Why Don't Smart Teens Have Sex? A Behavioral Genetic Approach

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Academic achievement and cognitive ability have been shown to predict later age at first sexual intercourse. Using a sample of 536 same-sex twin pairs who were followed longitudinally from adolescence to early adulthood, this study tested whether relations between intelligence, academic achievement, and age at first sex were due to unmeasured genetic and environmental differences between families. Twins who differed in their intelligence or their academic achievement did not differ in their age at first sex. Rather, the association between intelligence and age at first sex could be attributed entirely to unmeasured environmental differences between families, whereas the association between academic achievement and age at first sex could be attributed entirely to genetic factors.

First sexual intercourse is a normative developmental transition, but when individuals reach this milestone is often a source of intense concern and contentious debate. In fact, current U.S. health policy has made reducing or delaying adolescent sexual involvement an explicit public health goal (U.S. Department of Health and Human Services, 2000). This policy is motivated by the potential negative health consequences of unprotected intercourse, namely, sexually transmitted infection and unintended pregnancy. Because adolescents and young adults use condoms less consistently and less effectively than older adults, they account for a disproportionate number of new sexually transmitted infections, including over 30% of new HIV diagnoses (Centers for Disease Control, 2006). The United States, moreover, has one of the highest teenage pregnancy and teenage childbearing rates in the industrialized world (United Nations, 2008). These

concerns about the negative health consequences of teenage sexual activity have motivated a large corpus of psychological, sociological, and epidemiological research regarding both the "upstream" and "downstream" correlates of individual differences in age at first intercourse.

One critical precursor of teenage sexual behavior is educational performance. Research has consistently demonstrated a relation between higher academic achievement and later ages at first intercourse (Kirby, 2002a; Miller & Sneesby, 1988). School attendance and sense of connectedness to school all further decrease likelihood of sexual activity (Kirby, 2002b) and adolescents who become sexually active early in high school are less likely than peers to earn either a high school diploma or attend college (Frisco, 2008; Spriggs & Halpern, 2008). This association seems to have some intergenerational component; not only does educational achievement predict teens' own likelihood of engaging in sexual activity, but level of parental education also significantly predicts offspring age of first intercourse (Miller et al., 1997; Rosenthal et al., 2001; Schvaneveldt, Miller, Berry, & Lee, 2001).

Additionally, intelligence seems to play a role in sexual timing. A small, but consistent, body of research suggests intelligence is inversely associated with age at first sex. Halpern, Joyner, Udry, and Suchindran (2000) found that adolescents who scored highly on the Peabody Picture Vocabulary

Test (PPVT; a measure of verbal intelligence) were less likely to have had sexual intercourse than same-age peers, an association that persisted even after controlling for a breadth of statistical covariates typically associated both with intelligence and sexual activity: race/ethnicity, physical maturity, current academic achievement, physical and personal attractiveness, expectations regarding the future (attending college, mortality), perceived consequences of unintended pregnancy, religiousness, involvement in sports and clubs, current romantic relationships, maternal education, family structure, and maternal attitudes toward sex. Halpern et al.'s work offered convergent validity for previous studies that obtained this association in different samples and with different measures of intelligence, including the Armed Forces Qualifications Test (Mott, 1983) and Ravens Progressive Matrices (Cliquet & Balcaen, 1983). Similarly, other research implicates associations between performance on IQ tests and other aspects of adolescent sexual activity, such as teenage pregnancy and number of sexual partners during high school (Fergusson, Horwood, & Ridder, 2005; Shearer et al., 2002).

The associations between intelligence, academic achievement, and sexual activity are often interpreted in terms of the ''safeguarding'' hypothesis (e.g., Kirby, 2002b; Manlove, 1998; Ohannessian & Crockett, 1993): Smart and motivated teenagers have higher aspirations for their future (e.g., attending college, professional training) and thus have ''more to lose'' from an unintended pregnancy or sexually transmitted infection. Moreover, they are better able to anticipate that sexual activity might lead to these negative consequences. They therefore abstain from sexual activity as a means of safeguarding or securing their future accomplishments. This hypothesis is buoyed by a substantial body of work that associates timing of intercourse with educational aspirations, suggesting that individual goals are, to some degree, related to sexual behavior (Harris, Duncan, & Boisjoly, 2002; Hogan & Kitagawa, 1985; Jessor & Jessor, 1975; Miller & Sneesby, 1988; Schvaneveldt et al., 2001).

Despite its intuitive appeal, the safeguarding hypothesis relies on a problematic, *cognitive-development* model of adolescent risk taking. In the cognitive-development model, adolescent risk-taking behaviors are the result of flawed and immature reasoning processes, in which adolescents systematically underestimate the potential costs of a behavior. Thus, higher and more accurate perceptions of potential costs (whether due to educational interventions, higher cognitive ability, or maturation) are thought to lead to reductions in risk-taking behaviors. Research in cognitive psychology and neuroscience, however, has demonstrated that the premises of the cognitive-development model are inaccurate in three respects (Figner, Mackinlay, Wilkening, & Weber, 2009). First, adolescents do not show markedly immature reasoning abilities compared to adults; rather, logical reasoning and basic information processing abilities have developed to nearly adult levels by age 16 (Keating, 2004; Yurgelun-Todd, 2007). Second, adolescents do not systematically underestimate the potential costs of risk-taking behaviors; they can actually evaluate the potential consequences, costs and benefits of various risky behaviors (including having sex) as well as adults can (Beyth-Marom, Austin, Fischhoff, Palmgren, & Jacobs- Quadrel, 1993). It is true that adolescents with higher intelligence do perceive higher costs associated with sexual activity (e.g., feelings of guilt) and with unintended pregnancy (e.g., embarrassment, having to quit school), but they also perceive higher *benefits* (e.g., physical pleasure, decreased loneliness; Deptula, Henry, Shoeny, & Slavick, 2006). Third—and perhaps most importantly—adolescents' deliberative decision making and judgment seems to lapse during emotionally charged situations (Casey, Getz, & Galvan, 2008; Dahl, 2004; Figner et al., 2009). That is, while adolescents may be able to articulate the potential liabilities of a particular action during moments of low emotion, they seem to forget or ignore this in the ''heat of the moment.'' Casey et al. (2008) and Steinberg (2008) have linked this paradox to a developmental ''maturity gap,'' in which parts of the limbic system are remodeled in early and middle adolescence, while the prefrontal cortex continues to mature through early adulthood. This leaves adolescents more highly attuned to affect and rewards while lacking fully developed capacities for inhibition. Given this emerging knowledge about adolescent neural development and cognition, a teenager's rational calculus regarding the potential impact of sexual intercourse on long-term educational goals may be an ineffective ''brake'' for sexual behavior, which necessarily occurs in an affectively charged interpersonal context.

An alternative explanation for previously observed correlations between age at first sex and intelligence and academic achievement is that the same family background factors—either genetic or environmental in origin—contribute to both cognitive and sexual outcomes. That is, the ''type'' of families who have sexually active adolescents may also be the ''type'' of families who have adolescents

who perform poorly in school. Timing of sexual intercourse, intelligence, and academic achievement are all complexly intertwined with factors such as socioeconomic privilege, family, peers, qualities of schools and neighborhoods, and cultural belief systems. Researchers typically control for environmental variables by identifying probable confounds and including them in analyses as statistical covariates. Yet, it is simply impossible to account for all potentially relevant confounds: It depends on which variables are available in the data, whether a researcher chooses to include these variables in analyses, and how well these variables represent the constructs of interest. These limitations, inherent in all purely correlational research, suggest that the existing findings on age at first sex, intelligence, and academic achievement have likely been muddied by ''background'' variables that may not have been adequately accounted for in the literature to date.

In addition, traditional correlational designs rarely sample more than one person per biological family, and thus are typically incapable of controlling for *genetic* confounds. It is well established that there is substantial genetic variation in both intelligence (e.g., Bouchard, 2004) and academic achievement (e.g., Thompson, Dettermann, & Plomin, 1991). There is also genetic variance in age at first sex (Bailey, Kirk, Zhu, Dunne, & Martin, 2000; Dunne et al., 1997; Lyons et al., 2004; Martin, Eaves, & Eysenck, 1977), and specific genes related to dopamine receptors have been associated with earlier age at first sex, particularly in males (Miller et al., 1999). If the same genes were related both to lower IQ scores, achievement, and earlier age at first sex, then the association between achievement and age at first sex would be an artifact of a common underlying genetic liability. For example, performance on intellectual or academic assessments and timing of first sexual intercourse might both be influenced by genes related to traits such as impulsivity (resulting in quick, hastily reasoned answers during assessments and spontaneous involvement in sexually charged encounters) or antisociality (resulting in failures to conform to societal expectations regarding both sexual inhibition and school performance).

Behavioral genetic designs, including twin studies, are a rigorous methodology for testing how genetic and environmental background factors might influence relations between higher intelligence, higher academic achievement, and timing of first sex. Although twin designs are typically perceived as a means for estimating heritability coefficients, this is not our aim in the current article.

Rather, behavior genetic researchers are increasingly moving beyond heritability estimates and are instead leveraging the power of twin studies to test specific developmental hypotheses (Moffitt, 2005; Rutter, Pickles, Murray, & Eaves, 2001). In the present study, a behavior genetic approach allows us to reevaluate whether the relation between intelligence and academic achievement and age at first sex persists even after controlling for genetic and environmental confounds that differ between families.

To illustrate the logic of this approach, imagine a case of two monozygotic (MZ) twins, in which Twin A exhibits higher academic achievement and Twin B exhibits lower academic achievement. The critical question is whether Twin A and Twin B also differ in their age at first intercourse. This within-twin pair comparison controls for all genetic variables *and* all environmental variables shared by twins raised in the same home (e.g., family structure, socioeconomic status, neighborhood quality). If higher academic achievement somehow induces adolescents to delay sexual activity to protect educational and occupational goals, then Twin A will initiate sexual activity significantly later than Twin B. If, however, unmeasured genetic or environmental factors that differ between families account for the association between academic achievement and age at first sex, then Twin A and Twin B will demonstrate similar ages at first sex, despite their differing academic performance.

Behavioral genetic studies of sexual development have yielded novel and surprising results. Previously, we used a sample of twins from the National Longitudinal Study of Adolescent Health to examine the association between age at first sex and later delinquency during early adulthood (ages 18–24). After controlling for genetic and shared environmental factors that differ between families, earlier age at first sex actually predicted *lower* involvement in delinquency in early adulthood (Harden, Mendle, Hill, Turkheimer, & Emery, 2008). Using a similar analytic approach in a sample of female twin pairs from Denmark, Rodgers et al. (2008) examined the relation between intelligence, educational attainment, and age at first birth. They found that the effects of intelligence and education on age at first birth were primarily accounted for by shared environmental factors. That is, in a comparison of two sisters who differed in their intelligence and education, the smarter or more highly educated sister did *not* show a later age at first birth; rather, the effects of intelligence and education on age at first birth were entirely between families. Rodgers et al.

interpreted this to mean that intelligence and education are not causal agents for delayed fertility but rather ''proxies'' for between-family differences in family environment and cultural context that account for the later age at first birth evident among more intelligent or more educated women.

The Present Study

In this article, we use a sample of same-sex adolescent twin pairs from the National Longitudinal Study of Adolescent Health to examine associations between intelligence (as measured by the PPVT), academic achievement (as measured by grade point average [GPA]), and age at first sex. Specifically, we investigate whether adolescent twins who differ in their intelligence and academic achievement also differ in their timing of first sexual intercourse. We hypothesize that this association will be accounted for by unmeasured shared environmental and genetic differences between families. Given Rodgers et al.'s (2008) findings on education and age at first birth, we specifically hypothesize that the association will be due to socioenvironmental and cultural differences among families. In behavior genetic analyses, this would present itself as a significant effect of the ''shared environment.''

Method

Participants

Data were drawn from the National Longitudinal Study of Adolescent Health (AddHealth; Harris, 2005), a nationally representative study designed to assess adolescent health and risk behavior collected in three waves between 1994 and 2002. Sampling for AddHealth began with identification of all high schools in the United States that had at least 30 enrollees ($N = 26,666$). Schools were stratified according to geographic region, urbanicity, school size or type, and racial composition. From these strata, a random sample of schools was selected, some of which ranged from Grades 7–12 and some from Grades 9–12. If the school did not include seventh or eighth grade, the study recruited students from the feeder middle school sending students to that high school. Overall, 79% of the schools selected agreed to participate (final sample $N = 134$ schools). School population ranged from under 100 students to over 3000 students.

Ninety-six percent of the participating schools ($N = 129$) agreed to have students ($N = 90,118$) complete a confidential in-school survey during the 1994–1995 academic year. From the rosters of participating schools, a randomly selected subsample of 20,745 completed a follow-up, 90-min In-Home interview between April and December 1995 (Wave I interview; 10,480 female, 10,264 male). Participants ranged in age from 11 to 21 ($M = 16$ years, 25th percentile = 14 years, 75th percentile = 17 years). There have been three follow-up interviews with the Add Health participants: Wave II in 1996, Wave III in August 2001–2002, and Wave IV in 2007–2008. At the time of the Wave IV interview, participants were between 24 and 32 years old.

During the initial In-School interview, adolescents were asked whether they currently lived with another adolescent in the same household. This information was used to deliberately over-sample adolescent sibling pairs, even if one member of the pair did not attend a high school in the original probability sample. (Most of the full sibling pairs were obtained serendipitously, because both siblings attended a sampled high school.) Among the sibling pairs, there are 534 same-sex twin pairs, who are the focus of the current analyses. Twins were classified as either MZ, sharing 100% of their genes, or dizygotic (DZ), sharing 50% of their genes. Twin zygosity was determined primarily on the basis of self-report and responses to four questionnaire items concerning similarity of appearance and frequency of being confused for one's twin. Similar questionnaires have been utilized widely in twin research and have been repeatedly cross-validated with zygosity determinations based on DNA (e.g., Loehlin & Nichols, 1976; Spitz et al., 1996). There were 144 male–male MZ, 145 female–female MZ, 131 male–male DZ, and 114 female–female DZ pairs. Analyses were restricted to same-sex twins in order to prevent bias in estimates of genetic influence due to MZ twins necessarily being identical for sex. That is, to the extent that there are gender-specific differences in the etiology of age at first sex (such as differences in parental monitoring or other parenting between girls and boys, or different cultural expectations regarding the ''appropriate'' age for sexual initiation), same-sex MZ twins would be more similar than opposite-sex DZ twins, even if there were no genetic influences on age at first sex. Just over half of the twin pairs were non-Hispanic White (55.4%, $N = 297$ pairs), 125 (23.3%) were non-Hispanic Black, 80 (14.9%) were Hispanic/Latino, 21 (3.9%) were Asian, and the remaining 13 pairs (2.4%) reported they were another race/ethnicity or did not report race/

ethnicity. Jacobson and Rowe (1999) compared the sociodemographic composition of sibling pairs with the full AddHealth sample and found negligible differences.

Measures

The AddHealth interviews measured a broad array of health domains, including current mental, physical, emotional, and sexual health; exercise and diet; drug, tobacco, and alcohol use; family patterns of illness and disease; family relationships; peer influences; criminal and delinquent activity; school policies; and access to community services. The survey and its components were adapted from numerous sources (see Udry, 2003), but no intact scales from the literature were included in the survey. Questionnaire items were extensively pilot tested before use.

Intelligence. An abridged, computerized version of the PPVT–Revised (PPVT–R) was used in the AddHealth study (AddHealth Picture Vocabulary Test [AHPVT]; Halpern et al., 2000). The full PPVT–R correlates 0.62 with the Stanford-Binet and 0.64 with the Wechsler Intelligence Scale for Children (Full Scale IQ) and thus can be considered a measure of general intelligence. The AHPVT was constructed by selecting every other item in the full PPVT–R, resulting in 78 items. Basal and ceiling rules were changed to account for the smaller total number of items. In the AHPVT, the interviewer reads a word and the participant selects the picture (from four response options) that best corresponds to the definition of the word. Illustrations in the AHPVT were unchanged from the full PPVT–R. Raw scores were age-standardized based on responses of AddHealth participants. Because of the large sample size (total $N = 19,713$) of the AddHealth study, the standardization sample for the AHPVT actually exceeded that of the full PPVT–R ($N = 4,200$). Scaled scores on the AHPVT are in a standardized intelligence test metric, with a mean in the full sample equal to 100 and standard deviation equal to 15, and thus scores on the AHPVT will be abbreviated IQ throughout the current article. Within the same-sex twin pairs subsample, the mean AHPVT score was 98.0 ($SD = 15.0$). The twin pair correlation for AHPVT scores was 0.78 in MZ twins and 0.56 in DZ twins.

Academic achievement. During the Wave I In-Home interview, participants reported their grades in English/language arts, mathematics, history/social studies, and science during the most recent grading period, on a 1 (A) to 4 (D or lower) scale. Grades in the four subjects were averaged to yield a measure of overall GPA. The mean GPA was 2.17 ($SD = 0.74$). The twin correlation for GPA was 0.64 in MZ pairs and 0.48 in DZ pairs. Academic achievement was significantly correlated with cognitive ability ($r = -.31$).

Age at first sex. At Wave IV, participants reported whether they had ever had vaginal intercourse and their age (in years) at first intercourse. Nearly all participants had had sex by Wave IV (92.45%), and there were only 9 of 536 pairs in which both twins reported being virgins. Participants who were still virgins by Wave IV did not significantly differ from nonvirgins with respect to intelligence, GPA, parental education, parental income, or race/ethnicity (full results available upon request). When nonvirgin participants reported an age at first sex that was likely prepubescent and potentially nonconsensual (< 11 years, $N = 8$), this age was replaced with a missing value, resulting in an age at first sex variable ranging from 11.00 to 28.00 years ($M = 17.34$, median = 17.00, mode = 18.00, 25%–75% = 15–19; $SD = 2.90$). Of the 1,072 individual twins, 67 were missing data for age at first sex because they had not yet had sex and 184 did not report an age at first sex, resulting in available data for 821 individuals. All twin pairs were included in structural equation models, even when they had missing data for age at first sex, because they were informative regarding covariation between twins' intelligence and academic achievement. Twin pairs with missing age at first sex data, however, were not informative regarding the role of genetic factors in age at first sex. The correlations between age at first sex in the first and second twin of each pair was 0.48 in DZ pairs and 0.55 in MZ pairs. Age at first sex was significantly correlated with both academic achievement ($r = -.22$; remember that GPA was scored such that lower scores corresponded to better grades) and cognitive ability ($r = .19$).

Pubertal development. At Wave I, participants rated, ''How advanced is your physical development relative to other boys/girls your age?'' on a 5-point scale ranging from *I look younger than most* to *I look older than most*. Forty percent of participants rated their pubertal development as average relative to their peers, whereas 24% reported less advanced pubertal development than some or most of their peers and 36% reported more advanced pubertal development than some or most of their peers. Adolescents' reports of their pubertal development relative to peers were correlated 0.51 in MZ twins and 0.18 in DZ twins. Pubertal development relative to peers had minimal correlations with

academic achievement ($r = -.02$), age at first sex ($r = -.04$), and intelligence ($r = .13$).

Socioeconomic status.   Mothers responded whether they were receiving public assistance, their family's total pretax income in that past year, and how far they went in school. Less than 8% of the sample ($N = 42$ twin pairs) had mothers currently receiving public assistance. Family income ranged from $0 to $800,000 ($M = $47,000, 25th percentile = $22,000, 75th percentile = $60,000). Because the distribution of income had a long right tail, income was square root transformed and divided by 1,000 to yield a variable ranging from 0 to 28.28 ($M = 6.36$, $SD = 2.53$). Maternal education was coded on a 10-point scale (0 = *never went to school* [$N = 1$]; 1 = *8th grade or less* [$N = 32$]; 2 = *more than 8th grade but did not graduate from high school* [$N = 43$]; 3 = *went to business, trade, or vocational school instead of high school* [$N = 6$]; 4 = *high school graduate* [$N = 103$]; 5 = *completed a GED* [$N = 11$]; 6 = *went to business, trade, or vocational school after high school* [$N = 32$]; 7 = *went to college but did not graduate* [$N = 115$]; 8 = *graduated from college or university* [$N = 65$]; and 9 = *professional training beyond a 4-year college or university* [$N = 46$]).

Analyses

Means comparisons. As an initial descriptive analysis, we used IQ scores, dichotomized into ''higher'' and ''lower'' at the mean (100), to classify adolescents into four groups:

1. Adolescents with lower IQ (< 100) whose cotwins also had lower IQ.
2. Adolescents with lower IQ whose cotwins had higher IQ (> 100).
3. Adolescents with higher IQ whose cotwins had lower IQ.
4. Adolescents with higher IQ whose cotwins also had higher IQ.

A comparison between Group 1 (twins concordant for lower IQ) and Group 4 (twins concordant for higher IQ) is analogous to a traditional between-families analysis: Do adolescents with higher IQ show later ages at first sex compared to unrelated adolescents with lower IQ? In contrast, a comparison between Group 2 and Group 3 is a within-families analysis: Among twins discordant for high versus lower IQ, do the twins with higher IQ (Group 3) have later ages at first sex than their cotwins with lower IQ (Group 2)? The degree to which the between-family effect (Groups 1 vs. 4) is reduced in the within-family comparisons (Groups 2 vs. 3) indicates that unmeasured differences between families confound the association between intelligence and age at first sex.

We conducted these means comparisons in both the full sample of twins, and then separately for MZ and DZ twins. Conducting analyses separately by zygosity allows us to establish whether confounding variables are genetic or environmental in origin. Both MZ and DZ twins control for environmental factors shared by adolescents who have been raised in the same home. Therefore, if the association between intelligence and age at first sex is due to unmeasured environmental differences between families, the within-families effect (i.e., the difference between Groups 2 and 3) will be negligible in both MZ and DZ twins. However, if the association between intelligence and age at first sex is due to unmeasured genetic differences between families, then the within-families effect will be stronger when comparing discordant DZ twins (who only share 50% of their genes) than when comparing MZ twins (who share 100% of their genes).

Finally, we repeated the means comparisons using GPA, dichotomized into ''higher'' and ''lower'' relative to the sample mean (2). This dichotomization corresponds to grades that are mostly A's and B's (''higher GPA'') versus grades that are mostly C's or D's (''lower GPA'').

Multivariate twin models. Descriptive means comparisons can be useful for illustrating between-family and within-family associations. However, they use an artificial cutoff to classify twins into groups. This can result in twins who were very similar in intelligence or achievement but who straddled the cutoff point being classified as discordant, while other twins who were very different but both above or below the cutoff being classified as concordant. In order to analyze differences between twins continuously, and to estimate the role of sampling error accurately, we next fit a series of multivariate twin models in the software program Mplus (Muthén & Muthén, 1998–2007). We used a model-building approach to progress systematically from simpler to complex models (all models listed in Table 2). The first stage of model fitting focused on the relation between intelligence and age at first sex, and we conducted a series of nested model comparisons to select the best fitting and most parsimonious model. The best fitting model was then carried forward for models that incorporated additional measures of the shared environment (socioeconomic status, race/ethnicity),

to see if they accounted for the relation between intelligence and age at first sex. The next stage of model fitting examined the relation between intelligence, achievement, and age at first sex, and we again fit a series of nested model comparisons to arrive at the best fitting model. Finally, pubertal development was incorporated as a statistical covariate. Nested models were compared using differences in the models' chi-square values. Differences in chi-square are themselves chi-square distributed, and a significant ($p < .05$) chi-square difference indicates that the more constrained model fits significantly worse than the full model. In addition, model fit was evaluated using root mean square error of approximation (RMSEA), which measures error in approximating data from the model per parameter (Steiger, 1990) and gives useful information about how well a model approximates population values (Browne & Cudeck, 1993). RMSEA values of less than 0.05 indicate a close fit to the data, and values up to 0.08 represent reasonable errors of approximation.

Results

Means Comparisons

Results from the means comparisons are shown in Table 1. Because this analysis was intended to be purely descriptive, no inferential statistics or probability values were computed. The role of sampling error will be considered in the subsequent behavior genetic models, which appropriately deal with the nested structure of the data (adolescents within twin pairs). For the IQ comparisons using all twins, twins concordant for lower IQ had sex more than 1 year earlier ($M = 16.76$) than twins concordant for higher IQ ($M = 18.01$). Similarly, for the GPA comparisons using all twins, twins concordant for lower GPA had sex more than 1 year earlier ($M = 16.79$) than twins concordant for higher GPA ($M = 17.94$). These between-family mean differences, however, were substantially reduced when comparing within families. Among all twins discordant for lower IQ, twins with lower IQ were actually slightly older at first sex ($M = 17.54$) as their cotwins with higher IQ ($M = 17.17$). Similarly, among all twins discordant for lower GPA, twins with lower GPA had sex only slightly earlier ($M = 17.07$) than their cotwins with higher GPA ($M = 17.19$).

Results from the means comparisons are illustrated in Figure 1, which plots the mean difference between groups expressed as an effect size (Cohen's *d*). The bars on the far left represent the mean difference between adolescent twins concordant for lower IQ or lower GPA (Group 1) and adolescent twins concordant for higher IQ or higher GPA (Group 4). This mean difference ($d = 0.43$ for IQ and 0.41 for GPA) is confounded by unmeasured environmental and genetic differences between families. The bars in the middle represent the mean differences between DZ twins discordant for low versus higher IQ (black bar) or discordant for low versus higher GPA (gray bar). The DZ twin comparison controls for all shared environmental confounds and 50% of genetic confounds. Finally, the bars on the far right represent the mean

Table 1
Mean Age at First Sex for Twins Concordant and Discordant for IQ and GPA

	All twins			MZ twins			DZ twins		
	N	M	SD	N	M	SD	N	M	SD
IQ comparison group[a]									
1. Low IQ/twin low IQ	325	16.76	2.82	185	16.71	2.56	140	16.82	3.14
2. Low IQ/twin high IQ	105	17.54	2.79	46	17.50	2.84	59	17.58	2.78
3. High IQ/twin low IQ	102	17.17	2.47	44	17.18	2.20	58	17.16	2.67
4. High IQ/twin high IQ	269	18.01	3.03	156	18.48	3.06	113	17.38	2.89
GPA comparison group[b]									
1. Low GPA/twin low GPA	238	16.79	2.67	125	17.02	2.70	113	16.54	1.63
2. Low GPA/twin high GPA	138	17.07	3.07	66	17.20	3.43	72	16.96	2.71
3. High GPA/twin low GPA	122	17.19	2.88	58	17.05	2.63	64	17.31	3.11
4. High GPA/twin high GPA	303	17.94	2.89	182	18.04	2.71	121	17.79	3.15

[a]''Low IQ'' twins scored less than 100 on the AddHealth Picture Vocabulary Test (AHPVT); ''high IQ'' twins scored above 100.
[b]''Low GPA'' twins reported average grades that were C's or D's, whereas ''high GPA'' twins reported average grades that were A's or B's.
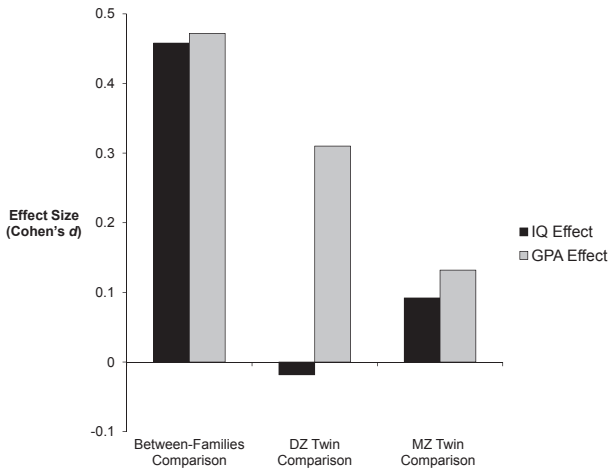
Figure 1. Effect sizes of IQ and GPA on age at first sex, when comparing unrelated adolescents, discordant dizygotic (DZ) twins, and discordant monozygotic (MZ) twins.

differences between MZ twins discordant for low versus higher IQ (black bar) or discordant for low versus higher GPA (gray bar). The MZ twin comparison controls for all shared environmental and all genetic confounds. The mean difference in age at first sex between twins discordant for IQ is negligible (and in the opposite direction) relative to the between-family effect for both DZs ($d = -0.15$) and MZs ($d = -0.13$). This suggests that the association between intelligence and age at first sex is due to unmeasured environmental confounds that differ between families. In contrast, the mean difference in age at first sex between discordant for GPA is significantly reduced in MZ twins ($d = -0.05$), while the DZ twin effect ($d = 0.12$) is intermediate to the between-families effect and the MZ twins effect. This pattern of results suggests that the association between academic achievement and age at first sex is due to unmeasured *genetic* confounds. These results will be further clarified by results from multivariate twin models.

Multivariate Twin Models

Intelligence and age at first sex.   As an initial step, we fit a bivariate twin model of the relation between intelligence and age at first sex (Model 1 in Table 2; illustrated in Figure 2). The left and right sides of Figure 2 represent the first and second twin, respectively. Variance in both intelligence and age at first sex was decomposed into three latent factors: (a) additive genetic influences, or *A*; (b) environmental influences that make twins more similar (i.e., the shared environment), or *C*; and (c)

environmental influences that make twins different (i.e., the nonshared environment), plus measurement error, or *E*. Together, these latent factors are often called *ACE* components. In this model, the paths from the *ACE* components to the phenotype (intelligence or age at first sex) are fixed to 1.0, and the variances of the *ACE* components are freely estimated. The sum of their variances equals the total variance in the phenotype. The estimated genetic variance (*A*) divided by the total variance equals the familiar heritability statistic. A more comprehensive explanation of the analysis of twin data is available in Neale and Cardon (1992).

The key part of the bivariate twin model are the regression paths from age at first sex on intelligence (b_{intel}) and on the *A* ($b_{A-intel}$) and *C* ($b_{C-intel}$) components of intelligence. The regression on *A* tests whether genetic factors that influence intelligence predict age at first sex. The regression on *C* tests whether environmental factors that influence intelligence predict age at first sex. Finally, the regression on intelligence itself tests whether, *after* controlling for these genetic and shared environmental factors, higher intelligence predicts later age at first sex. In other words, if twins differ in their intelligence, do they also differ in their age at first sex? (No separate regression on *E* is estimated, because within-twin pair differences in intelligence are perfectly confounded with nonshared environmental influences on intelligence. Indeed, an alternative specification of this model is to regress age at first sex on *E* rather than on phenotypic intelligence; the parameter estimate for the regression on *E* would be identical to the parameter estimate for the regression on intelligence.)

To test the significance of these regression paths, we compared three nested models (see Table 2: Model 1b, Model 1c, and Model 1d) with the full bivariate twin model. Results from these model comparisons are summarized in Table 2. Model 1b, which fixed the regressions on the *A* ($b_{A-intel}$) and *C* ($b_{C-intel}$) components of intelligence to zero, fit significantly worse than the full model, indicating that genetic and shared environmental differences between families significantly contributed to the association between intelligence and age at first sex and could not be eliminated from the model. In contrast, Model 1c, which fixed the regression on the *A* component of intelligence ($b_{A-intel}$) and the phenotypic regression on intelligence (b_{intel}) to zero, did not fit significantly worse than the full model. This indicates that twins who differed in their intelligence do not significantly differ in their age at first sex; rather, the relation between intelligence and

Table 2
Series of Multivariate Twin Models

Nested models	Variables included	Model specification	Indices of model fit		
			RMSEA	χ^2	$\Delta\chi^2$ (Δdf, p)
Model 1	IQ and age at first sex	Full model (see Figure 2)	0.000	18.90	–
Model 1b	IQ and age at first sex	No confounds ($b_{A\text{-intel}} = 0$; $b_{C\text{-intel}} = 0$)	0.047	36.85	**17.95 (2, <.001)**
Model 1c	IQ and age at first sex	*C* confounds only ($b_{A\text{-intel}} = 0$; $b_{intel} = 0$)	0.000	21.47	2.57 (2, .27)
Model 1d	IQ and age at first sex	*A* confounds only ($b_{C\text{-intel}} = 0$; $b_{intel} = 0$)	0.037	31.36	**12.46 (2, .001)**
Model 2	IQ, age at first sex, and race/ethnicity	Full model	0.023	40.05	–
Model 3	IQ, age at first sex, race/ethnicity, and SES (parental education, parental income, and receipt of public assistance)	Full model	0.037	76.54	–
Model 4	IQ, age at first sex, race/ethnicity, SES, and GPA	Full model (see Figure 4)	0.021	114.33	–
Model 4b	IQ, age at first sex, race/ethnicity, SES, and GPA	No confounds ($b_{A\text{-GPA}} = 0$; $b_{C\text{-GPA}} = 0$)	0.02	120.35	**6.02 (2, .049)**
Model 4c	IQ, age at first sex, race/ethnicity, SES, and GPA	*C* confounds only ($b_{A\text{-GPA}} = 0$; $b_{GPA} = 0$)	0.02	121.13	**6.80 (2, .033)**
Model 4d	IQ, age at first sex, race/ethnicity, SES, and GPA	*A* confounds only ($b_{C\text{-GPA}} = 0$; $b_{GPA} = 0$)	0.01	115.77	1.44 (2, .487)
Model 5	IQ, age at first sex, race/ethnicity, SES, GPA, and pubertal development	Full model	0.02	119.36	–

Note. Best fitting model from each nested model comparison is shaded in gray. Boldface indicates significant at $p < .05$. RMSEA = root mean square error of approximation; SES = socioeconomic status; GPA = grade point average.
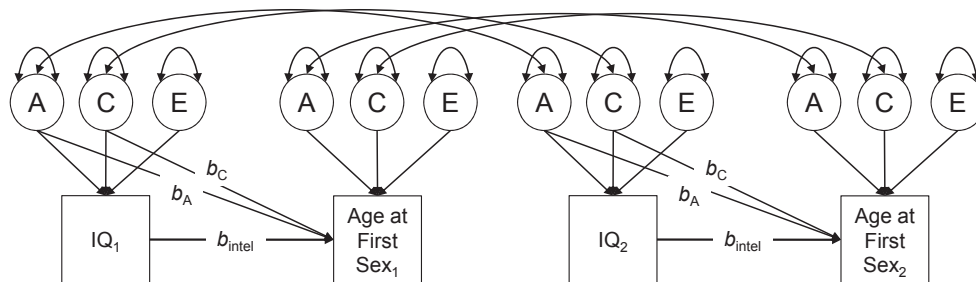

Figure 2. Bivariate twin model of intelligence and age at first sex.
Note. Variances of *ACE* components freely estimated. Correlations between *A* components fixed to equal 1.0 in MZ twins and 0.5 in DZ twins. Correlations between *C* components fixed to 1.0 in all twins. Gender (male = 1), African American race, Hispanic/Latino ethnicity, square root of parental income, maternal education, and receipt of public assistance included as statistical covariates of IQ and age at first sex (not illustrated).

age at first sex could be entirely accounted for by the unmeasured environmental differences between families (aka, shared environmental confounds). Finally, Model 1d, which fixed the regression on the *C* component of intelligence ($b_{C\text{-intel}}$) and the phenotypic regression on intelligence (b_{intel}) to zero, did fit significantly worse than the full model. This indicates that the relation between intelligence and age at first sex could not be solely accounted for by genetic confounds. Overall, these results are consistent with the means comparisons (described above), which also indicated that the association between intelligence and age at first sex was due to shared environmental confounds.

Table 3
Parameter Estimates of Best Fitting Models From Nested Model Comparisons

Parameter	Model 1c IQ and age at first sex		Model 2 IQ and age at first sex, controlling for race/ethnicity		Model 3 IQ and age at first sex, controlling for race/ethnicity & SES		Model 4d IQ, GPA, and age at first sex, controlling for race/ethnicity & SES		Model 5 IQ, GPA, and age at first sex, controlling for sex, race/ethnicity, SES, & pubertal development	
	Estimate	Standardized estimate (95% CI)	Estimate	Standardized estimate (95% CI)	Estimate	Standardized estimate (95% CI)	Estimate	Standardized estimate (95% CI)	Estimate	Standardized estimate (95% CI)
IQ regressions										
IQ → Age at first sex	[0]	[0]	[0]	[0]	[0]	[0]	[0]	[0]	[0]	[0]
A_{IQ} → Age at first sex	[0]	[0]	[0]	[0]	[0]	[0]	[0]	[0]	[0]	[0]
C_{IQ} → Age at first sex	1.73	0.34 (0.20, 0.49)	2.28	0.35 (0.15, 0.54)	2.96	0.36 (0.10, 0.63)	2.78	0.34 (0.04, 0.65)	2.74	0.34 (0.04, 0.64)
IQ → GPA	–	–	–		–	–	–0.18	–0.18	–0.18	–0.23 (−.30, −.16)
GPA regressions										
GPA → Age at first sex	–	–	–		–	–	[0]	[0]	[0]	[0]
A_{GPA} → Age at first sex	–	–	–		–	–	–1.57	–0.22 (−.37, −.08)	–1.56	–0.22 (−.37, −.08)
C_{GPA} → Age at first sex	–	–	–		–	–	[0]	[0]	[0]	[0]

Note. Boldface indicates significant at $p < .05$. SES = socioeconomic status; GPA = grade point average.

Parameter estimates from the best fitting model (Model 1c) are summarized in the first column of Table 3. The estimated variances of the *ACE* components of intelligence indicate that 43% of the total variance in intelligence was due to additive genetic influences, 34% to shared environmental influences, and 23% to nonshared environmental influences. The shared environmental influences on intelligence significantly predicted later age at first sex ($b = 1.73$, $\beta = .34$). Finally, of the residual variance in age at first sex (i.e., variance independent of intelligence), 17.5% was due to additive genetic influences, 32.5% was due to shared environmental influences, and 50% was due to nonshared environmental influences plus error.

Race/ethnicity and socioeconomic status. One highly contentious topic within behavioral genetics—and within psychology and education in general—is racial and socioeconomic disparities in cognitive ability and academic achievement. The next models (Model 2 and Model 3) explicitly controlled for race/ethnicity and three indices of socioeconomic status (parental education, parental income, and receipt of public assistance) in order to test whether the relation between age at first sex and shared environmental influences on intelligence was due to racial/ethnic or socioeconomic differences in intelligence. Model 2 was identical to Model 1c, except that dummy-coded variables for race (African American = 1, White/Asian/Other = 0) and ethnicity (Hispanic/Latino = 1, non-Hispanic = 0) were included as statistical covariates for both intelligence and age at first sex. Parameter estimates from Model 2 are shown in the second column of Table 3. Including race and ethnicity decreased the estimate of shared environmental variance in intelligence (from 34% to 24%), but the (standardized) effect of the shared environmental factors on age at first sex was unchanged ($\beta = .35$), indicating that race and ethnicity do *not* account for the association between intelligence and age at first sex.

Model 3 was identical to Model 2, except that in addition to race and ethnicity, we added measures of parental socioeconomic status. Parental income (square root transformed), maternal education, and whether the family received public assistance (yes = 1) were included as statistical covariates for both intelligence and age at first sex. Parameter estimates from Model 3 are shown in the third column of Table 3. Including indicators of socioeconomic status as covariates further decreased the estimate of shared environmental variance in intelligence (to 17%), but the influence of these

unmeasured shared environmental factors on age at first sex was again unchanged ($\beta = .36$). This suggests that between-family differences in education and wealth do *not* account for the association between intelligence and age at first sex.

The extent to which race/ethnicity and socioeconomic status accounted for the shared environmental variance in intelligence versus the association between intelligence and age at first sex is illustrated in Figure 3. The black bar represents the proportion of variance in intelligence due to shared environmental factors estimated from a model that contained no statistical covariates (Model 1c), race/ethnicity only (Model 2), and both race/ethnicity and three measures of socioeconomic status (Model 3). The gray bar represents the standardized regression between shared environmental influences on intelligence and age at first sex, estimated for the same three models. Although race/ethnicity and socioeconomic status accounted for shared environmental variance in intelligence, these variables did not account for the relation between intelligence and age at first sex.

Academic achievement. Model 4 added the effects of academic achievement, while still controlling for race/ethnicity and socioeconomic status (see Figure 4). In Model 4, academic achievement was regressed directly on intelligence, and residual variance in academic achievement (i.e., variance independent of intelligence) was decomposed into additive genetic, shared environmental, and nonshared environmental components. Age at first sex was regressed on the *A* and *C* components of
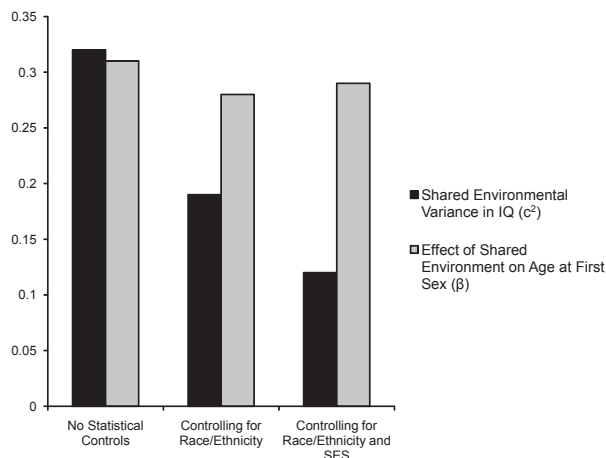

Figure 3. Impact of controlling for race and socioeconomic status (SES) on shared environmental variance in intelligence (c^2) and on effect of shared environmental variance in intelligence on age at first sex (β).

academic achievement ($b_{A\text{-GPA}}$ and $b_{C\text{-GPA}}$, respectively), which tests whether unmeasured genetic and environmental differences between families that are associated with adolescents' academic achievement influence adolescents' age at first sex. In addition, age at first sex was regressed directly on academic achievement (b_{GPA}), which tests whether, after controlling for unmeasured genetic and shared environmental confounds, academic achievement predicts later age at first sex. In other words, do twins who differ in their academic achievement also differ in their age at first sex? It is important to note that because intelligence (and the biometric components of intelligence) is also included, Model 4 tests the *unique* relation between academic achievement and age at first sex.

Model 4 was compared to three nested models (Model 4b, Model 4c, and Model 4d; see Table 2). Model 4b, which fixed the regressions of age at first sex on the *A* and *C* components of academic achievement to zero, fit significantly worse than the full model, indicating the unmeasured genetic and environmental differences between families significantly contributed to the relation between academic achievement and age at first sex. Model 4c, which fixed the regressions of age at first sex on the *A* component of academic achievement and on academic achievement itself to zero, also fit significantly worse than the full model, indicating that, unlike the pattern of results found for intelligence, shared environmental confounds did not entirely account for the relation between academic achievement and age at first sex. In contrast, Model 4d, which fixed the regression of age at first sex on the *C* component on academic achievement and on academic achievement itself to zero, did not fit significantly worse than the full model, indicating that the association between academic achievement and age at first sex could be entirely accounted for by genetic differences between families. That is, MZ twins who differed in their academic achievement but shared 100% of their genes, did not significantly differ in their age at first sex.

Parameter estimates from Model 4d are shown in the right side of Table 3. As expected, intelligence was significantly associated with academic achievement: Smarter students had better grades ($\beta = -.18$). (As a reminder, GPA was scored such that 1.0 corresponded to all A's and 4.0 corresponded to D's or lower.) The association between shared environmental factors associated with intelligence and age at first sex was unchanged by including academic achievement in the model ($\beta = .34$). Variance in academic achievement could
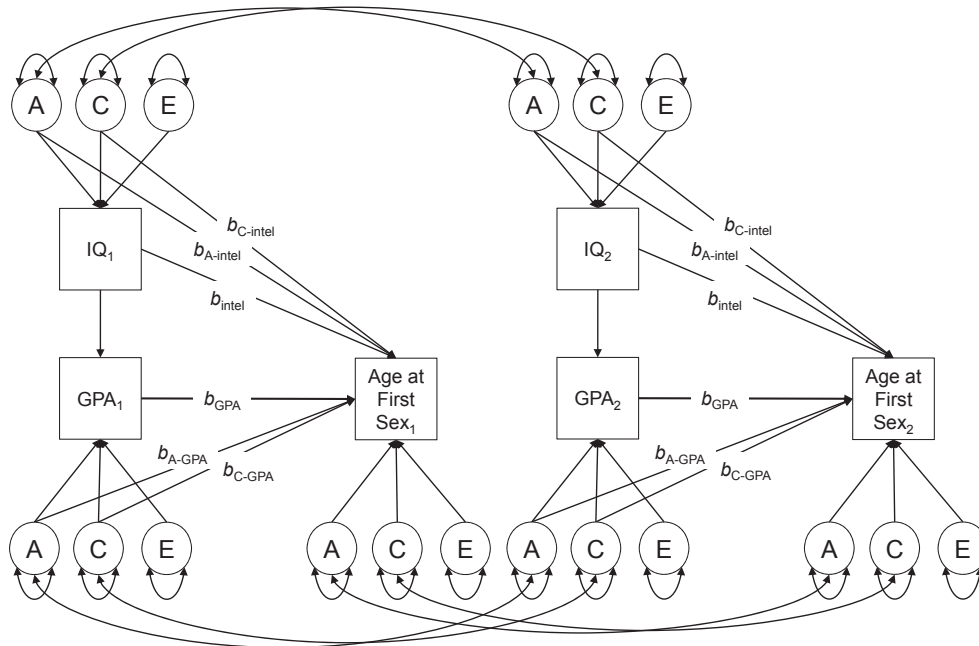
Figure 4. Multivariate twin model of intelligence, academic achievement, and age at first sex.

Note. Variances of *ACE* components freely estimated. Correlations between *A* components fixed to equal 1.0 in monozygotic (MZ) twins and 0.5 in dizygotic (DZ) twins. Correlations between *C* components fixed to 1.0 in all twins. Gender (male = 1), African American race, Hispanic/Latino ethnicity, square root of parental income, maternal education, and receipt of public assistance included as statistical covariates of IQ, grade point average (GPA), and age at first sex (not illustrated).

be attributed to additive genetic influences (35%), shared environmental influences (22%), and non-shared environmental influences (43%). Genetic factors associated with higher academic achievement, independent of intelligence, significantly predicted later age at first sex ($\beta = -.22$).

Pubertal development. In the final model, Model 5, we included reports of pubertal development relative to same-aged peers as a statistical covariate. Model 5 was identical to Model 4d, except that both GPA and age at first sex were regressed onto pubertal development, and twins' reports of their pubertal development were allowed to covary with each other by zygosity. Parameter estimates from Model 5 are shown in final column of Table 3. The estimate of the genetic association between achievement and age at first sex ($\beta = -.22$) was unchanged relative to Model 4d.

Discussion

This study uses genetically informed data from a large, nationally representative sample to test pathways by which intelligence and academic achievement are associated with later ages at first sexual intercourse. We found significant genetic and

shared environmental variance in intelligence ($h^2 = 0.43$, $c^2 = 0.34$), academic achievement ($h^2 = 0.36$, $c^2 = 0.21$), and age at first sex ($h^2 = 0.18$, $c^2 = 0.33$), indicating that these outcomes ''run in families,'' for both cultural and genetic reasons.

Consistent with the previous literature, both higher intelligence and higher academic achievement were associated with later age at first sex. In our between-family means comparisons, the effect sizes for intelligence and academic achievement were both approximately $d = 0.4$. However, results from twin models were inconsistent with the premise that higher intelligence and achievement are directly related to sexual activity. This conclusion is consistent with the emerging literature on adolescent development suggesting that risk-taking behavior is not simply the product of adolescents' systematically underestimating the consequences associated with certain forms of behavior, nor does increasing adolescents' knowledge of potential consequences reliably result in behavior change (Steinberg, 2004).

Subsequent analyses revealed that intelligence and academic achievement are indirectly related to age at first sex via different mechanisms. Intelligence was associated with later age at first sex entirely through unmeasured *environmental* differences between families. Families who, on average,

have higher intelligence also delay, on average, initiating sexual activity, but twins raised in the same family who differ in their intellectual capacities do not differ in their age at first sex. Thus, it is not intelligence, per se, that results in delayed sexual activity; rather, intelligence represents a proxy variable for socioenvironmental differences between families that are associated with both higher average levels of intelligence in family members and later average ages at first sex. In contrast to the pattern of results for intelligence, academic achievement was uniquely associated with later age at first sex via common *genetic* influences. That is, genetic factors that influence academic achievement, independent of intelligence, are also related to age at first sex. These factors entirely account for the association between achievement and age at first sex.

Unlike many previous investigations, we found non-negligible shared environmental influences on intelligence ($c^2 = 0.33$). This result is noteworthy, in and of itself. Shared environmental influences may have been more evident in the current study than in many previous behavioral genetic studies of intelligence, because the twin pairs in AddHealth represent a broader range of economic and racial/ethnic backgrounds than is typically represented in twin or adoption studies. Recent research on gene–environment interaction in child and adolescent intelligence by Turkheimer, Harden, and colleagues (Harden, Turkheimer, & Loehlin, 2007; Loehlin, Harden, & Turkheimer, 2009; Tucker-Drob, Harden, & Turkheimer, 2009; Turkheimer et al., 2003) indicate that shared environmental influences on intelligence are stronger among low-socioeconomic-status families. Including direct measures of socioeconomic status and race/ethnicity accounted for a substantial portion of the shared environmental variance in intelligence, reducing the estimate of c^2 from 34% to 17%, but did not account for any of the effect of shared environmental variance in intelligence on age at first sex.

The utility of a behavior genetics model is that it controls for all potential confounds that differ between families, both measured and unmeasured. The converse of this methodological advantage is environmental, and genetic variables remain a "black box": The twin design can identify whether relevant confounds are ultimately environmental versus genetic in origin, but it is not able to pinpoint specific variables that comprise the latent genetic and environmental factors. In the present study, we attempted to elucidate which particular shared environmental confounds account for the association between intelligence and age at first sex

by explicitly including variables of potential interest in a series of follow-up models. Although these variables slightly reduced the magnitude of the shared environmental path, they did not eliminate it. In other words, in the case of intelligence and age at first sex, relevant socioenvironmental and cultural differences between families cannot be fully accounted for by measured indicators of socioeconomic status or by race and ethnicity. This presents the obvious question of what additional environmental variables might be driving the relation between intelligence and age at first sex, about which we can only speculate.

One potential shared environmental variable may be parental involvement and parental monitoring. Socialization theories posit that parental involvement in children's intellectual enrichment and intellectual development positively impact intelligence (e.g., Scarr, 1997). In addition, for many parents, delaying their children's entrée into sexual maturity and parenthood is a highly salient parental goal (Geronimus, 2003), and involved parents may actively monitor their adolescents' activities and minimize opportunities for sexual experiences. Thus, adolescent intelligence and later age at first sex may come to be correlated because parents who are strongly involved with encouraging their children's intellectual development are the same parents who strongly gate their children's romantic activities.

Additionally, although the shared environment is commonly interpreted as the family environment, adolescent twins also share a breadth of extrafamilial environmental experiences that may be critical for understanding the relation between intelligence and age at first sex. One candidate is the adolescent's school environment. Fletcher (2007), using data from the National Education Longitudinal Study (NELS), presented evidence for social multiplier effects on rates of sexual initiation in U.S. high schools: The risk for an adolescent being sexually active changes with the how common sexual activity is among students in the high school. The effect of these school-wide norms could operate via increasing the acceptability of sexual behavior or via increasing the number of available sexual partners. If, for example, poor quality schools contained higher numbers of students who were heavily involved in delinquent behavior (including risky sex), then students enrolled in these schools would be expected to have lower cognitive abilities and higher rates of sexual activity.

Finally, the "shared environmental confound" may not be a single factor, but rather a composite of myriad, random, idiosyncratic, and unsystematic

differences between families that cannot be meaningfully decomposed into individual variables with reliable and independent effects. Plomin and Daniels (1987) first suggested this ''gloomy prospect'' with respect to nonshared environmental influences that make children in the same family different. More recently, Turkheimer (2009) has suggested that a similar problem underlies the difficulty in parsing omnibus genetic variance (which is typically high to very high) into individual measured genes (which typically have very, very small effect sizes). There is no compelling reason to anticipate that decomposing shared environmental influences will be unencumbered by the same methodological quandaries that plague research on the nonshared environmental or genetic influences. Ultimately, the ambiguity regarding *what*, exactly, is the shared environment underscores our earlier point regarding traditional correlational research: It is impossible to statistically control for every possible environmental and genetic confound. Thus, hypotheses that specify causal mechanisms between variables need to be rigorously tested, whenever possible, using experimental or quasi-experimental methodologies. Behavior genetic designs are one of several quasi-experimental options (Rutter et al., 2001).

In contrast to analyses of intelligence, our behavioral genetic analyses implicate *genetic* confounds in the association between academic achievement and age at first sexual intercourse. Notably, models of academic achievement were estimated controlling for the effects on intelligence, indicating that the genetic factors connecting academic achievement and age at first sex are independent of genes related to intelligence. There are several plausible mechanisms by which genes might influence this association. First, certain personality qualities—particularly high levels of conscientiousness—are not only heritable (e.g., Jang, Livesley, & Vernon, 1996) but also conducive for academic excellence (Chamorro-Premuzic & Furnham, 2003). Genes also influence types of psychopathology that would hamper academic performance, including high levels of test or school anxiety (Bolton et al., 2006), attention deficit hyperactivity disorder, and conduct or oppositional defiant disorder (Comings et al., 2000; Slutske et al., 1997). These genes might be mediated via dopaminergic neural circuits, which have been implicated in age at first sex (Miller et al., 1999), attentional disorders (Volkow et al., 2009), and animal models of cognitive abilities (Arnsten, Cai, Murphy, & Goldman-Rakic, 1994).

Alternatively, the genetic association between achievement and age at first sex may be due to genes influencing timing of pubertal maturation. When children mature physically is one of the strongest predictors of when they report sexual desire, first date, behaviors such as kissing and petting, and first sexual intercourse (Flannery, Rowe, & Gulley, 1993; Kim & Smith, 1998; Lam, Shi, Ho, Stewart, & Fan, 2002; Wyatt, Durvasula, Guthrie, LeFranc, & Forge, 1999). Using the first two waves of data from AddHealth, Rowe (2002) used a bivariate twin model to estimate the correlation between genetic influences on menarche and genetic influences on age at first intercourse, and found that the genetic correlation was 0.72 (see also Rodgers et al., 2008). In addition, individual differences in pubertal timing, particularly early pubertal timing, are associated with a breadth of academic outcomes, including lower achievement, truancy, and disciplinary problems (Dubas, Graber, & Petersen, 1991; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997). In the present study, we attempted to account for individual differences in pubertal timing by including adolescents' reports of their pubertal development relative to same-age peers as a statistical covariate. However, subjective reports of development are only modestly correlated with objective assessments of pubertal status (Dorn, Dahl, Woodward, & Biro, 2006). In addition, the AddHealth sample is age heterogeneous, with many participants having already completed puberty by the study's onset. Future research that incorporates objective measures of pubertal timing may help build a more comprehensive model of this complex set of developmental processes.

Limitations and Future Research

Our study has four additional limitations worth noting. First, as with many previous investigations of adolescent sexual behavior, we have focused on a single dimension—when the adolescent first had intercourse—but have not examined whether the etiology of sexual behavior is moderated by the context in which first sex occurs. Adolescents most commonly initiate sexual intercourse with a romantic partner, but adolescents may also initiate sex with nonromantic partners with whom there is no clear expectation of exclusivity or commitment (Manning, Longmore, & Giordano, 2005). Whether an adolescent is romantically involved with their first sexual partner has been shown to influence their perceptions of whether sex was ''too early'' (Cotton, Mills, Succop, Biro, & Rosenthal, 2004). In addition, adolescents vary in whether they use contraception with their first sexual partner; 74% of

girls and 82% of males report using contraception at first sex (Abma, Martinez, Mosher, & Dawson, 2004). Sexual decision making about who is an appropriate first sexual partner and whether to use contraception or condoms may be more directly influenced by intelligence and academic achievement than adolescents' decision making about when to first have sex. More generally, relationship context is a salient—and understudied—variable that may moderate the impact of adolescent sexual behavior for subsequent development. Future research that incorporates assessments of relationship context and contraceptive usage would provide a more nuanced understanding of the transition to sexual maturity.

Second, the current study focuses on first sexual intercourse, but intercourse is typically preceded by a number of partnered affectionate (e.g., hand-holding) and other sexual activities, which are also predicted by intelligence (Halpern et al., 2000). It remains unclear whether the many precursors of adolescent sexual activity operate specifically at the transition to intercourse per se, or whether they operate much earlier in the progression toward sexual involvement (e.g., delays in the shift to mixed-sex peer groups). How individual differences in sexual behavior relate to other, ostensibly more innocuous, developmental progressions is an important vein of inquiry for future research.

Third, there have been substantial secular changes in adolescent sexual norms, with increasing numbers of teenagers becoming sexually active (Kotchick, Shaffer, Forehand, & Miller, 2001). These changes may moderate the causes of individual differences in sexual behavior. For example, Dunne et al. (1997) found that genetic influences on age at first sex were higher in younger cohorts than older cohorts, which may be due to historical increases in sexual freedoms. The first studies documenting the relation between age at first sex, intelligence, and achievement were conducted over two decades ago (Cliquet & Balcaen, 1983; Miller & Sneesby, 1988; Mott, 1983), and the extent to which cognitive performance predicts sexual behavior, as well as the mechanisms underlying this association, may have changed with shifting social norms.

Fourth, there were a very few participants (N = 67 of 1,072 participants) who had still not yet had sex by the Wave IV assessment (age 24–32 years). These participants did not differ from nonvirgins with regard to intelligence, academic achievement, race/ethnicity, or socioeconomic status. Nevertheless, our estimates of the mean age at

sexual intercourse are slightly biased downward by excluding participants who were very late sexual initiators. In addition, while there is a persistent focus on early sexual behavior in the research literature, a late onset of sexual activity may be related to a broad array of constructs that might be relevant for the current analyses. These include qualities on the internalizing versus externalizing spectrum, such as inhibition, anxiety, or self-esteem, which could influence school performance and assessment behavior as well as sexual activity.

Conclusion

The current article examined intelligence and academic achievement as potential protective factors for early initiation of sexual activity. Our results demonstrate that intelligence and academic achievement are related age at first sex via different processes: Intelligence and age at first sex are associated via unmeasured environmental differences between families, whereas academic achievement and age at first sex are associated via common underlying genes. Further behavioral genetic research is necessary to identify the specific variables that account for these shared environmental and genetic processes, and to determine the role of genes in the numerous other individual, family, school, and neighborhood factors correlated with adolescent sexual activity.

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