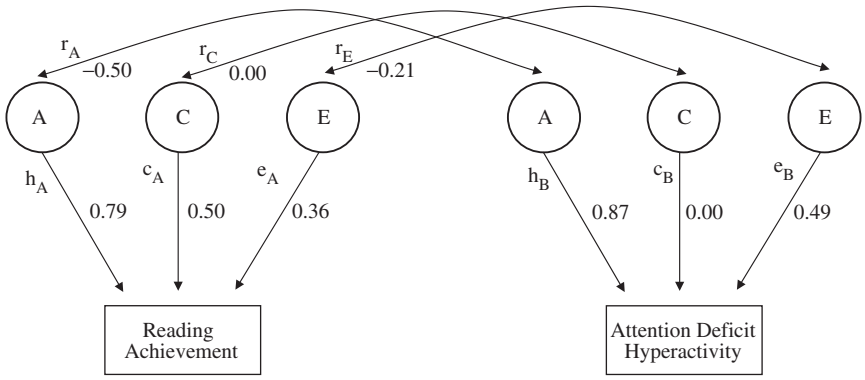

Figure 4. Correlated factors genetic model for the association between reading achievement and attention deficit hyperactivity disorder for boys. *Note*. $\chi^2 = 13.27$ ($df = 12$), $p = .35$, RMSEA = .00. A = additive genetic, C = shared environment, E = child-specific environment, r_A = genetic correlation, r_C = shared environmental correlation, r_E = child-specific environmental correlation. A bidirectional path between attention deficit hyperactivity for Twin 1 and attention deficit hyperactivity for Twin 2 has been modeled to control for a contrast effect ($r = -.05$), but is not shown for ease of presentation.

significantly to the comorbidity of reading disability and conduct disorder. The cross-concordance rates for reading achievement and ADHD were stronger for MZ twins (32%) than for DZ twins (10%), suggesting that genetic influences contribute significantly to the comorbidity of reading disability and ADHD.

We fitted bivariate liability threshold models to the tetrachoric correlations (Falconer, 1965; Neale et al., 1999; Thapar, Harrington, & McGuffin., 2001). To test whether genetic factors accounted for the association between reading disability and conduct disorder and that between reading disability and ADHD, we evaluated the significance of the term associated with the genetic correlation between reading disability and these disorders. The cross-twin, cross-trait tetrachoric correlations between conduct disorder and reading disability were .27 for MZ twins and .25 for DZ twins. The cross-twin, cross-trait tetrachoric correlations between ADHD and reading disability were .34 for MZ twins and .14 for DZ twins. Similar to the findings observed using continuously distributed phenotypes, we found that genetic influences did not contribute to the association between reading disability and conduct disorder, $\chi^2(1) = .01$. $p = .91$, but they did contribute to the association between reading disability and ADHD, $\chi^2(1) = 4.17$, $p < .05$.

Discussion

We found that the relation between boys' reading achievement and antisocial behavior is primarily due to environmental factors that are common to both. Leading candidates for this common environmental risk were tested: measures tapped stimulating home environment, child neglect, mother's reading skill, parental income, education, social class, deprivation, family size, maternal depression, and young maternal age. However, even in combination these only weakly mediated the association between reading achievement and antisocial behavior. Instead, the primarily environmental overlap between reading achievement and antisocial behavior appears to reflect unfolding reciprocal influences of reading achievement and antisocial behavior on each other over time. That is, the development of reading achievement and antisocial behavior are intertwined: as one changes, so does the other. This finding for antisocial behavior stands in contrast to the account for the overlap between reading achievement and ADHD; the relation between reading achievement and ADHD is best explained by genetic influences that are common to both.

In response to Hinshaw's (1992) influential review in which he concluded that ADHD is a more robust predictor of reading problems than antisocial behavior, researchers turned their attention to ADHD and began abandoning research focused on the role of antisocial behavior in the development of reading achievement. Our findings also showed that ADHD is closely related to reading achievement; however, they also suggested that antisocial behavior was an important predictor of reading problems, even after taking into account the comorbidity between ADHD and conduct problems. This may appear surprising given the high correlation between ADHD symptoms and antisocial behavior; however, a correlation of .70 means that 50% of the variance in antisocial behavior is *not* shared with ADHD symptoms. Moreover, our research suggested that despite the strong correlation between ADHD symptoms and antisocial behavior, the two had independent effects on reading achievement. Specifically, we found that the reason antisocial behavior was related to reading

achievement was because of environmental factors they had in common. In contrast, the reason ADHD was related to reading achievement was because of genetic factors they had in common. These results show that antisocial behavior and ADHD symptoms should not be considered equal, at least in terms of their relation with reading achievement.

Implications for Theory

Our findings suggest that contemporary developmental psychopathology theories that posit that the association between reading achievement and antisocial behavior is due to genetic influences only tell part of the story. Research focused on understanding the association between reading achievement and antisocial behavior may be better directed toward environmentally mediated effects. However, our findings suggest that genetic influences are important for explaining the association between reading achievement and ADHD. This finding replicates previous quantitative genetic studies (Gilger, Pennington, & DeFries, 1992; Light, Pennington, Gilger, & DeFries, 1995; Willcutt et al., 2003; Willcutt, Pennington, & DeFries, 2000). It suggests that molecular studies of candidate genes for ADHD might also prove fruitful for understanding reading achievement, whereas any search for candidate genes for antisocial behavior is not likely to be informative for reading achievement, and vice versa.

Our findings cannot rule out all biological explanations for the association between reading achievement and antisocial behavior; biological differences that are not genetic in origin may play a role. Several studies have found differences in the brain between children with a reading disorder and those without and between children with a conduct disorder and those without (see Nigg & Huang-Pollock, 2003 and Vellutino, Fletcher, Snowling, & Scanlon, 2004 for reviews). For example, right-handed children with dyslexia have been found to have reduced left hemispheric capacity and the same has been found for delinquent youths, suggesting that cerebral dysfunction may contribute to the association between reading achievement and antisocial behavior. Our findings do not rule out this possibility, but instead suggest that any neurobiological factors common to reading achievement and antisocial behavior may not be inherited.

Implications for Policy

Our findings suggest two main implications for educational practices. First, the finding that the association is reciprocally influenced and present during the first few years of formal schooling suggests that the association can be broken up by intervening before school begins. Second, the finding that boys' ability and behavior when they enter school influences changes in their reading achievement and antisocial behavior after 2 years of instruction suggests that the association can be broken up by intervening in the early stages of school. Although this is not necessarily easy to achieve, several studies illustrate the positive effect academic interventions can have on children's antisocial behavior. For example, children who participated in the High/Scope Perry Preschool program aimed at enhancing intellectual skills at ages 3 and 4 had lower levels of antisocial behavior and fewer felony arrests than controls at age 23 (Schweinhart & Weikart, 1997). Similarly, children who participated in the Chicago Child–Parent Center Program had lower rates of juvenile arrests and violent arrests 15 years after the intervention (Reynolds, Temple, Robertson, & Mann, 2001) and children who participated in a Mastery Learning intervention showed lower levels of aggressive behavior (Kellam, Mayer, Robok, & Hawkins, 1998). Kellam et al. (1998) also examined the effect of an intervention designed to reduce aggression (i.e., the Good Behavior Game) on reading achievement, but failed to find an effect of the intervention on reading. Thus, to the best of our knowledge, it is not yet known if interventions for aggression can improve reading.

Implications of Gender Differences

We found that the association between reading achievement and antisocial behavior is stronger for boys than for girls. This suggests that interventions aimed at improving reading achievement through improving behavior problems, or vice versa, will likely be more beneficial for boys. For the most part, we found the same pattern of relations for girls, suggesting that these intervention programs may also have a small benefit for girls. However, given the low prevalence of reading problems and behavior problems in girls, it may be more cost effective to focus these interventions on boys. Moreover, given the stronger effect of prior antisocial behavior on future reading achievement and the lack of effect of prior intellectual ability on future antisocial behavior, programs for girls should be more focused on changing antisocial behavior than changing intellectual ability. However, few studies have focused on this association in girls; thus, more research is needed before intervening. In addition, future re-

search may benefit from examining other forms of aggression, such as relational aggression that is more typically seen in girls. Our results may have been attenuated because of our focus on overt aggression, which is less common in girls.

Strengths and Limitations

The present study has several strengths. First, it is based on a large sample and its findings represent the UK population. Second, it uses a genetically sensitive design. Previous research has suggested that the association between reading achievement and antisocial behavior may be genetically mediated, but this hypothesis had not been tested using a large-scale, genetically sensitive design. Third, it uses a longitudinal design with the key constructs measured at two time points, to test reciprocal causation. Few longitudinal studies have been used to test reciprocal influences over time. Finally, it captures the beginning of formal schooling, a key developmental transition for both the onset of reading ability and antisocial behavior.

The present study has several limitations. First, the children in our sample are twins. Although language development is delayed in twins (Rutter & Redshaw, 1991), the average reading ability of the twins in our sample was equivalent to the TOWRE norms based on a sample of singletons. Moreover, twins and singletons do not differ in mean levels of behavior problems (Gjone & Novik, 1995; Kendler, Martin, Heath, & Eaves, 1995; Levy, Hay, McLaughlin, Wood, & Waldman, 1996; Simonoff et al., 1997; van den Oord, Koot, Boomsma, Verhulst, & Orlebeke, 1995), and the association between reading achievement and antisocial behavior in our twin sample is similar to that observed in singleton samples (Hinshaw, 1992). Second, the reading test we used is limited, providing only a measure of word recognition. Third, we did not measure behavior problems and language as they first emerged during the first 2 years of life, but there appears to be an environmental contribution to their overlap at this early age as well (Dionne, Tremblay, Boivin, Laplante, & Perusse, 2003; Plomin, Price, Eley, Dale, & Stevenson, 2002). Fourth, we could not rule out all common developmental antecedents; however, we tested a wide range of constructs and found they explained very little of the association between reading achievement and antisocial behavior, even combined. Fifth, the models tested were not fully reciprocal (i.e., IQ, not reading, was used as an index of age 5 learning ability). However, reading at age 5 would have a restriction of range because most children have yet to be taught how to read. Sixth, the association between reading achievement and antisocial behavior was examined only from age 5 to age 7. Previous research suggests that the association becomes stronger with age (Arnold, 1997). It is possible that as children get older, genetic influences that are common to both reading achievement and antisocial behavior may come to explain more of the association. Alternatively, the reciprocal environmental influences we identified may accumulate strength. These opposing hypotheses should be tested.

Conclusion

This study helps move the field forward by showing that the association between young boys' reading achievement and their antisocial behavior is primarily environmentally mediated and probably explained by a reciprocal process that unfolds over time. If replicated, these findings may help guide interventions by showing that targeting either reading achievement or antisocial behavior during the preschool and early primary school years is likely to produce changes in both behaviors.

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