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EMPIRICAL RESEARCH

Rethinking Timing of First Sex and Delinquency

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Abstract The relation between timing of first sex and later delinquency was examined using a genetically informed sample of 534 same-sex twin pairs from the National Longitudinal Study of Adolescent Health, who were assessed at three time points over a 7-year interval. Genetic and environmental differences between families were found to account for the association between earlier age at first sex and increases in delinquency. After controlling for these genetic and environmental confounds using a quasi-experimental design, earlier age at first sex predicted lower levels of delinquency in early adulthood. The current study is contrasted with previous research with non-genetically informative samples, including Armour and Haynie (2007, *Journal of Youth and Adolescence, 36*, 141–152). Results suggest a more nuanced perspective on the meaning and consequences of adolescent sexuality than is commonly put forth in the literature.

Keywords Adolescent sexual behavior · First sex · Sexual debut · Delinquency · Behavior genetics

Introduction

Armour and Haynie recently published a report on "Adolescent sexual debut and later delinquency" in the February 2007 issue of *Journal of Youth and Adolescence*. The authors' stated intent was to examine whether earlier "sexual debut," relative to peers, increases the risk for delinquent behavior. The authors successfully utilized a large and complex data set—the National Longitudinal Study of Adolescent Health—to demonstrate that adolescents who have experienced first sex in the past year exhibited higher levels of delinquency compared to adolescents who remained virgins. In addition, adolescents who experienced first sex earlier than their same-school peers exhibited an increase in delinquency later in adolescence and early adulthood, controlling for previous delinquency. Overall, this was an interesting analysis of the relations among variables of considerable developmental interest. We have no quibbles with either the content or the execution of the analysis itself. Nevertheless, Armour and Haynie's (2007) interpretation of their results typifies a problem commonly encountered in the literature on adolescent development—drawing unwarranted causal conclusions from non-experimental data.

Armour and Haynie are no doubt well aware of the logical pitfalls of concluding that *X* causes *Y* from non-experimental data. Yet, like many researchers, they interpret an epidemiological association between the timing of first sex and delinquency—with little or no qualification—as evidence that early first sex causes later delinquency. For example, they conclude that "the timing...of events such as sexual activity has profound consequences" (p. 149) and that "experiencing early or late sexual debut continues to have consequences for delinquent behavior occurring in young adulthood" (p. 150). An alternative explanation for their findings, of course, is that a third variable, either *environmental or genetic*, is associated with both timing of first sex and later delinquent behavior. That is, the association between early sex and later delinquency reported by Armour and Haynie (2007) could be an artifact of uncontrolled confounds.

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In keeping with standard practice of epidemiological research, Armour and Haynie (2007) included a number of covariates as statistical controls; among them were gender, race, receipt of public assistance, parental education, family structure, previous substance use and depression, importance of religion, school GPA, relative pubertal status, and virginity pledge status. Any list of statistical controls, however, is necessarily arbitrary and incomplete, because there is always the possibility that some unmeasured covariate remains to confound the analysis. For example, other studies using the AddHealth data have found that delay in timing of first sex for girls was predicted by parent-teen closeness and parental monitoring (Manlove et al. 2007); as well as by maternal disapproval of daughter's sexual activity, and frequency of maternal communication with the parents of her daughter's friends (McNeely and Shew 2002). Similar research has found that frequent parental religious attendance and daily family religious activity predict later first sex for both females and males (Manlove et al. 2006). Any of these additional variables, in turn, could also be related to delinquency in early adulthood, confounding the observed association with timing of first sex. Armour and Haynie (2007) cannot be faulted for failing to include these—and other—additional variables, because the limitations of statistical controls are shared with all of non-experimental developmental psychology. It is simply impossible to measure and statistically control for every conceivable environmental third variable.

In addition, regardless of how many environmental third variables are measured and included as statistical controls, traditional epidemiological research is incapable of controlling for *genetic* selection factors. Although psychologists and sociologists may be more used to thinking of adolescent sexual behavior in terms of its social antecedents—including family environments, peer affiliations, and sociodemographic factors—sexual behavior is also influenced by genes. Genetic influence on sexual behavior is directly predicted by evolutionary theory: Sexual behavior is the most proximal determinant of reproductive fitness, the evolutionary mechanism by which gene frequencies are modified or maintained. Supporting this prediction, a number of twin studies have demonstrated that siblings who are more genetically similar exhibit more similar ages at first sex, indicating that genetic differences between individuals account for population variation in sexual timing (Bailey et al. 2000; Dunne et al. 1997; Martin et al. 1977; Lyons et al. 2004; Rodgers et al. 1999). Also, functional polymorphisms for dopamine receptor genes are associated with earlier age at first sex, particularly in males (Miller et al. 1999). Additionally, twin studies have indicated that genetic factors influence other fertility-relevant phenotypes associated with age at first sex (Udry and Cliquet 1982), including age of menarche (Kaprio et al. 1995; Rowe 2002), and age at first birth (Kohler et al. 2002; Niess et al. 2002; Waldron 2004). Overall, these results are consistent with a larger body of research demonstrating that measurements of ostensibly social experiences partly reflect genetic differences between individuals (Plomin and Bergeman 1991). In contemporary society, individuals have great latitude to select their social "niches"—including friends, occupations, and sexual partners—and, over time, these social niches become reflective of their genetically-influenced behavior and personality (Scarr and McCartney 1983).

Delinquency and conduct disorder are also influenced by genetic factors, as evidenced by twin studies, children-of-twin studies, adoption studies, and molecular genetic studies (e.g., Arsenault et al. 2003; Cadoret et al. 1983; D'Onofrio et al. 2007; Scourfield et al. 2004; Slutske et al. 1997; Young et al. 2002; for reviews see Miles and Carey 1997; Raine 2002; Rhee and Waldman 2002; Rowe 2001). To our knowledge, there are no extant genetically-informative studies that can speak directly to whether the genes related to age at first sex overlap with the genes related to externalizing psychopathology. Nevertheless, previous research on adolescent motherhood—a correlate of early age at first sex—suggests that the genes influencing delinquency may overlap with the genes influencing reproductive timing. Certainly, adolescent mothers frequently report histories of conduct problems (Bardone et al. 1996; Emery et al. 1999; Woodward and Fergusson 1999). Moreover, a twin study by Waldron (2004) indicated that adolescent motherhood is related to *future* externalizing problems in women via common genetic risks. Research with Children-of-Twins has shown that adolescent motherhood is related to *offspring* behavior problems, in part via genetic transmission (Harden et al. in press). Similarly, elevated levels of delinquency evident in young adults who had sex at a relatively young age may be due to common genetic influences, perhaps via genes expressed in dopaminergic neural systems, which have been associated with both antisocial behavior (Young et al. 2002) and earlier age at first sex (Miller et al. 1999).

Behavior Genetic Designs as Quasi-Experimentation

Given that timing of first sex is related to a host of genetic and environmental selection factors, it is necessary to use a quasi-experimental research design capable of "pulling apart" age at first sex from all typically confounded variables. Behavior genetic designs can provide such quasi-experimental control. The most obvious use of a behavior genetic design is to control for genetic influences: In a comparison of monozygotic twins who differ in their age at first sex, any differences in delinquency between them

cannot be attributed to genetic confounds. In addition, when comparing twins who differ in their age at first sex, differences in adjustment between them cannot be attributed to any aspect of the familial environment they share, such as sociodemography, family structure, and family relationships (Dick et al. 2000). This last point should be underscored, because the methodological advantage is often overlooked. Twin studies not only control for genetic selection, but they also control for shared environmental selection, including both measured and unmeasured experiences. Comparing twins, therefore, provides a rigorous test of whether the relation between timing of first sex and delinquency is causal. If Identical Twin A has sex earlier than Identical Twin B, and Twin A also shows high levels of delinquency than Twin B, this association cannot be due to *any* genetic or environmental third variables shared by the twins, not just those which can be measured and included as statistical covariates. Of course, no iron-clad causal conclusions can be wrought from purely correlational data: the within-twin pair comparison is confounded by variables that differ within twins and are related to both variables of interest. Because these uncontrolled confounds remain, we use the term "quasi-causal" to refer to associations between risk factors and outcomes that remain even after controlling for genetic and shared environmental confounds using a quasi-experimental design.

Hypotheses

In the current paper, we analyze the association between timing of first sex and later delinquent behavior, using a subset of twin pairs from the same data set as Armour and Haynie (2007). This analysis was designed to test two hypotheses: (a) Both genetic and shared environmental factors will influence adolescents' age at first sex; (b) In a comparison of twins, which controls for these genetic and shared environmental factors, the twin who experiences first sex earlier than his or her co-twin will not demonstrate higher levels of delinquency later in adolescence and early adulthood.

Method

Participants

Data were drawn from the National Longitudinal Study of Adolescent Health (Add Health; Udry 2003a), a nationally representative study designed to assess adolescent health and risk behavior collected in three waves between 1994 and 2002. Sampling for Add Health 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 3,000 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 = 17 years). Approximately 1 year later, students repeated the in-home interview (Wave II interview). Ages ranged at this time from 11 to 23 years (M = 16 years, 25th percentile = 15 years, 75th = 17 years). Overall, N = 14,700 adolescents (7,144 male, 7,556 female) participated in interviews for both Wave I and II. A third wave of data collection, targeting factors involved in the transition from adolescence to young adulthood, was collected through an interview administered between August 2001 and April 2002 (N = 15,170; 8,030 female, 7,167 male). Participants were between 18 and 28 years of age at Wave III (M = 22 years, 25th = 21 years, 75th percentile = 23 years).

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 siblings pairs, even if one member of the pair did not attend a high school in the original probability sample. (However, 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, the focus of the current analyses. Twins were classified as either monozygotic (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 (Loehlin and Nichols 1970; Spitz et al. 1996). There were 144 male–male MZ, 145 female–female MZ, 131 male–male DZ, and 114 female–female DZ. 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. Jacobson and Rowe (1999) compared the sociodemographic composition of sibling pairs to the full AddHealth sample and found negligible differences with regard to age, ethnicity, or maternal education.

Measures

The Add Health 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 2003b), but no intact scales from the literature were included in the survey. Questionnaire items were extensively pilot tested before use.

Age at First Sex

At each assessment wave, participants reported whether they had ever had vaginal intercourse and their age at first intercourse. To minimize telescoping in the retrospective report of age at first sex, we used the age reported by the participant in the earliest wave that he or she endorsed having had sex. Age at first sex ranged from 5.08 to 24.00 years (M = 16.44, 25–75% = 15–18; SD = 2.48). The modal response was 18.00 years. Of the 534 twin pairs, 346 pairs had reports for age at first sex for both twins, 122 had reports for age at first sex for at least one twin, and 66 pairs had reports for neither twin. 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' delinquency scores. 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 distribution of non-missing age at first sex data was approximately normal, although negatively skewed (due to a few participants who reported first sex at extremely young ages) and positively kurtotic. The correlation between age at first sex in the first and second twin of each pair was 0.30 in DZ pairs and 0.42 in MZ pair.

Delinquency

At Wave I, adolescents were asked how often *in the last 12 months* they had engaged in each of 15 delinquent behaviors: *Never* (0), *One or two times* (1), *Three or four times* (2), or *Five or more times* (3). At Wave III, the frequency of delinquent acts was assessed using a 19-item scale. It is important to note that self-report questionnaires may be more sensitive and valid instruments for assessing delinquency than measures based on official law enforcement records, because arrest rates tend to be confounded by family income and race.

Because our goal was to compare our results to those of Armour and Haynie (2007), we attempted to replicate their variables as precisely as possible. Of the 15 items relevant to delinquency at Wave I, they selected six items: paint graffiti, deliberately damage someone else's property, taking something from a store without paying for it, steal something worth more than $50, steal something less than $50, and sell marijuana or other drugs. Like the authors, we summed these six items at Wave I, and then we rank-transformed the sums to yield *Wave I Delinquency* scores (M = 0, SD = 0.85, range = –0.63 to 3.04). At Wave III, participants were not asked about painting graffiti, or taking something from a store without paying for it. The remaining four items were summed with scores on the item "Have you ever entered a house or building to steal something?," (D. Haynie, personal communication). Again, the sums were rank-transformed to yield *Wave III Delinquency* scores (M = 0, SD = 0.71, range = –0.32 to 2.98).

Analyses

We fit a series of structural equation models (SEM) in the software program Mplus (Muthen and Muthen 1998–2007). Models were compared using two measures of goodness-of-fit, BIC and RMSEA, as well as differences in χ^2. The BIC estimates the ratio of posterior to prior odds in comparisons of an estimated model with a saturated one (Raftery 1993; Schwarz 1978). BIC outperforms other model fit criteria in its ability to discriminate between multivariate behavior genetic models, particularly for complex model comparisons in large samples, and is more robust to distributional misspecifications (Markon and Krueger 2004). RMSEA measures error in approximating data from the model per model parameter (Steiger 1990). RMSEA values of less than 0.05 indicate a close fit, and

values up to 0.08 represent reasonable errors of approximation. Browne and Cudeck (1993) have noted that the RMSEA provides very useful information about the degree to which a given model approximates population values. Interpretation of BIC and RMSEA values are comparative, with lower values for both indicating better model fit. Differences in model χ^2 are themselves distributed as χ^2, with *df* equal to the difference between the models' *df*.

Results

Armour and Haynie (2007)'s final analysis (summarized in their Table 4, p. 150) was a regression of Wave III delinquency on timing of first sex, controlling for Wave I delinquency and a variety of other statistical covariates. Similarly, the models presented in the current paper analyze the relation between age at first sex and Wave III delinquency, controlling for Wave I delinquency. The full model is shown in Fig. 1, with the first twin in each pair on the top and the second twin in each pair on the bottom. The left hand portion of this model, concerning age at first sex, is identical to a classical twin model (Neale and Cardon 1992). Variance in age at first sex is divided into three components: additive genetic (*A*), shared environmental (*C*), and non-shared environmental (*E*). The *shared environment* comprises all environmental influences that make children from the same family similar to each other. For example, if twins shared a particular religious affiliation caused them both to delay sexual activity, this effect of religion would be subsumed by the *C* component. The *non-shared environment* comprises all environmental influences that make children in the same family different, as well as measurement error. For example, if one twin was affiliated with a deviant peer group that accelerated the timing of first sex, while the other twin was affiliated with a conforming peer group that delayed the timing for first sex, this differential peer effect would be subsumed in the *E* component. The correlation between additive genetic components is 1.0 in MZ twins, who are genetically identical, whereas this correlation is 0.5 in DZ twins, who share 50% of their genes. Regardless of zygosity, shared environmental components are correlated 1.0 across twins, whereas non-shared environmental components are uncorrelated across twins. Thus another way to conceptualize the *E* component, which will be important for understanding the results of the current paper, is as a measure of how much MZ twins differ in a given phenotype.

The most familiar parameterization of the classical twin model standardizes the variance of the *A*, *C*, and *E* components and estimates the paths from these components to the phenotype. The parameterization used in the current project fixes the paths to one and estimates the variances of the *A*, *C*, and *E* components. In this way, the scale of each component is determined by the scale of the phenotypic variable. The sum of the component variances equals the total phenotypic variance; the proportion of additive genetic variance to total phenotypic variance equals the heritability coefficient.

The decomposition of variance in age at first sex is not, in and of itself, the primary analysis of interest. The key feature of this model is that Wave III delinquency is regressed on the *ACE* components of age at first sex, controlling for Wave I delinquency. The regressions of Wave III delinquency onto *A* (b_{a3}) and *C* (b_{c3}) reflect the

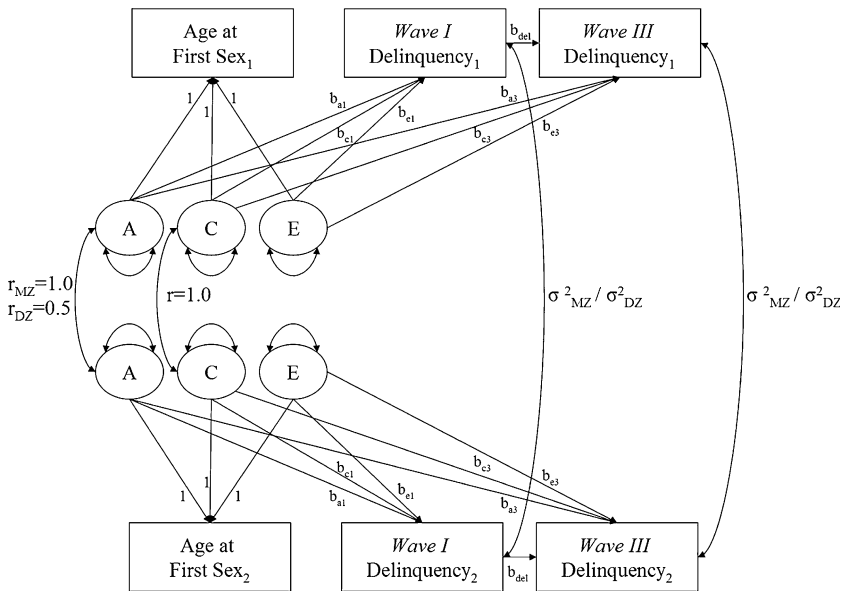

Fig. 1 Multivariate ACE model of age at first sex and delinquency at Waves I and III

extent to which genetic and shared environmental influences, respectively, on age at first sex predict later delinquent behavior. The regression on *E* (b_{e3}) reflects whether differences between twins in their ages at first sex predict differences in their later delinquency—the quasi-causal effect. That is, does the adolescent who has sex earlier than his or her twin demonstrate an increase in delinquency behavior at Wave III, as would be predicted by the causal hypothesis? Similarly, Wave I delinquency is regressed on the *ACE* components of age at first sex. These regressions also divide the association between delinquency and age at first sex into that attributable to common familial influences—either environmental or genetic—and that operating within twin pairs. In most cases, however, the assessment of delinquency at Wave I *preceded*, or was roughly contemporaneous to, initiation of sexual activity. The association between Wave I delinquency and age at first sex, then, is not of interest in evaluating whether timing of sexual intercourse causes an increase delinquency. It may be, however, indicative of whether early sexual intercourse and delinquent activity are manifestations of a single, underlying externalizing syndrome. Finally, the residual variance in Wave I and Wave III delinquency covaries across twins, and the magnitude of these covariances were estimated separately for MZ and DZ pairs.

Results from the full model are summarized in the first column of Table 1. Nearly 31% of the variation in age at first sex was accounted for by additive genetic factors [1.89/(1.89 + 0.64 + 3.58) = 0.309]; about 10% by shared environmental factors, and the remaining 59% by non-shared environmental factors. (Despite the relatively large standard errors for estimates of *A* and *C* variance, these variance components could not be fixed to zero without a

Table 1 Results from multivariate twin models

	Model 1 Full	Model 2 *E* Regr Only	Model 3 *A* Regr = *C* Regr	Model 4 No *E* Regression
Variance components				
A	1.89 (.94)	1.31 (1.10)	1.07 (.83)	1.02 (.93)
C	.64 (.81)	1.22 (.95)	1.32 (.73)	1.40 (.80)
E	**3.58 (.33)**	**3.60 (.34)**	**3.72 (.34)**	**3.71 (.34)**
Regression coefficients				
b_{del}	.07 (.04)	**.14 (.03)**	**.10 (.03)**	**.13 (.03)**
b_{a1}	–.07 (.11)	–.11 (.19)	**–.19 (.04)**	–.18 (.04)
b_{c1}	–.45 (.45)	–.23 (.22)	**–.19 (.04)**	–.18 (.04)
b_{e1}	–.01 (.02)	–.003 (.02)	.002 (.02)	.002 (.02)
b_{a3}	–.01 (.09)	[0]	**–.07 (.03)**	–.01 (.03)
b_{c3}	–.21 (.17)	[0]	**–.07 (.03)**	–.01 (.03)
b_{e3}	**.05 (.02)**	**.04 (.02)**	**.06 (.02)**	[0]
Residual covariances				
$\sigma^2(Del_{11}–Del_{21})_{MZ}$	.19 (.14)	**.24 (.07)**	**.24 (.05)**	**.25 (.05)**
$\sigma^2(Del_{11}–Del_{21})_{DZ}$	.12 (.15)	**.19 (.08)**	**.20 (.05)**	**.20 (.05)**
$\sigma^2(Del_{13}–Del_{23})_{MZ}$	**.14 (.05)**	**.17 (.03)**	**.15 (.03)**	**.16 (.03)**
$\sigma^2(Del_{13}–Del_{23})_{DZ}$	–.01 (.05)	.02 (.04)	.007 (.04)	.01 (.04)
Effect sizes				
$R^2–Del_{11}/Del_{21}$	.20/.18	.11/.10	.12/.10	.11/.10
$R^2–Del_{13}/Del_{23}$	.10/.12	.04/.05	.07/.09	.03/.04
Model fit				
BIC	8136.253	8136.716	8131.870	8139.852
RMSEA	0.000	0.017	0.000	0.033
χ^2, *df* (*p*)	19.26, 22 (.63)	25.94, 24 (.36)	21.08, 24 (.63)	32.13, 25 (.154)
$\Delta\chi^2$, Δdf (*p*)	–	6.68, 2 (.04)[a]	1.82, 2 (.40)[a]	11.05, 1 (<.01)[b]

Note: Standard errors are in parentheses except where otherwise noted. Parameter estimates significantly different from zero are in bold face type

[a] Compared to Model 1

[b] Compared to Model 3

significant decline in model fit. Results are available from first author upon request.) Delinquency at Wave I positively predicted delinquency at Wave III (b_{del} = 0.07); however, this effect was not significantly different from zero [95% confidence interval (CI) = –0.015, 0.155]. All three ACE components of age at first sex were negatively related to Wave I delinquency, which was assessed prior to or contemporaneous with initiation of sexual activity. Again, however, none of these effects were significantly different from zero. For Wave III delinquency, genetic and shared environmental influences on age at first sex predicted lower future delinquency. That is, adolescents from the 'type' of families who are predisposed towards earlier sexual activity trended towards higher levels of delinquency in early adulthood. These effects were also not significantly different from zero. In contrast, the within-twin pair effect of age at first sex on Wave III delinquency was *positive* and significantly different from zero (b_{e3} = 0.052; 95% CI = 0.016, 0.089). That is, in a comparison of two co-twins, the twin who delayed initiating sexual activity relative to his or her co-twin actually showed *higher* levels of delinquency in early adulthood. Despite the imprecision with which most of the regression coefficients were estimated, the ACE components of age at first sex accounted for 18–20% of the variance in Wave I delinquency and 10–12% of the variance in Wave III delinquency. (R^2 estimates computed automatically by Mplus software.) Moreover, a *phenotypic* model (not shown), in which delinquency at Waves I and III were regressed directly onto age at first sex rather than on the ACE variance components, fit significantly worse than the full model (χ^2 = 43.49, $\Delta\chi^2$ = 24.23, Δdf = 4, $p < .001$).

The large standard errors around the regressions from Wave III delinquency to the genetic and shared environmental influences on age at first sex would seem to suggest that these regressions are unimportant. Model 2 tested this hypothesis by fixing these regression coefficients to zero. If familial influences on sexual initiation are unrelated to later delinquency, fixing the associated regression coefficients should have no significant effect on model fit. Results from Model 2 are summarized in the second column of Table 1. As shown in the bottom line, the change in model fit was significant ($\Delta\chi^2$ = 6.68, Δdf = 2, p = 0.04). Moreover, there was a slight increase in both RMSEA and BIC, and the estimated proportion variance accounted for (R^2) was decreased by nearly half. Therefore, familial influences related to age at first sex were important in accounting for the association between age at first sex and later delinquency.

One possible explanation for this apparent paradox—the estimated regressions onto *A* and *C* were not significantly different from zero, yet fixing them to zero resulted in a significant detriment to model fit—is that we lacked adequate sample size to discriminate between shared environmental and genetic confounds. To test this hypothesis, we fixed the regressions of Wave I delinquency on the *A* and *C* components of age at first sex to be equal, and we fixed the regressions of Wave III delinquency on *A* and *C* to be equal. Results from Model 3 are shown in the third column of Table 1. As shown on the bottom line, equating the effects of shared environmental and genetic influences related to age at first sex on contemporaneous and later delinquency produced no significant change in model fit ($\Delta\chi^2$ = 1.82, Δdf = 2, p = 0.40). The RMSEA was unchanged compared to Model 1, and the BIC was several points lower. The proportion of variance in Wave III delinquency accounted for decreased only slightly (7–9%). Finally, the regression coefficients were estimated with considerably more precision. Higher levels of Wave I delinquency significantly predicted higher levels of Wave III delinquency (b_{del} = –0.10; 95% CI = 0.04, 0.17). Genetic and shared environmental influences related to later age at first sex significantly predicted both Wave I delinquency (b_{a1} = b_{c1} = –0.19; 95% CI = –0.26, –0.11) and Wave III delinquency (b_{a3} = b_{c3} = –0.07; 95% CI = –0.13, –0.003). The within-pair effect of age at first sex on Wave I delinquency was positive in direction, but it was small in magnitude and not significantly different from zero (be1 = 0.002; 95% CI –0.04, 0.04). In contrast, the within-pair effect of age at first sex on Wave III delinquency was both positive in direction and significantly different from zero (b_{e3} = 0.06; 95% CI = 0.02, 0.09). Model 3, then, suggested a quasi-causal *protective* effect of sexual activity for delinquent behavior.

Our final model tested the importance of the regression of Wave III delinquency onto the *E* component of age at first sex. As mentioned previously, this regression is the key test of causal hypotheses concerning the association between early sexual activity and delinquent behavior. Model 4, which fixed this regression to zero, fit significantly worse than Model 3 ($\Delta\chi^2$ = 11.05, Δdf = 1, $p < 0.001$). Results are summarized in the fourth column of Table 1. BIC and RMSEA both increased from Model 3 to Model 4, and R^2 for Wave III delinquency decreased by about half. Age at first sex, then, was important for the prediction of later delinquency levels, even after controlling for related genetic and shared environmental differences between families. Surprisingly, however, this effect was in the opposite direction of that found in epidemiological research. In a comparison of two twins, in which Twin A had sex earlier than Twin B, Twin A actually demonstrated *lower* levels of delinquency in early adulthood than Twin B—a quasi-causal association that controlled for all the genetic and environmental confounds shared by twins. Overall, Model 3 was the best, most parsimonious fit to the data.

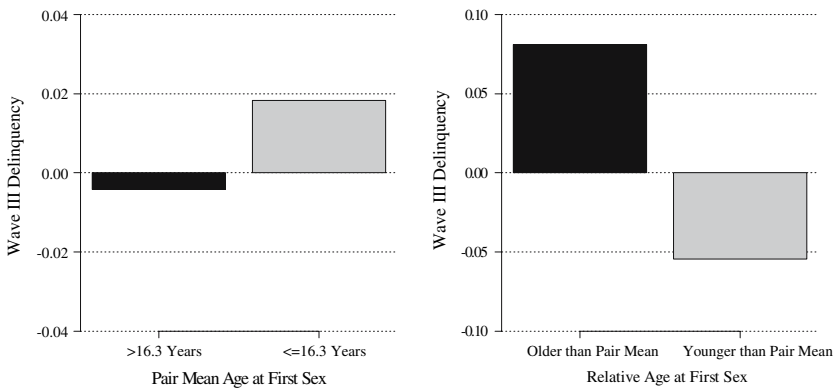

Fig. 2 Mean Wave III delinquency by twin pair mean for age at first sex and relative age at first sex (whether participant's reported age was younger or older than twin pair mean)

These results are illustrated in Fig. 2. On the left hand side, participants have been divided based on the *mean* age of first sex for the twin pair (less than 16.4 years versus greater than 16.4 years). Consistent with previous research, participants from twin pairs with earlier average sexual initiation (grey bar) have higher average delinquency at Wave III than participants with from pairs with later average sexual initiation (black bar).[1] On the right hand side, however, participants have been divided based on whether their own age at first sex was earlier or later than the mean age for the twin pair. In this comparison, the opposite pattern is evident: twins whose ages at first sex are earlier than their co-twins (grey bar) have lower levels of delinquency than their delaying siblings (black bar).

Discussion

Among same-sex twin pairs in the AddHealth sample, 31% of the variance in age at first sexual intercourse was due to additive genetic influences and 10% to shared environmental influences. The remaining 59% of variance in age at first sex, therefore, was due to non-shared environmental influences, indicating that even identical twins differ considerably in their timing of first sex. Those twin pairs who, on average, have earlier ages at first sex also demonstrated, on average, higher levels of delinquency in early adulthood. Once we controlled for between-family differences, however, the twin who had an earlier age of first sexual intercourse actually demonstrated *lower* levels of delinquency.

Our results have several important implications regarding the association between age of first sexual intercourse and delinquent behavior. It may appear contradictory that the between-twin pair relation and the within-twin pair relation were in different directions, but this pattern of results emphasizes a more general methodological point: Ecological correlations (i.e., correlations among group means) are silent about individual-level mechanisms. The textbook (e.g. Skrondal and Rabe-Hesketh 2004) example concerning ecological versus individual correlations is the relation between meat consumption and life expectancy. Between nations, this correlation is positive, because meat consumption indexes degree of industrial development. Within nations, however, the individual correlation is negative, because meat consumption increases the risk for obesity-related illnesses. The ecological correlation between meat consumption and life expectancy is uninformative about the mechanisms by which meat consumption affects individual health. Just as the between-nation "effect" of meat consumption comprises the effects of all uncontrolled factors correlated with meat consumption between nations (e.g., industrialization), the between-twin pair "effect" for average age of first sex comprises the effect of early sexual timing plus *the effects of all uncontrolled factors correlated with early sexual timing between families.* The between-family correlates of age at first sex, obviously, make for an impressively long list of both genetic and environmental factors. In contrast, the within-twin pair "effect," estimated in our analyses as the regression on the non-shared environmental component of age at first sex, comprises the effect of early sexual timing plus the effects of only those influences that systematically vary within twin pairs—a considerably shorter list of potential confounds. Epidemiological research, which often samples only one person per nuclear family, is incapable of distinguishing between the individual correlation and the ecological correlation (when the "ecology" in question is that of the family). In the current study, the ecological correlation between age at first sex and delinquency is negative, such that earlier *average* age at first sex predicts higher *average* delinquency. The individual correlation, however, is positive. Overall, the pattern of our results clearly demonstrate how the ecological fallacy may

[1] Because of clustering within twin pairs, basic inferential statistics that assume independence of observations would be incorrect. Therefore, standard errors and/or confidence intervals around means are not displayed. These means comparisons are intended to be purely descriptive.

obscure understanding of relations among variables. We conclude that the apparent relation between early age of first sex and later delinquency is likely to be a spurious consequence of uncontrolled genetic and environmental differences between families.

The results of the current study may be surprising, because the assumption that sexual activity, *in and of itself*, is somehow pathological and damaging for adolescents' psychological well-being is embedded in much psychological and epidemiological research on this topic. This assumption is evident in authors' stated rationales for investigating timing of first sex. For example, Armour and Haynie (2007, p. 141) note "the concern that sex outside of marriage is likely to have deleterious consequences for youth." Similarly, Capaldi et al. (1996) state that "experiencing [first intercourse] much earlier or later than the cohort may have psychosocial consequences for the individual" (p. 344) and go on to characterize adolescent sexual activity as, among other things, a "weapon with which to provoke or humiliate parents" (Capaldi et al. 1996, p. 345). Whitbeck et al. (1999) urged preventative measures that "counter...the adoption of sexually permissive attitudes" (p. 944). This assumption is also evident in researchers' operationalization of "risky sex," which frequently conflates sexual activity that is not conventional, but not necessarily physically hazardous (e.g., having more than two sexual partners in the past year), with sexual activity that *is* physically hazardous (e.g., not using condoms or any form of protection against disease). In sum, early sex has become nearly synonymous with risky sex, and the risk implied is to the adolescent's psychological as well as physical well-being.

Although the current results are contrary to embedded assumptions, they are actually consistent with previous research. Specifically, three quasi-experimental (longitudinal or behavior genetic) studies that examined whether timing of first sex influences *subsequent* psychosocial functioning, controlling for psychological differences that precede sexual initiation, have all failed to find adverse effects for sexual timing. First, Billy et al. (1988) assessed a sample of urban seventh-, eighth-, and ninth-graders at two time-points separated by a 2-year interval. Controlling for characteristics at Time 1, they found that experiencing first sex was associated with decreases in academic achievement at Time 2 (only in Whites), but was not associated with self-esteem, locus of control, religiosity, or deviance proneness (defined as cheating, drinking, or smoking) at Time 2. Overall, Billy et al. (1988) concluded that "adolescent premarital coitus does not precipitate overwhelming changes in an adolescent's psychological framework." Second, Bingham and Crockett (1996) examined differences in 12th-grade psychosocial adjustment among early, middle, and late initiators, controlling for adjustment prior to first sex (assessed during middle school). They found that the timing of first sex was associated with no psychosocial outcomes, including minor deviance, drunkenness, drug use, church attendance, academic plans, grades in school, family relationships, or peer relationships, after controlling for previous adjustment. Therefore, they concluded that early timing of first sexual intercourse does not cause a divergent pattern of psychosocial development; rather, the pattern evident in early initiators is a continuation of a preexisting developmental trajectory. Third, Buchting (1998), using a sample of over 8,000 male twins from the Vietnam Era Twin Registry, demonstrated that the association between age of first sex and subsequent alcohol use resulted from genetic factors that were common to both, rather than an effect of age at first sex per se.

We find it striking, given the volume of research on the antecedent and concurrent correlates of age at first sex, that there are so few longitudinal or behavior genetic that examine the putative psychosocial consequences of early age at first sex while attempting to control for preexisting differences in functioning or liability. Moreover, the current study is the first to combine the longitudinal and behavior genetic paradigms to investigate this topic. Obviously, additional research is needed. The limitations of the extant literature, and of the current paper, suggest three directions for future research:

1. Test other longitudinal associations using quasi-experimental designs. Additional research has documented associations between age at first sex and subsequent psychosocial adjustment, but without methodologically controlling for preexisting differences in functioning. For example, early age at first sex has been linked to earlier leaving of the parental home, lower educational attainment, experiencing a pregnancy termination (either via abortion or miscarriage), and receiving medical treatment to facilitate pregnancy (Magnusson and Trost 2006); greater psychological distress among college women (Leitenberg and Saltzmann 2003); and experimentation with cocaine (Kandel and Davies 1990) and other substances (Mott and Haurin 1988; Dorius et al. 1991). Also, Cauffman and Steinberg (1996) found that physical involvement with a boyfriend among seventh- and eighth-grade girls predicted disordered eating. The mechanisms underlying these associations need to be better understood and should be tested using quasi-experimental designs capable of resolving selection versus causation. As underscored by a limitation of the current study, namely, the inability to resolve shared environmental from genetic confounds, future studies may have to employ relatively large sample sizes to ensure adequate statistical power.

2. Discriminate among the various ecological contexts for adolescent sexual activity. One possibility is that

adolescents who have earlier timing of first sex exhibit decreased levels of delinquency in early adulthood, once genetic and environmental differences between families are controlled for, because some adolescents are experiencing sex within steady monogamous relationships. Maintaining steady dating relationships in middle adolescence predicts declines in depressive symptoms, alcohol use, and delinquency (Davies and Windle 2000). On the other hand, adolescents who transition into or maintain multiple casual relationships showed patterns of escalating problem behaviors, particularly for alcohol use (Davies and Windle 2000). This suggests that the relational context surrounding sexual activity—for example, the adolescent's emotional attachment and commitment to his or her partner, the reciprocity of that attachment and commitment, the duration of their acquaintance, and his or her sense of sexual intercourse being a desirable and voluntary outcome—has important implications for its developmental consequences. Similarly, sociocultural context may moderate the developmental consequences of early sexual activity. Billy et al. (1988) found that experiencing first sex predicted declines in academic achievement only for Whites, with no associations evident for Blacks. The current study is limited in that it does not incorporate information about ecological context, including the nature of the relationship with the first sexual partner, the adolescent's sociocultural context, or even whether the first sexual experience was voluntary versus the result of abuse or assault. Future research would benefit from incorporating contextual measurements to ascertain the circumstances that moderate how sexual experiences influence subsequent functioning.

3. Consider positive functions for adolescent sexual activity. The current study suggests that there may be positive functions for early initiation of sexual activity, in that the co-twin with earlier age at first sex demonstrated *lower* levels of delinquency in early adulthood. This result echoes a small but important body of previous research. In one of the first pieces of sex research, Kinsey et al. (1953) concluded that premarital sexual activity resulted in minimal "psychological disturbance" and may result in healthier non-romantic relationships and greater happiness later in life. More recent research has indicated that early sexual timing is associated with popularity (Prinstein et al. 2003); high self-esteem (for a review see Goodson et al. 2006; Paul et al. 2000); positive self-concept (Pedersen et al. 2003); high levels of body pride (Lammers et al. 2000), and increasing closeness to the same-sex best friend (Billy et al. 1988). To the extent that sexual activity occurs in the context of a dating relationship, Davies and Windle (2000) found that steady dating in middle adolescence predicted greater "emotional adjustment," as measured by declines in depressive symptoms, alcohol use, and delinquency.

In the domain of adult sexual functioning, earlier age at first sex was found to predict greater coital orgasmic capacity in adult women (Raboch and Bartak 1983) and to discriminate sexually functional versus non-functional older men (age 64 years; Vallery-Masson et al. 1981). Women reporting an earlier age at first sex demonstrate less reactivity and faster recovery (as measured by cortical response) in response to stress (Brody 2002). Also, earlier age at first sex is associated with increased intercourse frequency in adulthood for both genders (Brody 2002). Of course, drawing causal conclusions from any of these studies is difficult, because of the central issue we raise here: They fail to control for genetic and shared environmental selection. Nevertheless, the findings at least suggest the possibility that gaining sexual experience in adolescence may have positive implications for stress reactivity, adult sexual functioning, quality of peer relationships, and decreased externalizing. Future research should examine the mechanisms underlying the associations with positive outcomes, including the extent to which associations are due to genetic or environmental selection, as well as the ecological circumstances (e.g., within the context of intimate attachments) in which they are likely to occur. Moreover, there may be nonlinear or discontinuous effects of age at first sex, such that only sexual activity beyond a certain age threshold (e.g., reproductive maturity) is associated with positive correlates.

Conclusion

Twins differ considerably in their age at first intercourse, indicating that non-shared environmental factors are the strongest influence on sexual timing. Nevertheless, the twins who experienced first sex earlier than their co-twins did not demonstrate higher levels of delinquency, suggesting that early sexual timing does not, in and of itself, elevate delinquent behavior. Of course, early adolescent sexual activity has been repeatedly linked to other detrimental correlates, notably inconsistent and ineffective contraceptive and condom use, resulting in pregnancy and disease. These biomedical correlates, however, are not inevitable. For example, other Western industrialized countries (e.g., Australia) have similar rates and patterns of teenage sexual activity (Coley and Chase-Lansdale 1998) but have drastically lower rates of teenage pregnancy (United Nations 1991), a difference attributable to American adolescents' comparably scanty sexual health knowledge (Weaver et al. 2005), ineffective contraceptive use (United Nations 1991), and lower abortion rates (Singh and Darroch 2000). Such cross-cultural comparisons suggest that reducing the rates of sexually transmitted disease and adolescent pregnancy may be possible without unduly

pathologizing transitions to sexual activity, even those made relatively early, as inherently and globally damaging.

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