Published in final edited form as:
Dev Psychol. 2009 July ; 45(4): 973–987. doi:10.1037/a0016225.

School Performance and Genetic and Environmental Variance in Antisocial Behavior at the Transition from Adolescence to Adulthood

Wendy Johnson[1,2], **Matthew K. McGue**[2], and **William G. Iacono**[2]

Wendy Johnson: ; Matthew K. McGue: mcgue001@umn.edu; William G. Iacono: wiacono@tfs.psych.umn.edu

[1] Department of Psychology, University of Edinburgh

[2] Department of Psychology, University of Minnesota - Twin Cities

Abstract

Antisocial behavior increases in adolescence, particularly among those who perform poorly in school. As adolescents move into adulthood, both educational attainment and the extent to which antisocial behavior continues have implications for their abilities to take on constructive social roles. We used a population-representative longitudinal twin study to explore how links between genetic and environmental influences at ages 17 and 24 may be implicated in the developmental processes involved. At age 17, expression of both genetic and nonshared environmental vulnerabilities unique to antisocial behavior was greater among those with low GPA than among those with higher GPA. This suggested that maintenance of high GPA buffered the impact of both genetic and environmental influences encouraging antisocial behavior. When GPA was high, both genetic and environmental influences involved in both traits encouraged good school performance and restrained antisocial behavior. At age 24, however, correlated family environmental influences drove the association between educational attainment and antisocial behavior. Antisocial characteristics involving school performance and educational attainment that transcend generations may slot individuals into social categories that restrict opportunities and reinforce antisocial characteristics.

Symptoms of Adult Antisocial Behavior (AAB) have long been associated with poor academic achievement and school failure (for example reviews, see Farrington et al., 1990; Hinshaw, 1992a, 1992b). Young people emerging into adulthood and displaying behavioral symptoms such as stealing and setting fires tend to perform poorly within the academic environment, as evidenced by low grade point averages (Fergusson & Horwood, 1995) and achievement test scores (Nelson, Benner, Lane, & Smith, 2004) and poor reading ability (Rutter, Graham, Chadwick, & Yule, 1976). They are also more likely to drop out of high school (Frick et al., 1991) and less likely to attend college (Hinshaw, 1992a). In many youth displaying overtly adult antisocial behavior, school performance difficulties and childhood disruptive behaviors have been associated for several to many years. One theoretical orientation has focused on the behaviors themselves: Childhood disruptive behaviors and school performance difficulties may be linked for any or all of four major reasons: 1) Disruptive behaviors may lead to achievement difficulties, 2) Achievement difficulties may lead to disruptive behaviors, 3) They may covary, and 4) The associations may result from underlying common causes such as difficulty comprehending or unwillingness to conform to behavioral expectations (Hinshaw, 1992a). Most researchers in the area, however, have concluded that all four possibilities contribute to the association (Conduct Problems Prevention Research Group, 1992). Moreover, there is consensus that mediation by attention problems, though germane to disruptive

behaviors in childhood, falls away as an important contributor to the more serious manifestations of AAB in late adolescence and adulthood. Some researchers have attributed the association to social or environmental causes, pointing to the common presence of poverty and associated neighborhood problems, and exposure to violence. Parental failure to provide appropriate emotional support, stimulation, and control among young people displaying both school performance difficulties and AAB symptoms have also been suggested as playing causal roles (e.g., Hinshaw, 1992a).

Researchers have also acknowledged a second theoretical orientation that seeks to explain the association between school performance difficulties and antisocial behavior using individual characteristics. From this perspective, the individual characteristics that contribute to antisocial behavior also impair school performance, blocking the attainment of educational qualifications and jobs that can provide financial stability. This orientation can be traced to the beginning of the 20th century, when psychiatrists attributed kleptomania and pyromania to distortions of personality (Binder, 1987). As with research seeking environmental explanations, recent work from this orientation has focused on identifying the specific individual characteristics involved (e.g., Eysenck, 1977; Fergusson, Swain-Campbell, & Horwood, 2004; Lykken, 1995, Lynam, Moffitt, & Stouhamer-Loeber, 1993; Raine, 2002), including poor conditionability, neurological and cognitive deficits, and specific personality traits like impulsivity. As a generality, research from this orientation can be thought of as exploring specific biologically-related processes that make some young people likely both to suffer school performance difficulties and to behave antisocially.

Genetic and Environmental Influences on Antisocial Behavior

Researchers working from both orientations have been aware that antisocial behavior tends to run in families. Disentangling the roles of external environment and internal characteristics, however, requires separating the roles of genetic and environmental influences that both act to create familial similarity. Two major factors have made this a difficult research task. First, environmental experiences such as child maltreatment and domestic violence are antisocial behaviors and the perpetrators are themselves antisocial to at least some degree. In children, these experiences may contribute to both school performance difficulties and antisocial behaviors (e.g., Frick, 2004; Frick et al., 1991). To the extent that there could be genetic influences on AAB, this means that the offspring of antisocial parents are likely both to inherit genetic predispositions to AAB and to experience antisocial home environments, a situation termed gene-environment correlation. Second, both the twin and adoption study methods that have commonly been used to distinguish these kinds of influences have limitations that apply directly to this distinction. Studies of twins reared together generally cannot reliably distinguish between nonadditive (epistatic) genetic and environmental influences that create family similarity, and adoption studies often do not adequately represent the antisocial portion of the population because such people are less likely adopt (Stoolmiller, 1999).

Despite these research limitations, there is substantial evidence across many studies that 40–50% of the variation in antisocial behavior is subject to genetic influence (Miles & Carey, 1997; Rhee & Waldman, 2002). Moreover, these data indicate that the role of environmental influences is similarly strong. The degree to which these environmental influences contribute to familial similarity, however, is less clear. These methodological issues are complicated by the observation that familial environmental influences appear to have some importance in childhood and adolescence, but not in adulthood (Miles & Carey, 1997; Rhee & Waldman, 2002). Researchers have interpreted the age-related decrease in familial environmental influences as resulting from age-related opportunities to select a broader range of environmental experiences that either positively or negatively reinforce antisocial behavior. The developmental process involved, however, has not been articulated.

Despite this, there is increasing recognition that both environmental circumstances and individual characteristics have roles in this developmental process (Miech, Caspi, Moffitt, Wright, & Silva, 1999). Moreover, there is growing recognition of the interactions and/or correlations between genetic and environmental influences involved in the association between individual differences in vulnerability to environmental risk factors for antisocial behavior and school performance difficulties. Several adoption studies have shown greater likelihood of criminal behavior in adoptees with both relatively antisocial biological and adoptive parents than in adoptees with either relatively antisocial biological or adoptive parents alone (e.g., Cloninger, Sigvardsson, Bohman, & von Knorring, 1982; Ge, Conger, Cadoret, & Neiderhiser, 1996; Riggins-Caspers, Cadoret, Knutson, & Langbehn, 2003). Recent twin studies have provided similar indications involving antisocial behavior (e.g., Button, Scourfield, Martin, Purcell, & Mcguffin, 2005; Feinberg, Button, Neiderhiser, Reiss, & Hetherington, 2007). These kinds of effects suggest greater genetic influences on antisocial behavior in the presence of adverse environmental conditions that could also lead to poor school performance, though of course they say nothing about any effect that school performance itself may have on antisocial behavior.

Such genetic influences must operate through psychophysiological characteristics, or endophenotypes (Gottesman & Gould, 2003), of some kind. Some of the characteristics that may be involved include low resting heart rate, greater heart rate variability, reduced skin conductance and startle potentiation, greater testosterone and cortisol response to provocation, abnormal brain activation patterns in the prefrontal and right temporal cortex, and higher blood serotonin levels (Raine, 2002). These characteristics have been interpreted as markers of failure to respond to instrumental conditioning and negative reinforcement (Lykken, 1995) that may also affect school performance, but these associations and their genetic linkages require further investigation. One genetic polymorphism in the promoter region of the gene encoding an enzyme that metabolizes the neurotransmitter monoamine oxidase A (MAOA) has been reasonably consistently shown to confer vulnerability to antisocial behavior when coupled with childhood maltreatment (Caspi et al., 2002; Foley et al., 2004; Kim-Cohen, et al. 2006; but see Haberstick et al., 2005). Childhood maltreatment of this type would be an example of the kind of parental abuse and neglect that may leave children unable to learn in the typical school environment, leading to chronic performance difficulties. Other biological circumstances that appear to confer vulnerability to antisocial behavior due to neurological damage that may also impede school performance include obstetrical and birth complications (Raine, 2002). These biological circumstances may or may not involve genetic influences.

Most of the evidence involving interactive effects between genetic and environmental influences indicates genetic vulnerability to environmental risk factors, but it has also been noted that sometimes the psychophysiological markers of antisocial behavior are manifested only in those from higher social class backgrounds or better family circumstances usually considered protective and associated with better school performance (e.g., Maliphant, Hume, & Furnham, 1990; Raine, Reynolds, Venables, & Mednick, 1997; Scarpa, Romero, Fikretoglu, Bowser, & Wilson, 1999). Raine (2002) has suggested that when a child biologically vulnerable to antisocial behavior lacks social risk factors for antisocial behavior, the biological factors may explain the antisocial behavior, which may indicate greater genetic influences on antisocial behavior in better environments. A more nuanced explanation, however, is that the genetic influences on particular biological markers involved in antisocial behavior may be constant across the range of social environments, while other genetic influences on antisocial behavior may be smaller in better environments and greater in more adverse environments. If environmental influences are constant across the environmental range, the potentially constant genetic influences on particular biological markers will be more apparent in the better environments than in more adverse environments because they will comprise a greater proportion of the total genetic influences on antisocial behavior in the better environments

(Johnson, 2007). If genetic and/or environmental variance in antisocial behavior is greater in better environments, it could be because adverse environments suppress the development of positive individual characteristics. If the reverse is the case and variance in antisocial behavior is greater in adverse environments, it could because adverse environments enhance the expression of individual vulnerabilities to negative characteristics. The difference between these two situations is clearly of relevance to understanding developmental processes.

Most studies exploring associations between school performance and antisocial behavior have been limited to childhood and adolescence. Yet one of the primary purposes of education is preparation for adult citizenship. School performance in adolescence clearly contributes to educational attainment beyond adolescence, and educational attainment clearly contributes to the availability of occupational opportunities that make it more or less possible to attain financial independence and constructive social roles. Antisocial behavior in adolescence contributes to antisocial behavior in young adulthood, and the two developmental processes may be interwined. Though most adolescents display reduced levels of antisocial behavior as they move into young adulthood (Moffitt, 1993; Moffitt & Caspi, 2001), those who do not may encounter limits in the educational and occupational opportunities available to them. It is thus surprising that few, if any, studies have examined how the links between school performance and antisocial behavior change with the transition from adolescence to young adulthood.

This Study's Research Contribution

This introduction has highlighted some of the reasons that clarifying and distinguishing the ways in which school performance is involved in the manifestation of antisocial behavior has proved a difficult problem in developmental psychopathology and identified a critical developmental period that has received little attention in this area of research. Recent methodological advances in quantitative genetics make it possible to consider the links involved in new ways. In particular, Purcell (2002) has outlined a model that allows for the explicit examination of how genetic and environmental influences on a particular trait may vary in the context of correlation with genetic and environmental influences on an associated trait, and Johnson (2007) has articulated how this model can contribute to our understanding of the social and developmental forces involved. In the current study, we made use of this model to explore these links over the transition from adolescence to young adulthood. Our data come from the Minnesota Twin Family Study (Iacono, Carlson, Taylor, Elkins, & McGue, 1999), a longitudinal, population-representative sample of twins assessed at ages 17 and 24. In carrying out our study, we addressed four questions falling into two major areas.

First, what are the observable associations between grade point average and antisocial behavior at age 17, between antisocial behavior at age 17 and later educational attainment at age 24, and between educational attainment at age 24 and changes in antisocial behavior between ages 17 and 24? Measurement of these associations allowed baseline inferences about the likely roles of environmental circumstances and individual characteristics in linking school performance and antisocial behavior (Miech et al., 1999) that we then explored in greater detail with the recently developed quantitative genetic model. Second, what are the links between the genetic influences on school performance and the genetic influences on antisocial behavior, and between environmental influences on school performance and environmental influences on antisocial behavior? Third, are these links constant, or do they vary at different levels of school performance, as would be the case if they were moderated by school performance? Fourth, if they vary, how do they vary? Does variance in antisocial behavior increase or decrease with better school performance? These general questions capture the idea that genetic and environmental influences transact[1] during the latter part of what is clearly a developmental process. We addressed them by focusing on three specific bivariate associations: School grades

and antisocial behavior at ages 17 and 24, antisocial behavior at ages 17 and 24, and educational attainment and antisocial behavior at age 24.

Method

Sample

Participants were male and female twin pairs from the ongoing Minnesota Twin Family Study (MTFS). The MTFS is a population-based accelerated longitudinal study of same-sex twins and their parents. It consists of two cohorts, recruited at ages 17 and 11. We made use of the older cohort in this study. It has been assessed at ages 17, 20, and 24, with assessments at age 29 in progress. The cohorts were recruited by using publicly available databases to determine the current status and location of more than 90% of the same-sex twin pairs born in Minnesota in the targeted years of 1972–1978 for males and 1975–1979 for females. Located twins who lived within a day's drive of Minneapolis with at least one biological parent were invited to complete a day-long, in-person assessment at our labs at the University of Minnesota. Those with significant mental or physical handicap were excluded. Less than 20% of located, eligible families declined participation.

The twins and their parents were generally representative of the Minnesota population during the period of the twins' birth. Fathers had a little more than 14.5 years of education on average, mothers about 1 year less. The average Hollingshead (1957) occupational level for the families was about 4. This indicates jobs at the skilled "blue collar" level, commensurate with an average level of education slightly beyond high school. The full range of occupational levels was represented, however, with some parents working in highly professional occupations and others unemployed or working in semi-skilled jobs. The standard deviation was just under 2 Hollingshead levels. Of importance in understanding the representativeness of the sample, more than 80% of the families who did not participate completed a brief mail or telephone survey. This made it possible to compare participants and non-participants on some measures. Parents in non-participating families were significantly but only modestly less educated than those in participating families. The mean difference was less than .3 years of education. Non-participating families did not differ significantly in self-reported mental health from those participating. Iacono et al. (1999) provide a complete analysis of non-participants and description of the ascertainment and assessment procedures used in the MTFS.

The male sample consisted of 578 twins (289 pairs). Of these, 376 were monozygotic (MZ) and 202 were dizygotic (DZ). The female sample consisted of 674 twins (337 pairs), including 446 MZ and 228 DZ twins. At age 24, 532 (92%; 342 MZ, 190 DZ) males and 631 (93%; 410 MZ, 221 DZ) females returned for assessment. There were no significant differences between those who returned at age 24 and those who did not in family occupational level or age 17 antisocial behavior. Those who returned at age 24 had slightly higher grades in school at age 17 than those who did not (p=.012, average grade point average of 3.06 for those who returned and 2.85 for those who did not, standardized mean difference of .28). Consistent with the demographics of Minnesota for the birth years sampled, over 98% of the twins were Caucasian.

Measures

Academic Achievement at Age 17—MTFS does not in general collect data on actual grades. Rather, participants reported grades in language arts, math, social studies, science classes, and overall by indicating that the grades were much better than average (A's=4), better than average (B's=3), average (C's=2), below average (D's=1), or much below average (i.e.,

[1]We use the word 'transact' here and throughout this paper to refer to interplay between genetic and environmental influences in general. The word 'interact' is often used for this, but it has a particular statistical meaning to which we do not wish to limit our reference.

failing=0). Possible scores thus ranged from 0 to 4. This approach was taken because of the disparity in grading formats, procedures, and standards taken in the various school systems from which the MTFS families are drawn. We made use of the average of the subject reports, and refer to this variable as GPA for Grade Point Average. A random sample of 67 participants from the younger MTFS cohort assessed at age 14 showed a correlation between grade point average computed from school transcripts and our reporter measure of .89. Those participants who did not end up completing high school generally still provided grade reports at the age 17 assessment, either because they were still in school at that time or because they reported the kinds of grades they have been earning before they dropped out.

In addition to this measure of school performance, we included three measures of the participants' ability and motivation to continue education in order to examine how ability may contribute to the associations between school performance and antisocial behavior. Participants received an abbreviated version of the Wechsler Adult Intelligence Scale - Revised (WAIS in the following; Wechsler, 1981) at age 17. The abbreviated version consisted of 2 verbal (Vocabulary and Information) and 2 performance (Block Design and Picture Arrangement) subtests. These subtests were selected for their high correlation (.90) with total WAIS IQ based on all subtests. At age 17, participants also received the reading test from the Wide Range Achievement Test. We made use of the grade-equivalent reading score (WRAT in the following) as a second measure of ability because reading ability is so fundamental to academic performance.

Educational Attainment—At age 24, participants reported both educational attainment to date and their current enrollment in educational programs, if relevant. We used this information to compile a 12-point scale of educational attainment. Scale values ranged from 0 for failure to complete high school through 4 for completion of a post-high school vocational training program, through 8 for completion of a 4-year college degree, to 12 for completion of a professional degree (PhD, JD, MD, etc.). Participants who dropped out of high school but returned to complete a general equivalency degree were coded as 1; those who completed high school in the usual manner but attained no education beyond that were coded as 2. Participants who were enrolled in master's degree programs were coded as 9; those who had completed master's degrees but were no longer enrolled in school were coded as 10. Observations ranged from 0 to 11, corresponding to the range from failure to complete high school to currently enrolled in a professional degree program. The mean for the scale was 6.3, corresponding to slightly more than completion of a community college degree; standard deviation was 2.4. The variable was approximately normally distributed.

Antisocial Behavior—MTFS participants were interviewed at ages 17 and 24 to assess symptoms of psychopathology according to the edition III-R of the American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders* in effect at time of assessment. Interviewers had either bachelor's or master's degrees in psychology and were trained extensively in the diagnostic interview process. We made use of symptom counts of adult antisocial behavior (AAB; the adult criteria for antisocial personality disorder) at both ages. The age 24 AAB symptoms were assessed using a structured interview designed by MTFS staff that is comparable to the Structured Clinical Interview for Axis II Personality disorders (First, Gibbon, Spitzer, & Williams, 1994). The symptom assignments were made during a review of the interview conducted by teams of at least two graduate students with advanced training in descriptive psychopathology and differential diagnosis that resulted in consensus among the team members on the appropriate number of symptoms for each interview subject. Mean symptom counts for AAB were .60 (*sd*=1.10) at age 17 and .92 (*sd*=1.07) at age 24 on a scale that included observations ranging from 0 to 7. Both variables were thus significantly skewed and we made use of the log of their observed values plus 1 (because the log of 0 is not defined) throughout our analyses[2].

Socioeconomic Status (SES)—We measured family SES at age 17 based on parental occupational status Hollingshead (1957) code of the parent with the highest status because the participants in our study were living at home with at least one biological parent.

Analytical Approach

Examination of Observed Associations Between School Performance and AAB at Ages 17 and 24—Our first research question involved the observed associations between GPA and antisocial behavior at age17, between antisocial behavior at age 17 and educational attainment at age 24, and between educational attainment at age 24 and changes in antisocial behavior between ages 17 and 24. Miech et al. (1999) outlined the process we used: (1) Examine the association between AAB at age 17 and GPA, after controlling for other typical predictors of educational attainment such as IQ, reading ability, and parental SES. (2) Evaluate the degree to which AAB at age 17 predicted educational attainment at age 24, again controlling for other typical predictors of educational attainment such as IQ, age 17 GPA, reading ability, and parental SES. This measures the extent to which the association continued across the transition into adulthood, suggesting a causal link between age 17 AAB and later educational attainment. Finally, (3) Examine the association between GPA at age 17 and educational attainment at age 24, and the association between age 17 GPA and increases in AAB from ages 17 to 24. This measures the extent to which there are reciprocal effects of educational attainment on changes in AAB over the period.

Examination of the Genetic and Environmental Links Between AAB and SES at Ages 17 and 24—To address our three remaining research questions, we made use of a model developed by Purcell (2002) that allows for the explicit quantification of genetic and environmental influences on a particular trait in the context of correlation with and moderation by genetic and environmental influences on an associated trait. To explain this multivariate model, we begin with the standard quantitative genetic model for a single variable, here AAB. This univariate model is based on the understanding that the phenotypic (observed) variance (Vp) in a trait is a linear function of additive genetic (A^2) and shared (C^2) and non-shared (E^2) environmental variance components ($Vp = A^2 + C^2 + E^2$). Shared environmental variance reflects influences that distinguish among families but act to make members of the same family similar. Parental attitudes toward education and neighborhood violence are common examples. Nonshared environmental variance includes measurement error, but it also reflects influences that act to make members of the same family different. An important example with respect to AAB may be different experiences with police arrest. The distinction between shared and nonshared environmental variance can be subtle. For example, twins growing up in the same family may experience the same event (e.g., witnessing violent parental conflict), but that event is only a shared environmental influence to the extent that it makes the twins similar for AAB measured later, and the twins may have reacted to it very differently. This standard model for a single variable is shown in Figure 1.

Under this model, each of the variance components is assumed to be constant throughout the population and independent of the others. Moreover, there is no provision for the possibility that the influences on AAB are associated with those on any other trait, in particular school performance. These assumptions can be relaxed to hint at the processes underlying such associations. One way to do this is to test whether school performance exerted moderating effects on the components of variance in AAB while making provision for the possibility of

[2]Log transformation reduced skewness from 2.39 in Age 17 AAB to 1.35, and from 1.81 in Age 24 AAB to .43. Skewness, particularly in Age 17 AAB, resulted not only from accumulation of 0 values but also from a few participants with large numbers of symptoms. We could have dichotomized the AAB variable at 0 and all other values, but this would have eliminated the difference between an individual with 1 AAB symptom and the individual with 7. Examination of the regression residuals indicated that the regression coefficients we generated underestimated the actual magnitudes of association between our educational and AAB variables.

overlap in the influences on AAB and school performance. That is, rather than modeling variance in AAB as consisting of constant variance components, we used a model that both allowed the variance components of AAB to vary with school performance and measured the extent of overlap of influences between AAB and school performance.

Variation in the genetic and/or environmental influences of variance in AAB with level of some other variable such as school performance is an example of moderation of those influences by the other variable. This reflects direct transaction between genetic and/or environmental influences on the two variables. Overlap in the genetic influences on two traits is an example of genetic correlation (r_{GE}), and similar kinds of overlap can occur for shared and nonshared environmental influences. Like observed correlations, genetic and environmental correlations range from −1 to 1.

The models we used to include provision for these phenomena adapted the standard quantitative genetic model shown in Figure 1 by explicitly measuring the possibility that the variance components of AAB themselves varied as continuous functions of school performance. The adapted model, described by Purcell (2002), is shown in Figure 2, using educational attainment as the example of school performance. In this model, the paths expressing the genetic and environmental influences on educational attainment (indicated as a triangle) are considered constant as in Figure 1. We have not explicitly labeled these paths in Figure 2 (though we measured them) to keep the focus on the paths contributing to variance in AAB. The paths that are labeled in the figure express the genetic and environmental influences on AAB. These paths are all linear functions of the form × + M*b, where × and b are regression coefficients and M is the level of educational attainment.

There are two sets of paths contributing genetic and environmental influences to AAB: those common to educational attainment as well, and those unique to AAB. The extent to which educational attainment moderates the variance in AAB is measured on all 6 paths. The resulting model thus provides estimates of the genetic and environmental variances common to educational attainment and AAB and the extent to which these vary with educational attainment, as well as estimates of the genetic and environmental variances unique to AAB and the extent to which these vary with educational attainment. Estimates of r_{GE} and the environmental correlations can be derived from the relative magnitudes of the variances common to AAB and educational attainment and those unique to AAB. In most multivariate models, these correlations are constant like the variance components. In the presence of significant moderation of genetic or environmental influences on AAB, however, the corresponding genetic or environmental correlations vary as functions of educational attainment. Measurement of genetic and environmental correlation and moderation of genetic and environmental influences enable us to address the extent to which environmental circumstances and/or individual characteristics are involved in the link between educational attainment and AAB.

If poor school performance breeds AAB and successful school performance restricts it, AAB is at least partly caused by school performance. If, instead or in addition, AAB is caused by pre-existing, biologically-based vulnerabilities that also result in poor school performance, the association between AAB and school performance is due to individual characteristics. In order for this latter explanation to be accurate, there must be genetic or environmental influences common to AAB and school performance. But this kind of common genetic or environmental influence is not sufficient to render this the primary explanation. The very existence of this kind of correlation between AAB and school performance implies that the social system in which it exists will not be static over time (Johnson, 2007). This is because individuals who differ in ways that influence both AAB and school performance will actively seek out as well as passively receive different experiences involving both, but they will do this with varying

degrees of success over time. This suggests that genetic and environmental differences in AAB will be expressed to differing degrees with different school performance experiences. As long as there is genetic or environmental variance common to AAB and school performance, the presence of both genetic and environmental moderation and correlation is likely, and they will be related in lawfully constrained ways (Johnson, 2007).

Data Adjustment and Analytical Implementation—Because twins are the same age and, in our sample, the same gender, age and gender effects may act to increase their apparent similarity (McGue & Bouchard, 1984), which can affect estimates of genetic and environmental influences. Though the vast majority of participants were assessed during the year in which they were age 17 at intake, the assessments targeted for age 24 actually took place at varying ages surrounding 24. To adjust for this, we regressed the effects of age and gender from the variables at each age, and used the standardized residuals, which also placed all the variables on a uniform scale. There are well-known gender differences in both antisocial behavior and school performance, and the technique we used obviously removed these effects from our data. Future work should explore these differences, but the primary research questions and approach we took were sufficiently novel, and maintenance of statistical power of sufficient importance, that we believed it worthwhile to explore our objectives initially in the full sample. We estimated the phenotypic associations described above using multiple regression with maximum likelihood estimation of raw data in the computer program Mplus (Muthen & Muthen, 1998–2006). Our analyses adjusted for the clustered nature of the twin data using a sandwich estimator and were based on full information likelihood methods, assuming that data were missing at random (Little & Rubin, 1987).

We implemented the quantitative genetic models in the computer program Mx (Neale, Boker, Xie, & Maes, 2003), specifying them as recommended by Purcell (2002). For our second research question involving how genetic and environmental influences on school performance and AAB transact to shape the association between educational attainment and AAB at age 24, we were primarily interested in the model shown in Figure 2. We used it to explore both the age 24 snapshot and the developmental process involved in moving from the situation involving GPA and AAB at age 17 to the situation involving educational attainment at age 24. To address how genetic and environmental influences on GPA and AAB at age 17 transact to shape the association between GPA and AAB, we estimated models directly analogous to Figure 2 that explored associations between GPA and AAB at age 17. To address how and to what degree genetic and environmental influences on AAB were linked at ages 17 and 24, we estimated models again directly analogous to Figure 2 that explored the associations between AAB at age 17 and AAB at age 24. We also estimated several other models to check the uniqueness of the associations we observed to the variables in question and to test the directions of effects. Thus, we estimated analogous models exploring associations between IQ and AAB at age 17 and reversed the order of the variables in all the models in order to explore whether there were reciprocal moderating effects of AAB on school performance. We also estimated models exploring associations between GPA at age 17 and AAB at age 24, and between GPA at age 17 and educational attainment at age 24.

Because of the complexity of the quantitative genetic models produced, we allowed parsimony to dictate the results presented. To do so, we dropped all terms indicating moderating genetic and/or environmental effects from the models when we could do so without significant change in model-2*log likelihood or significant change in the parameters that remained. We corroborated the appropriateness of this using Akaike's Information Criterion (AIC, (Akaike, 1983), an information theoretic fit statistic. These kinds of fit statistics provide objective criteria for selecting models that minimize the amount of information required to express the data, making it possible to determine the most parsimonious or efficient representation of the data. The point of doing this was not to deny the potential existence of smaller interactive effects on

the paths that were not significant in this particular sample, but to focus attention on the paths and thus the social forces that were most important in these data.

Results

Descriptive statistics

Table 1 shows the zero-order correlations among the variables used in our study. As would be expected, all of the measures of academic ability were substantially positively correlated among themselves. AAB at age 17 was similarly correlated with AAB at age 24. In contrast, AAB at either ages 17 or 24 was not significantly correlated with parental SES. There was a small negative correlation (−.09) between AAB and IQ at age 17, but the correlation with IQ was no longer significant at age 24. Though the associations involving IQ and reading ability and AAB were at most marginal, the associations involving school performance and AAB were in the small to moderate range: the correlation between GPA at 17 and AAB at the same age was −.30, and GPA at 17 predicted age 24 AAB at −.20. Age 17 AAB predicted attained education at age 24 at −.31, and the correlation between age 24 AAB and attained education was −.21.

The correlations between members of monozygotic (MZ) and dizygotic (DZ) twin pairs for the study variables are given in Table 2. Genetic influences are indicated to the extent that MZ correlations are greater than DZ correlations, and their relative magnitude can be approximated for a given variable as twice the difference between the two correlations (Falconer & Mackay, 1996). Shared environmental influences are indicated to the extent that DZ correlations are more than half the MZ correlations, and their relative magnitude can be approximated for a given variable by doubling the DZ correlation and subtracting the MZ correlation (with a lower bound of 0). Nonshared environmental influences are indicated to the extent that MZ correlations are less than 1.00. Thus influences on parental SES were completely shared environmental because both members of each twin pair shared the same parental home. The rest of the variables showed both substantial genetic and substantial nonshared environmental influences. Only WAIS IQ and attained education at age 24 showed important shared environmental influences. The observations for these variables at these ages in this sample were very similar to those from other studies.

Observed Associations Between School Performance and AAB at Ages 17 and 24

Table 3 shows the results of the three tests we carried out to investigate our first research question involving the associations between school performance and AAB at ages 17 and 24. The multiple regression of age 17 AAB on age 17 GPA and associated variables accounted for 9% of the variance in AAB at age 17. The only significant coefficient was −.29 for age 17 GPA. The second test involved the degree to which AAB at age 17 predicted educational attainment at age 24, after controlling for the other variables associated with school performance. As the table shows, this multiple regression accounted for 33% of the variance in educational attainment, and all the variables except reading ability were uniquely significant predictors. Our primary interest, however, was in the coefficient for age 17 AAB, which was −.16 and significant at $p<.001$. Finally, the third test involved the degree to which educational attainment at age 24 was related to changes in AAB from ages 17 to 24. This multiple regression accounted for 18% of the variance. As expected, AAB at 17 was a highly significant predictor of age 24 AAB at .38 ($p<.001$), which indicated some stability in rank order of AAB between the two ages. Age 17 GPA did make a significant contribution to that change at −.07 ($p=.03$), but educational attainment at age 24 did not contribute significantly to age 24 AAB when age 17 AAB and GPA were controlled (−.03, $p=.45$). Taken together, these three tests suggested the presence of reciprocal effects linking AAB and school performance, but they provided no

information on the genetic and environmental sources of these effects or how they might vary within the population.

Links Between Genetic and Environmental Influences on AAB and Educational Attainment at Age 24, and on AAB and GPA at Age 17

As described above, we considered several models of the genetic and environmental influences linking school performance and AAB to explore our remaining research questions. We addressed the question of how genetic and environmental influences on educational attainment and adult antisocial behavior transact to shape their association with the model involving AAB and educational attainment at age 24, and the top part of Table 4 shows the model fit statistics determining the most parsimonious version of this model. Educational attainment at age 24 had no significant moderating effects on the genetic and shared environmental influences on AAB at age 24; the model including the relevant paths did not fit significantly better than the model with no moderating paths. This was the case when we reversed the roles of these variables as well: AAB had no moderating effects on the genetic and environmental influences on educational attainment. We used the model associating GPA and AAB at age 17 to address the question involving how genetic and environmental influences on school grades and antisocial behavior at age 17 transact to shape their association at this younger age. The bottom part of Table 4 shows the model fit statistics from all the models used to determine the most parsimonious version. Here GPA at age 17 did moderate the genetic and nonshared environmental influences on AAB, and it was the genetic and nonshared environmental influences *unique* to AAB at age 17 that were affected; the model including the relevant paths fit significantly better than the model with no moderating paths, and this model's fit did not differ significantly from that of the model including all 6 moderating paths.

We reversed the variables in this model to explore the directions of the moderating effects, estimating the potential moderating effects of AAB at age 17 on GPA at age 17. The most parsimonious version of this model included a moderating effect of AAB on nonshared environmental influences on GPA ($\Delta\chi^2$ =10.65 on 1 df, p=.001; ΔAIC=7.14). The moderating effect was that nonshared environmental influences unique GPA increased with increasing levels of AAB. As genetic and shared environmental influences were constant across this range, their proportion of total variance decreased with increasing levels of AAB. The correlation between genetic influences on GPA and AAB was −.53 and constant across the range of AAB. Though the nonshared environmental correlation varied with level of GPA, it was not of major importance, being well under .25 for most of the range. We also estimated the moderating effects of GPA at age 17 on AAB at age 24 to examine the consistency of the effects we had observed over time. The most parsimonious version of this model showed moderating effects only on the nonshared environmental influences on AAB ($\Delta\chi^2$ =19.22 on 1 df, p<.001; ΔAIC=17.22). Otherwise, the model produced estimates of genetic and environmental influences and their correlations that were very similar to those for the effects of GPA on AAB at age 17.

None of the other models we fit to check for the involvement of other variables or reciprocal effects included any paths with moderating influences. Thus, AAB at ages 17 and 24 exerted no moderating influences on educational attainment at age 24: the reciprocal moderating effects between genetic and environmental influences on GPA and AAB did not persist to educational attainment at age 24. IQ also exerted no moderating influences on AAB at either age, and there were no important correlations between genetic or environmental influences on IQ and AAB at either age. Thus the moderating effects of GPA on AAB appeared to involve school performance rather than academic ability. Finally, parental SES did not moderate either genetic or environmental influences unique to AAB at either age. Because there were no moderating effects in any of these models, we will not describe these results further.

Our final questions involved how and to what degree genetic and environmental influences on antisocial behavior were linked at ages 17 and 24, and genetic and environmental influences on GPA at age 17 were linked to genetic and environmental influences on educational attainment at age 24. In the AAB model, neither genetic nor environmental influences on AAB at age 17 moderated genetic or environmental influences on AAB at age 24. Most of the genetic influences on AAB at the two ages were the same: the genetic correlation was .84, and the genetic variance at the two ages was about the same:.49 at age 17 and .46 at age 24. In contrast, both shared and nonshared environmental influences on AAB at the two ages were effectively uncorrelated. This indicated substantial, though not complete, continuity in the genetic influences on AAB at the two ages, though it appeared that the environmental influences were different. In the school performance model, shared environmental influences common to the two forms of school performance moderated shared environmental influences on educational attainment ($\Delta\chi^2$ =18.83 on 1 df, p<.001; ΔAIC=16.83). Shared environmental influences on educational attainment were important (.80 at 2 standard deviations below mean GPA) when age 17 GPA was low and fell to unimportance (.09 at 2 standard deviations above mean GPA) with increasing GPA. Genetic and nonshared environmental variances were constant at .33 and .26, respectively, across the range. The genetic correlation was .78, the shared environmental correlation was 1.00, and the nonshared environmental correlation was .10.

The estimated components of variance, proportions of variance, and genetic and environmental correlations from the most parsimonious models with important moderating effects at the two ages described in Table 4 are given in Table 5, along with their 95% confidence intervals. The confidence intervals for the genetic and environmental correlations are very wide. The models have little power to specify these correlations exactly (Carey & DiLalla, 1994), so this is typical for these kinds of correlations. We did, however, have substantial power to detect the presence of genetic (85–90%) and nonshared environmental (80–85% at age 17, 70–75% at age 24) covariance between antisocial behavior and school performance. Thus, importantly, the genetic correlations and some of the nonshared environmental correlations in the model linking age 17 AAB and GPA were significant. About 36% of the variation in level of educational attainment at age 24 was under genetic influence, and about 34% was under shared environmental influence. The remaining 30% was under nonshared environmental influence. These relative proportions likely reflected both the tendency for educational attainment to cluster around graduation milestones and some distribution of parental financial resources for education as genetic influences on GPA at age 17 were relatively more important and shared environmental influences less important: 70% of the variation in GPA was under genetic influence and only 3% was under shared environmental influence, with the remaining 27% under nonshared environmental influence. At age 24, 33% of the variation in AAB was under genetic influence, 16% was under shared environmental influence, and 51% was under nonshared environmental influence. The allocation of variance in AAB age 17 varied with level of GPA. The numerical results are summarized in Table 5 and presented graphically in Figures 3 and 4.

Figure 3 shows the three components of variance in AAB at age 24 as functions of educational attainment at the same age. Because there was no significant moderation of variance by educational attainment, the three variance components of AAB were constant across level of educational attainment. As shown in Table 5, the genetic and environmental correlations were also constant across the range of educational attainment: both the genetic and nonshared environmental correlations were effectively 0. The shared environmental correlation was rather large but, because the shared environmental variance component was rather small at only 16% of the variance, this correlation did not reach significance and was not of major importance in understanding the pattern of associations. Because there were no moderating effects and no genetic or nonshared environmental correlations, this pattern suggested that the association between educational attainment and age 24 AAB was primarily driven by environmental

circumstances created by lack of educational attainment. At the same time, shared environmental influences were involved, to the extent of their magnitude, in a manner that suggested the importance of individual characteristics common to both.

Figure 4 shows the three components of variance in AAB at age 17 as functions of age 17 GPA. Because there was no significant moderation of shared environmental variance by GPA, its variance component of AAB was fixed to be constant across the range of GPA. Because there was significant moderation of genetic and nonshared environmental variance, these variance components of AAB, stated on an unstandardized basis, did vary with GPA. Both genetic and nonshared environmental variance in AAB decreased sharply across the four standard deviation range of GPA, falling from .88 to .14 for genetic influences and from 1.02 to .17 for nonshared environmental influences. This means that the lower overall levels of AAB among adolescents with high GPA's arose primarily because there were few among them who showed high levels of AAB. In contrast, among adolescents with low GPA's the range of AAB was generally larger: there were both adolescents who showed high and low levels of AAB, and this greater overall variance had both genetic and nonshared, but not shared, environmental sources. This indicated that low GPA enhanced the expression of individual vulnerabilities to AAB rather than that low GPA suppressed the development of the ability to restrain antisocial behavior.

Because GPA moderated the genetic and nonshared environmental influences unique to AAB, the pattern of genetic and environmental correlations across the range of GPA was the opposite of that for the variance components: the genetic and nonshared environmental correlations increased across the range of GPA. Their magnitude was substantive for the genetic correlations but not for the nonshared environmental correlations. This pattern indicated that both genetic and nonshared environmental influences on individual characteristics were important. When GPA was low, however, its association with AAB appeared to result primarily from environmental circumstances, though genetically influenced individual characteristics continued to play some significant role. Shared environmental influences were of no importance.

Discussion

In this study, we used new quantitative genetic methodology to explore some of the closely interrelated processes linking antisocial behavior and school performance at the little studied transition between adolescence and young adulthood. Our purpose was to articulate the patterns of influence in order to develop a clearer understanding of the processes involved in the development and manifestation of these behaviors. To do so, we asked four specific research questions. Our first question involved quantifying the observable associations between GPA and AAB at age 17, between AAB at age 17 and educational attainment at age 24, and between school performance at both ages and changes in AAB between ages 17 and 24. We examined these associations in order to make baseline inferences about the likely roles of environmental circumstances and individual characteristics in linking school performance and AAB (Miech et al., 1999). Together, the results suggested that the association between school performance and antisocial behavior at age 24 was the result of individual characteristics that existed at age 17. There was no evidence that educational attainment or lack thereof in the intervening seven years contributed to changes in level of AAB between ages 17 and 24.

Genetic and Environmental Influences Linking School Performance and AAB

But how did the association at age 24 arise? Our second question explored how genetic and environmental influences on educational attainment and AAB transacted to shape their association at age 24. Both educational attainment and AAB were under genetic as well as environmental influences, but there were no genetic or nonshared environmental correlations. As noted above, we did have good power to detect these kinds of correlations (70–75% at age

24). The association was driven completely by correlated shared environmental influences. It was static across the range of educational attainment, as there were no moderating effects, but this very possibly reflected an absence of statistical power. It was not possible to estimate statistical power to detect moderating effects directly because the analysis required modeling of both means and covariances in the raw data as well as multiple patterns of variation in moderating parameters, but the free estimates of the moderating parameters suggested that at least one would have had to have been 2 to 3 times as large in order to be significant. The shared environmental mediation suggested that there were family influences and circumstances (but not genetic or unique environmental influences) that affected both educational attainment and AAB. Examples of family influences that might have such effects would be neighborhood characteristics involving school quality and peer influences in our sample of twins from many different school districts and family cultural values surrounding academic achievement, responsible behavior, and use and availability of family economic resources (e.g., Behrman, Pollock, & Taubman, 1995; Leventhal & Brooks-Gunn, 2004; Osborne, 2004, Ryan, 2001). Because our study could distinguish potentially confounding genetic effects, it provides evidence that the shared environmental influences are truly the operative processes.

Our third question explored the antecedents to the age 24 observations by modeling the association between age 17 GPA and AAB. GPA is a measure specific to the individual that predates and predicts both later educational attainment and AAB. It is thus perhaps surprising that our results for GPA suggested very different processes from those for educational attainment. Both genetic and nonshared environmental influences on AAB decreased with increasing GPA, yet the genetic correlation was small when GPA was low and increased throughout the range of GPA so that it was large when GPA was high. The nonshared environmental correlation was small throughout the range, though it too increased across the range of GPA. This suggested that school failure in the form of poor GPA may have acted to increase the expression of both genetic and nonshared environmental vulnerabilities to AAB that were independent of the sources of the poor GPA, while high GPA suppressed this expression. For example, poor GPA could arise because of low intelligence, but the genetic and environmental influences on low intelligence could be distinct from those on AAB. At the same time, because of the frustration and social marginalization associated with low GPA in the school environment, adolescents of low intelligence could be more vulnerable to emotional and social pressures to behave antisocially that have their own sets of genetic and environmental influences. Our analysis provided no direct evidence for or against this specific interpretation, but it is the kind of process that would generate the results we did observe, and aspects of such a process have been well documented (e.g., Fergusson & Horwood, 1995; Hinshaw, 1992; Osborne, 2004; Ryan, 2001; Schwartz, Gorman, Nakamoto, & McKay, 2006). In any event, the processes appeared to take place at the level of the individual rather than the family, as shared environmental influences were not involved.

Unlike several many other studies (Carey & DiLalla, 1994; Rhee & Waldman, 2002), we did not observe substantial shared environmental influences on AAB at age 17. This may have been due to our use of the clinical adult antisocial symptom count as our measure of AAB in these barely-adult-age twins, as this measure tabulates only relatively serious antisocial behavior. By age 24, this same measure did show significant shared environmental influences, similar to those from other studies.

The answers to our question involving the linkages between genetic and environmental influences on AAB at ages 17 and 24 indicated that the genetic influences on the trait at the two ages were both very highly correlated and very similar in magnitude. Thus essentially the same genetic factors appeared to influence AAB to the same degree at the two ages. Both shared and nonshared environmental influences were also very similar in magnitude, but their correlations at the two ages were low. This suggested that the environmental circumstances

contributing to AAB between age 17 and age 24 change. In contrast, the linkages between both genetic and shared environmental influences on GPA at age 17 and educational attainment at age 24 were highly correlated, though nonshared environmental influences were not. This could be the case if, for example, peer influences were more important in influencing AAB while family influences were more important in influencing school performance across the transition from living at home with parents to living independently as young adults. We are not aware of previous work that has addressed the relative importance of these sources of influence on these traits in this way, so this would be an interesting question to explore in future research. The decreasing importance of shared environmental influences on educational attainment with increasing GPA indicated that the familial influences contributing to low GPA were more important in limiting educational attainment than the familial influences contributing to high GPA were in enhancing educational attainment. This is another question that should be explored in greater detail in future research.

Limitations

This study is subject to some methodological limitations that should be taken into account when considering the significance and generalizability of the results. First, the sample is representative of the population born in Minnesota in the 1970's, but it is primarily of European-American descent and thus should not be considered representative of the population of the United States as a whole. In particular, it probably does not include sufficient representation of some of the worst concentrations of urban poverty in the United States. Second, moderation effects of the kind we explored are often small and difficult to detect (Rowe, 2003). Though we clearly had power to identify several moderating effects, there may have been others present that would have required a larger sample size to detect. For example, Tuvblad, Grann, and Lichtenstein (2006) reported that the proportion of variance in antisocial behavior contributed by genetic influences unique to antisocial behavior increased with increasing parental SES in a Swedish sample of twins aged 16–17 that was about 80% larger than ours. In our data, parameter estimates for this limited model indicated a similar pattern, but the increase in genetic influences with parental SES was far short of statistical significance. While the difference in significance could result from differences in conditions between the United States and Sweden, the difference in sample size and thus power could also be a factor.

Third, though our data included both males and females, but we did not examine the data for possible gender differences in the associations we explored. In fact, we explicitly removed the effects of gender differences from the data before beginning our analyses. Future research should address the possibility of gender differences in this area. Finally, our estimates of GPA and educational attainment relied on self-reports, and the reports of GPA were not specifically tied to actual grades. Our measure of GPA did, however, correlate very highly with actual GPA based on report cards in our sample at age 14, and our measure of educational attainment made use of well-established educational milestones that are generally well-recalled even many years after attainment. Despite its limitations, our study potentially offers important insights into the way school performance is involved in antisocial behavior at the transition from adolescence to adulthood.

Integrating Our Results at Ages 17 and 24

Our results at age 17 suggested that the processes linking the lower end of the range of age 17 GPA to antisocial behavior differed from those linking high GPA and antisocial behavior. More specifically, our results suggest that something about very low age 17 GPA encouraged expression of genetically influenced antisocial behavior, while something about high GPA minimized expression of these genetic and environmental influences on antisocial behavior. These moderating effects on expression of genetic and environmental influences disappeared by age 24, but evidence for some effects of shared environmentally influenced individual

characteristics remained. It is easy to speculate about the effects involved: very low GPA at age 17 is associated with failure to graduate from high school or to continue to receive further educational training of any kind, and family financial resources and attitudes toward education clearly also play some role in attainment of post-secondary education. In our society, people who attain very high levels of education generally have more lucrative and financially secure occupational opportunities that provide psychological rewards that reinforce the tendency to refrain from antisocial behavior. Moreover, many of the very personal characteristics that lead to educational success, such as conscientiousness (e.g., Chamorro-Premuzik & Furnham, 2003), disciplined behavior (Duckworth & Seligman, 2006), and acceptance of responsibility, reflect the absence of antisocial behavior. In contrast, people without a high school education face severely limited occupational prospects. Some of the very personal characteristics that can lead to school failure, such as lack of conscientiousness, substance abuse (e.g., Cox, Zhang, Johnson, & Bender, 2007) and/or disruptive behavior (e.g., Schwartz, Gorman, Nakamoto & McKay, 2006) that lead to school expulsion, reflect antisocial behaviors in themselves. Compounding these personal problems, however, the resulting failure to find a place in the world may cause frustration and demoralization as well as more concrete problems such as poverty and exposure to violence that act to increase expression of antisocial behavior in ways not directly related to school failure.

Shared environmental influences also played some role in the pattern of our results regarding antisocial behavior at age 24, as they were correlated with shared environmental influences on educational attainment. In a sample such as ours that encompasses a broad range of neighborhoods within a geographic area, these shared environmental influences likely reflect cultural effects of both neighborhoods and local schools as well as strictly familial influences. This is because many of our twin pairs are the only representatives of their neighborhoods or schools in our sample.

Our results regarding antisocial behavior at age 24 suggest that it resulted from individual characteristics also involving school performance that are reinforced by both familial and broader neighborhood cultural influences that are common to them. We emphasize the contribution to shared environmental influences of broader neighborhood cultural influences in addition to familial influences for two reasons. First, we observed antisocial behavior at age 24 and by this age most people, including our participants, no longer live in the family home in which they grew up. Whatever shared environmental influences our participants showed were carried with them into the homes they found for themselves. Second, our antisocial behavior data at age 17 did not show such shared environmental influences. This may reflect the relative racial/cultural homogeneity and lack of abject poverty in our sample, as many studies show greater shared environmental influences on AAB in adolescence than in adulthood.[3]

More interestingly, it may also reflect the way in which genetic influences on parental attitudes toward education and choice of residence and AAB were linked in this sample. That is, GPA predicted educational attainment quite well but far from perfectly; in our data the correlation was .47. One of the constraints on this correlation must be parental financial resources for continuing education. Moreover, parents have a tendency to make equal financial resources available for education to all their children (Behrman, Pollak, & Taubman, 1995). Our results at age 17 suggest that at this age poor school performance was not the only source of genetic and environmental influences on expression of antisocial behavior, yet this expression was driven by individual rather than familial experiences of school failure. This could be the case

[3]It did not result from our combining males and females in our treatment of the data, as there was no evidence for shared environmental influences on AAB at age 17 in either the male or the female portions of the sample, and the twin correlations from the data in the two genders before removal of the age and gender effects were very similar to those we present.

if the poor school performance were relative rather than absolute: if poorer schools where overall achievement is lower still give high grades to those students who perform relatively better. There is at least anecdotal evidence that this does take place in the United States. At the same time, even the better students emerging from the poorer schools may have trouble attaining education beyond high school due to both limitations in their parents' willingness and/or ability to support them financially and to the weakness of their overall academic backgrounds when compared to those of students from other schools, creating effects that are shared with their siblings in adulthood due to the restrictions in occupational opportunities imposed by limited education, but which were not shared with them in childhood. We note that, to the extent that this interpretation may have accuracy, the focus is on school performance rather than on IQ, as IQ played no role in the interactive effects we observed.

Conclusion and Future Directions

Disentangling the way in which school performance is associated with antisocial behavior in our society has long challenged psychologists, epidemiologists, and sociologists alike. Despite some limitations in our study's ability to generalize to the whole population of the United States, its results introduce intriguing possibilities to explore in greater detail in future research. In particular, they point out that individual characteristics involving school performance and ultimately educational attainment that transcend generations may slot individuals into social categories that restrict their opportunities and reinforce the antisocial characteristics that slotted them into the restrictive social categories in the first place. Moreover, they suggest that preventing school failure can act to reduce exposure to other environmental influences contributing to antisocial behavior, and to reduce expression of both genetic and nonshared environmental influences on antisocial behavior, at least in adolescence, though this may also contribute to minimizing its expression in young adulthood. Whatever the accuracy or generalizability of this interpretation, our results also demonstrate clearly the great potential of the new quantitative genetic methods we used to illuminate the ways in which social forces transact with individual differences to shape individual outcomes. Antisocial behavior is a costly social phenomenon that clearly has many different individual and social risk factors that have resisted simple resolution. Researchers need to apply powerful techniques such as we used here that explicitly acknowledge the roles of both individual characteristics and social forces if they hope to understand and ultimately to minimize those costs.

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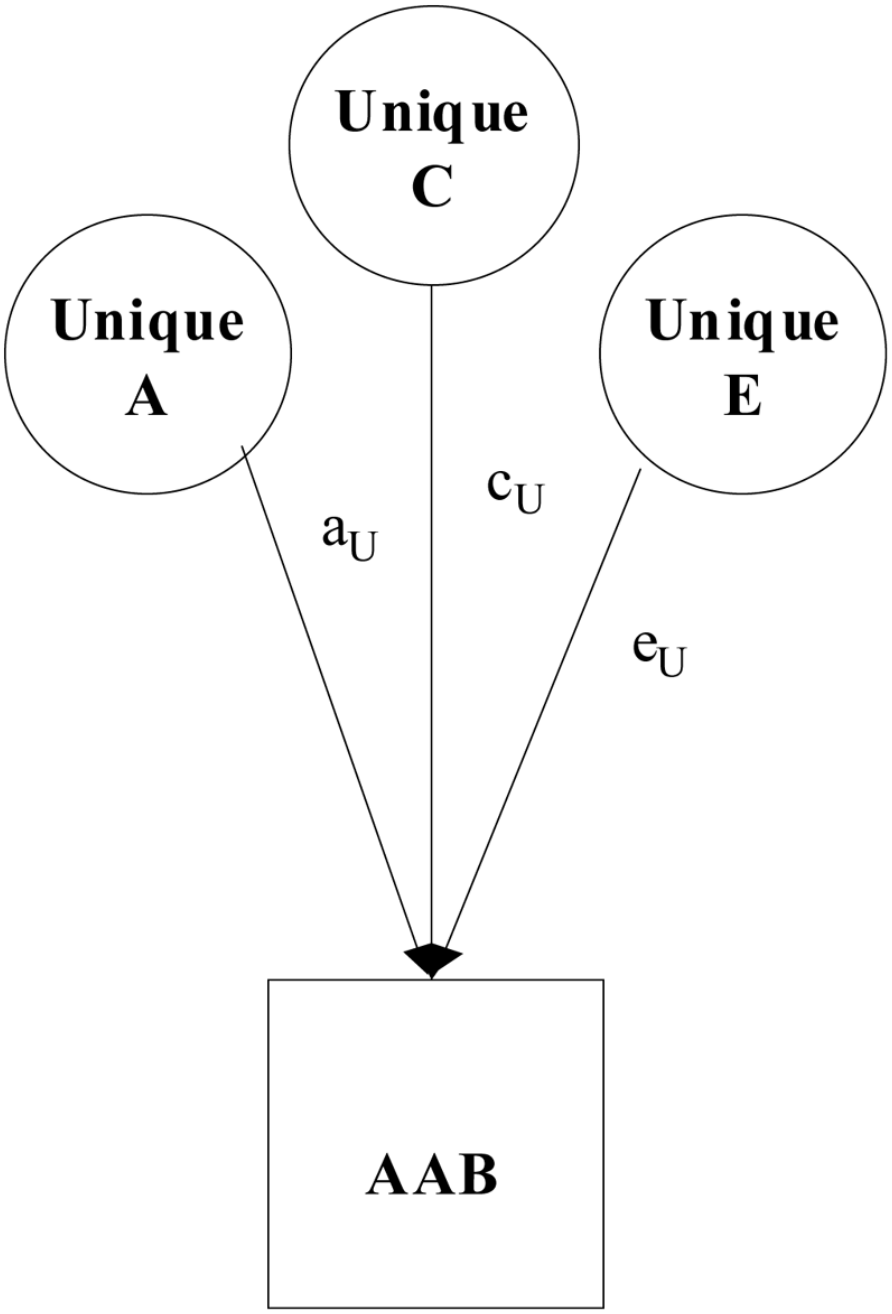

Figure 1.
Standard model of independent genetic and environmental influences. A refers to genetic influences, C to shared environmental influences, and E to nonshared environmental influences. The parameters of each path are constant. AAB is Adult Antisocial Behavior.

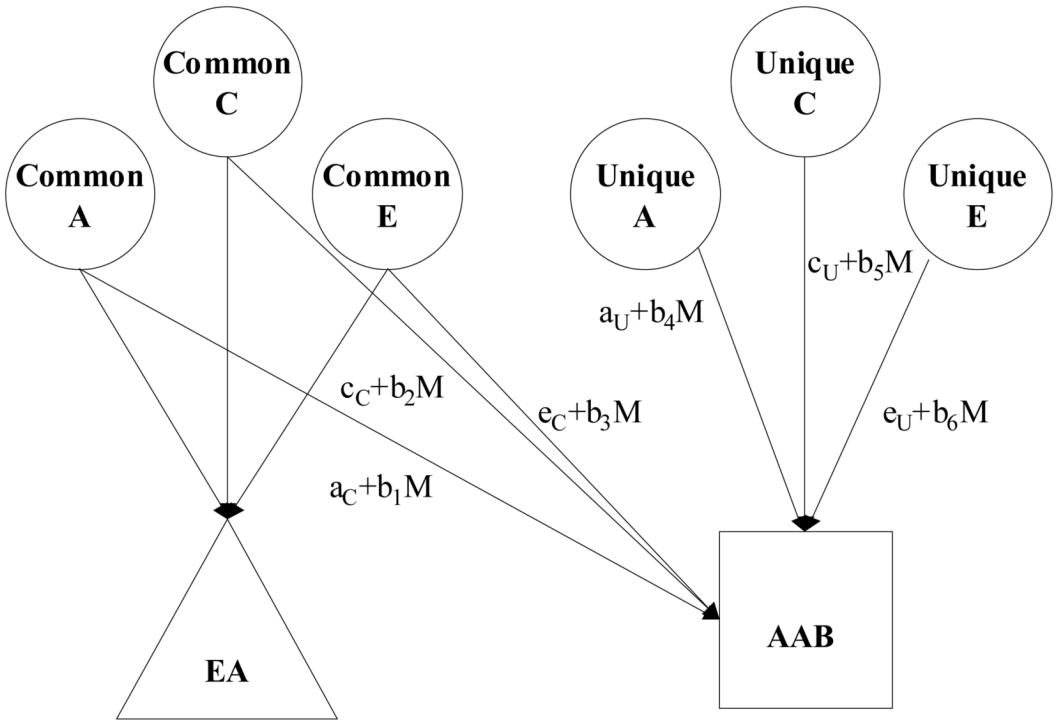

Figure 2.

Model of moderation of genetic and environmental influences by educational attainment in the presence of possible overlap in genetic and environmental influences on the two variables. A refers to genetic influences, C to shared environmental influences, and E to nonshared environmental influences. Under this model, educational attainment (EA) is represented as a triangle because we are conceptualizing it as an environmental variable exerting a moderating influence on Adult Antisocial Behavior (AAB). Variance in AAB can result from any combination of the labeled paths: A, C, and/or E that also influence EA, and/or A, C, and/or E unique to AAB. Each of these paths can vary with level of EA, noted above by M for moderator in the path equations. The model includes estimates of the paths representing influences on EA as well. These paths are constant and not labeled.

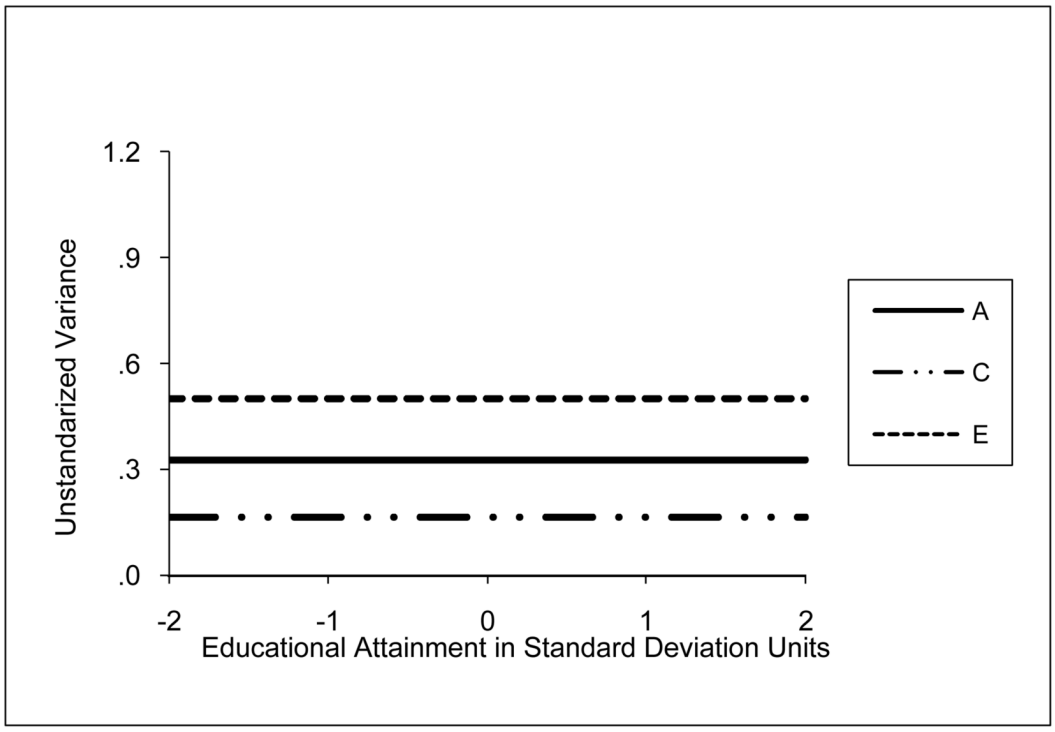

Figure 3.
Variance (unstamdarized) in Adult Antisocial Behavior as a function of Educational Attainment at age 24, by source of variance. A refers to genetic variance, C to shared environmental variance, and E to nonshared environmental variance.

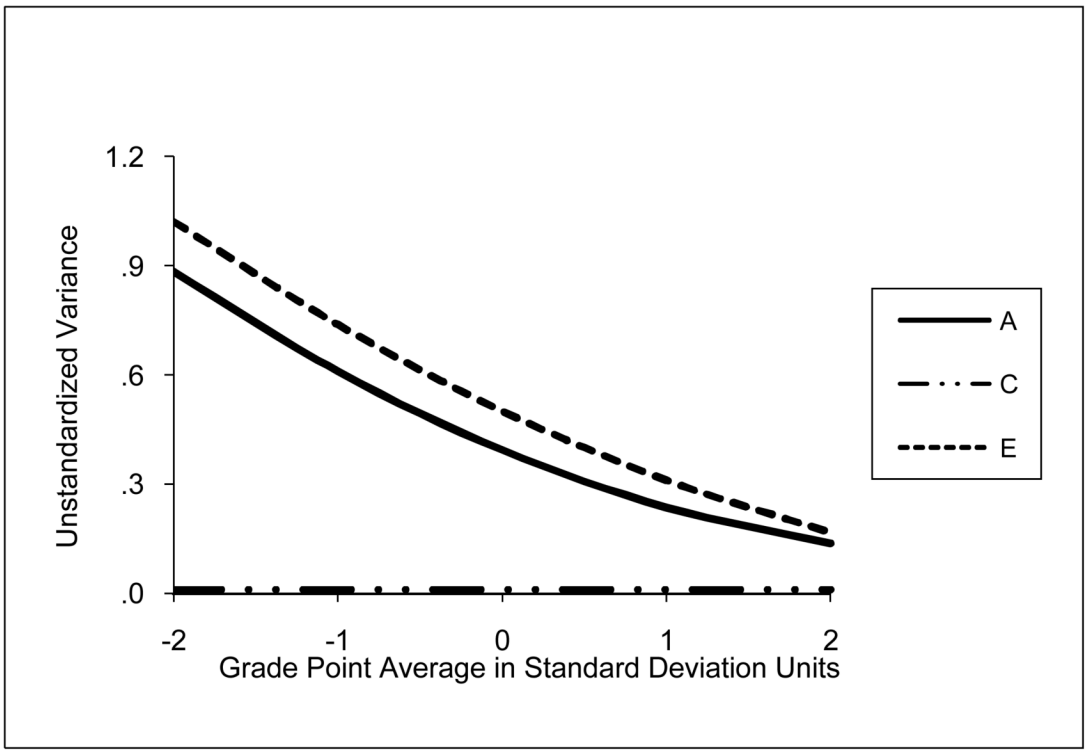

Figure 4.
Variance (unstandardized) in Adult Antisocial Behavior as a function of Grade Point Average at age 17, by source of variance. A referes to genetic variance, C to shared environmental variance and E to nonshared environmental variance.

Table 1

Zero-Order Correlations Among Study Variables

Variable	1	2	3	4	5	6	7
1. Parental SES	1.00						
2. WAIS IQ	.25	1.00					
3. WRAT Reading	.22	.48	1.00				
4. Age 17 GPA	.20	.31	.33	1.00			
5. Age 17 AAB	−.04	−.09	−.08	−.30	1.00		
6. Attained Education	.36	.32	.28	.47	−.31	1.00	
7. Age 24 AAB	−.06	−.05	−.05	−.20	.41	−.21	1.00

Note: SES is socioeconomic status; WAIS is Wechsler Adult Intelligence Scale; WRAT is Wide Range Achievement Test; GPA is grade point average; attained education was measured at age 24; AAB is log-transformed symptoms of DSM Adult Antisocial Behavior. Correlations of .09 or greater differ significantly from 0 at $p<.01$, adjusting for paired twin data.

Table 2

Twin Correlations of Study Variables

Variable	MZ	DZ
1. Parental SES	1.00	1.00
2. WAIS IQ at 17	.81	.51
3. WRAT Reading	.66	.23
4. Age 17 GPA	.63	.29
5. Age 17 AAB	.48	.21
6. Attained Education	.70	.56
7. Age 24 AAB	.46	.26

Note: SES is socioeconomic status; WAIS is Wechsler Adult Intelligence Scale; WRAT is Wide Range Achievement Test; GPA is grade point average attained education was measured at age 24; AAB is symptoms of DSM-IV Adult Antisocial Behavior; MZ is monozygotic; DZ is dizygotic. Correlations are double-entered Pearsons. N's varied due to sample attrition across the 7-year period between assessments and due to small amounts of missing data, but there were 411 MZ and 215 DZ pairs at age 17.

Table 3

Standardized Coefficients from Phenotypic Regression Models Associating AAB and Variables Related to School Performance at Ages 17 and 24

	Regression Coefficient	Standard Error	Significance Probability	R^2
Association Between Age 17 GPA and Age 17 AAB				
Parental SES	.02	.03	.47	.09
IQ	.00	.04	.98	
WRAT Reading	.00	.04	.94	
Age 17 GPA	−.29	.04	<.001	
Intercept	−.02	.03	.45	
Effect of Age 17 AAB on Age 24 Educational Attainment				
Age 17 GPA	.32	.04	<.001	.33
Parental SES	.23	.04	<.001	
IQ	.13	.04	<.001	
WRAT Reading	.03	.04	.40	
Age 17 AAB	−.16	.04	<.001	
Intercept	.00	.03	.91	
Effect of School Performance on Changes in AAB from 17 to 24				
Age 17 AAB	.38	11.12	<.001	.18
Parental SES	−.02	−.65	.52	
IQ	.02	.53	.60	
WRAT Reading	.00	.10	.92	
Age 17 GPA	−.07	−2.10	.03	
Age 24 Educational Attainment	−.03	−.76	.45	
Intercept	.00	.09	.93	

Note: SES is socioeconomic status; WAIS is Wechsler Adult Intelligence Scale; WRAT is Wide Range Achievement Test; GPA is grade point average; attained education was measured at age 24; AAB is log-transformed symptoms of DSM Adult Antisocial Behavior.

Table 4

Fit statistics from the models of variance components allowing for gene-environment interaction and correlation

Model	−2*LL	df	χ^2	Δdf	*p*	AIC
Age 24 AAB as a function of age 24 educational attainment						
All parameters free	3675.4	2154	---	---	---	3709.4
Fix all moderation paths	3687.4	2160	12.0	6	ns	3709.4
Age 17 AAB as a function of age 17 GPA						
All parameters free	6172.2	3599	---	---	---	6206.2
Fix common A, C, & E	6172.5	3602	.3	3	ns	6200.5
Fix common A, C, & E, and unique A moderation paths	6177.9	3603	5.4	1	ns	6203.9
Fix common A, C, & E, and unique C moderation paths*	6172.5	3603	.0	1	.020	6198.5
Fix common A, C, & E, and unique E moderation paths	6208.1	3603	35.6	1	<.001	6234.1
Fix common A, C, & E, and unique C & A moderation paths	6191.9	3604	19.4	2	<.001	6215.9
Fix common A, C, & E, and unique C & E moderation paths	6208.1	3604	35.6	2	<.001	6232.1
Fix common A, C, & E, and unique A & E moderation paths	6234.3	3604	61.8	2	<.001	6258.3
Fix all moderation paths	6299.4	3605	126.9	3	<.001	6321.4

Note: A refers to genetic influences, C to shared environmental influences, and E to nonshared environmental influences. There are possible common and unique moderation paths for each of A, C, and E. Best-fitting models are indicated by *. Fixed moderation paths were constrained to 0, which means that those sources of influence were present but did not vary across the levels of the moderators. AIC is Akaike Information Criterion.

Table 5

Estimates of variance components and proportions of variance in symptoms of Adult Antisocial Behavior and their moderating variables, and their genetic and environmental correlations

Variance components	Age 24 Educational Attainment	Age 24 AAB −2 *sd*	Age 24 AAB 0 *sd*	Age 24 AAB 2 *sd*
Genetic	.35 (.14,.60)	.33 (.00,.58)	.33 (.00,.58)	.33 (.00,.58)
Shared environmental	.33 (.10,.54)	.16 (.00,.46)	.16 (.00,.46)	.16 (.00,.46)
Nonshared environmental	.30 (.26,.36)	.50 (.42,.61)	.50 (.42,.61)	.50 (.42,.61)
Proportions of variance				
Genetic	.36 (.16,.58)	.33 (.00,.58)	.33 (.00,.58)	.33 (.00,.58)
Shared environmental	.34 (.12,.54)	.16 (.00,.46)	.16 (.00,.46)	.16 (.00,.46)
Nonshared environmental	.30 (.27,.35)	.51 (.43,.62)	.51 (.43,.62)	.51 (.43,.62)
Correlations with moderator				
Genetic		−.03 (−1.00,1.00)	−.03 (−1.00,1.00)	−.03 (−1.00,1.00)
Shared environmental		−.71 (−1.00,1.00)	−.71 (−1.00,1.00)	−.71 (−1.00,1.00)
Nonshared environmental		−.05 (−.18,.08)	−.05 (−.18,.08)	−.05 (−.18,.08)
Variance components	Age 17 GPA	Age 17 AAB −2 *sd*	Age 17 AAB 0 *sd*	Age 17 AAB 2 *sd*
Genetic	.70 (.52,.82)	.88 (.49,1.29)	.39 (.21,.51)	.14 (.04,.24)
Shared environmental	.03 (.00,.19)	.01 (−.02,.16)	.01 (−.02,.16)	.01 (−.02,.16)
Nonshared environmental	.27 (.23,.33)	1.02 (.77,1.32)	.50 (.44,.58)	.17 (.11,.26)
Proportions of variance				
Genetic	.70 (.52,.82)	.46 (.28,.61)	.43 (.23,.54)	.44 (.14,.73)
Shared environmental	.03 (.00,.19)	.01 (.00,.20)	.01 (.00,.20)	.03 (.00,.80)
Nonshared environmental	.27 (.23,.33)	.53 (.40,.68)	.56 (.48,.65)	.53 (.32,.73)
Correlations with moderator				
Genetic		−.33 (−.45, −.15)	−.49 (−.63,.71)	−.84 (−1.00,. −.52)
Shared environmental		−1.00 (−1.00,1.00)	−1.00 (−1.00,1.00)	−1.00 (−1.00,1.00)
Nonshared environmental		−.09 (−.16, −.02)	−.13 (.01,.23)	−.23 (−.39, −.05)

Note: The variance components are raw; they do not sum to 1.00. The proportions of variance sum to 1.00. 95% confidence intervals are given in parentheses. GPA is grade point average; AAB is log-transformed symptoms of DSM Adult Antisocial The variance components and correlations for AAB at each age are given at the mean levels of the moderating variables and at 2 standard deviations above and below their mean levels.