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Early Adverse Environments and Genetic Influences on Age at First Sex: Evidence for Gene \times Environment Interaction

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Youth who experience adverse environments in early life initiate sexual activity at a younger age, on average, than those from more advantaged circumstances. Evolutionary theorists have posited that ecological stress precipitates earlier reproductive and sexual onset, but it is unclear how stressful environments interact with genetic influences on age at first sex. Using a sample of 1,244 pairs of twins and non-twin full siblings from the National Longitudinal Study of Adolescent Health, the present study tested for gene-by-environment interactions ($G \times E$) on age at first sex (AFS). Multivariate interaction models indicated that genetic influences on AFS were suppressed among low-socioeconomic-status (SES) and ethnic-minority compared with higher SES and ethnic-majority youth. Father absence did not uniquely moderate genetic influences on AFS. These results are broadly consistent with previous findings that genetic influences are minimized among individuals whose environments are characterized by elevated risk; however, future research would benefit from samples with larger numbers of individuals at the very low end of the SES spectrum.

Keywords: $G \times E$ interaction, age at first sexual intercourse, environmental adversity, vantage sensitivity, SES

Youth who experience adverse childhood environments initiate sexual activity earlier, on average, than youth from more advantaged circumstances (e.g., Belsky, Steinberg, Houts, Halpern-Felsher, and the National Institute of Child Health and Human Development, 2010; Coley & Chase-Lansdale, 1998). During the last three decades, researchers have advanced several explanatory theories for this association. Most prominently, researchers with evolutionary perspectives draw from the meta-theoretical *life history* framework (Charnov, 1993; Stearns, 1992), which emphasizes

a tradeoff between an organism's allocation of resources to physical growth versus the production of offspring. According to life history theory, organisms in environments with abundant and dependable resources bias the allocation of resources toward a slower, "quality-oriented" reproductive strategy characterized by delayed reproduction and greater investment in fewer offspring. In contrast, organisms in environments with scarce or unstable resources bias the allocation of resources toward a faster, "quantity-oriented" reproductive strategy characterized by early reproduction and limited parental investment in greater number of offspring.

Although life history theory was originally developed to explain interspecies differences in average time to sexual maturity, Belsky, Steinberg, and Draper (1991), in their highly cited *psychosocial acceleration theory*, applied the life history framework to individual differences in human sexual development, including differences in pubertal timing, age at first sex (AFS), and age at childbearing. They posit that a principal function of the first 5–7 years of life is to provide a child with a sense of the availability and predictability of resources and of the trustworthiness and dependability of others. Children from environmentally disadvantaged backgrounds are hypothesized to develop behavior patterns that accelerate reproduction. That is, early rearing environments "set" an individual's reproductive behavior.

Belsky et al. (1991) defined environmental disadvantage rather broadly, including factors such as poverty, father absence, parental fighting, and harsh or inconsistent parent–child relations. Other theorists have emphasized the role of the father as a key determinant of the association between early sexual onset and familial

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ecological stress. Draper and Harpending (1982, 1988) first posited that father absence played a particularly important role in female sexual behavior. Ellis (Ellis, 2004; Ellis et al., 2003) subsequently developed *paternal investment theory*, which emphasizes the quality of paternal caregiving as a key regulator of pubertal timing and onset of reproductive behavior in young girls. Several studies have found that consistent with this theory, father absence, one indicator of low paternal investment, uniquely predicts early onset of sexual activity in girls (e.g., Devine, Long, & Forehand, 1993; Ellis et al., 2003). More recent studies of pubertal timing (Tither & Ellis, 2008) and risky sexual behavior (Ellis, Schlomer, Tilley, & Butler, 2012) have found that variation in the low end of paternal investment appears to be most relevant for regulation of pubertal timing and risky sexual behavior in young girls.

A complicating factor in any theory of environmental mechanisms is the role of genes. Previous behavioral genetic research indicates that age at first sexual intercourse (AFS) is partially heritable, meaning that a proportion of the observable differences in AFS between individuals within a population can be attributed to genetic differences (see Harden, 2013, for a review). The magnitude of heritability estimates for AFS has varied, ranging from relatively modest (.24–.36; e.g., Lyons et al., 2004; Segal & Stohs, 2009; Waldron et al., 2007) to quite substantial (.49–.72; e.g., Dunne et al. 1997; Mustanski, Viken, Kaprio, Winter, & Rose, 2007). There is also evidence for both cohort and gender effects. Most notably, Dunne et al. (1997) found that heritability for AFS was substantially higher for males (.72) and females (.49) born in the late 1950s and the 1960s than for males (.00) and females (.32) born between the 1920s and early 1950s. These findings underscore that heritability estimates are inherently time and population specific and are thus expected to vary as a function of sample characteristics. Indeed, such variability might provide clues with regard to the interplay between environmental context and genetic influences on AFS: Dunne et al. (1997) proposed that as social mores proscribing premarital sex became less culturally salient over successive generations, individual differences in AFS became increasingly a function of genetically influenced characteristics.

Behavioral genetic studies of sexual phenotypes have documented heritable variation in AFS but have not typically considered the interplay between genetic influences and environmental regulators of sexual development. Evolutionary researchers acknowledge the existence of genetic influences on reproductive phenotypes and have convincingly argued that nonzero heritability estimates in industrialized, Western populations do not necessarily invalidate evolutionary arguments (e.g., Ellis, 2004). Nevertheless, this stream of research has primarily focused on how early environments might instigate a cascade of social and psychological outcomes that in turn regulate reproductive strategy, including timing of AFS, and few studies have specifically described how these environmental experiences might interact with genetic influences. The goal in the current article, then, was to incorporate evolutionary thinking regarding the environmental antecedents of sexual timing into behavioral genetic research on age at first sex. Specifically, we investigated *gene-by-environment interaction* ($G \times E$).

In a $G \times E$ interaction, genetic influences on the phenotype depend on environmental context, and an organism's response to

the environment depends on genotype. In quantitative genetic studies, such as described in the current article, $G \times E$ interactions are most often reported in terms of how environmental context moderates genetic influences (although see Harden, Hill, Turkheimer, & Emery, 2008), while candidate $G \times E$ interactions are typically reported in terms of how genetic influences moderate the effect of environmental context. These parameterizations are two sides of the same coin. Throughout the current article, we emphasize both sides of the interaction—how environment depends on genotype and how genotype depends on environment.

Although any finding of significant moderation is generally termed a $G \times E$ effect, there are a number of distinct patterns of $G \times E$ results, each of which is consistent with a different underlying mechanism. First, as predicted by a *diathesis-stress* model, individuals might differ in their genetic vulnerability to adverse environments. Put differently, adverse environments might activate or accentuate genetic vulnerabilities. Consequently, genetic variance—which refers to the variance in a phenotype accounted for by differences in genotype—are higher under conditions of increased environmental adversity and minimized in high-quality environments, as illustrated in the first panel of Figure 1. Second, individuals might differ in their genetic predispositions to profit from advantageous environmental contexts, as predicted by the *bioecological model* (Bronfenbrenner & Ceci, 1994) and more recently by the *vantage sensitivity* model (Pluess & Belsky, 2013). Accordingly, genetic variance increases under conditions of high environmental quality but is suppressed under conditions of low environmental quality, as illustrated in the second panel of Figure 1 (labeled “genetic suppression”). Third, a *differential susceptibility* model posits that people differ in their susceptibility, or plasticity, to environmental influence, such that those with greater plasticity are more sensitive to environments that are marked by both enrichment (leading to outcomes more positive than their less-sensitive counterparts) and deprivation (leading to outcomes more negative than their less-sensitive counterparts (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Ellis, Essex, & Boyce, 2005). Because differential susceptibility predicts that heritable polygenic variation contributes to differences in responses to the environment (Ellis et al., 2012) and that susceptible individuals differ from nonsusceptible individuals most markedly in both very good and very bad environments, genetic variance is maximized at opposing extremes of an environmental moderator (both very low and very high environmental quality) and show negligible influence in “average” environments (e.g., South & Krueger, 2013). This is illustrated in the third panel of Figure 1.

Evolutionary theorists have generally made no specific predictions regarding the expected pattern of interaction between ecological stress and genetic influences for outcomes such as AFS. Moreover, very few behavioral genetic studies have examined $G \times E$ in AFS. The few twin studies that have been conducted, however, have shown diminished genetic influences on sexual behavior and related phenotypes in more adverse or socially constraining environments (consistent with the genetic suppression pattern outlined above). For example, Waldron et al. (2008) found that additive genetic effects accounted for 0% of the variance in AFS for women who had experienced childhood sexual abuse in contrast to 39% for nonabused women. Although childhood sexual abuse is qualitatively distinct from the risk factors examined in the

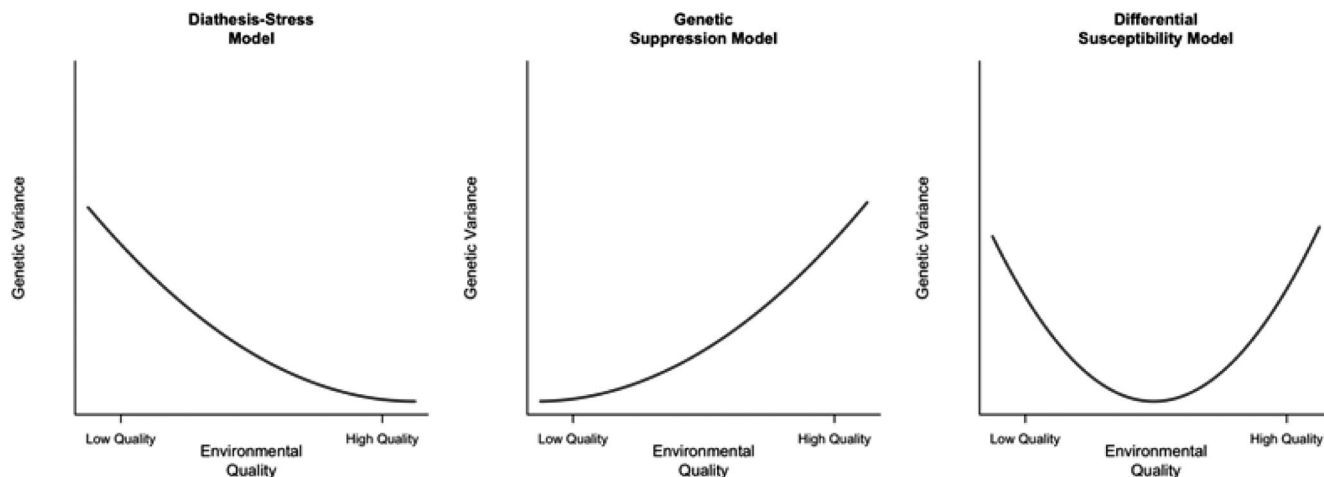


Figure 1. Descriptive patterns of Gene × Environment results.

present study, Waldron et al.'s (2008) results are broadly consistent with the hypothesis that genetic influences on age at first sex might be suppressed in adverse contexts. Similarly, Rodgers, Rowe, and Buster (1999) found evidence for racial differences in the heritability of AFS: Among African American adolescents (who experience, on average, lower average socioeconomic status and higher rates of father absence), the heritability of AFS approached zero, compared with approximately 50% in Whites.

In addition, two candidate gene studies (Gibbons et al., 2012; Manuck, Craig, Flory, Halder, & Ferrell, 2011) have examined the association between life history–relevant phenotypes and specific genetic variants as a function of ecological stress. Manuck et al. (2011) found a significant interaction between a polymorphism of the estrogen receptor- α gene, *ESR1*, and the quality of the family environment in predicting age at menarche. Consistent with the twin model results described, in which genetic differences were strongest among advantaged populations, the difference among *ESR1* genotypes with respect to age at menarche was largest in high-quality family environments. Finally, Gibbons et al. (2012) reported that polymorphisms in the serotonin transporter gene and dopamine D4 gene interacted with racial discrimination to predict “life history strategy cognitions” in African American adolescents; results were consistent with a differential susceptibility pattern. As many results from candidate G × E studies are likely to be false positives (Duncan & Keller, 2011), and neither interaction result has been independently replicated, these results should be interpreted as preliminary.

Goals of the Current Study

The goal of the current study was to test whether three broad markers of environmental risk—low socioeconomic status, biological father absence in childhood, and racial/ethnic minority status—moderate the heritability of AFS. Following previous theoretical and empirical work, we hypothesized that genetic influences on AFS would be minimized for youth who experience each of these environmental risks. In addition, because much research on reproductive timing has focused specifically on early reproductive timing in girls, we included both male and female

adolescents in our sample and examined gender differences in the magnitude of genetic influences on AFS. We tested our hypotheses using a nationally representative sample of twins and non-twin full siblings from the National Longitudinal Study of Adolescent Health.

Method

Participants

Participants comprised a subsample of 1,244 same-sex twin and non-twin full-sibling pairs (281 monozygotic [MZ] pairs, 246 dizygotic [DZ] pairs, 717 nontwin full-sibling pairs) from the National Longitudinal Study of Adolescent Health (Udry, 2003). In order to maximize power to detect interactive effects, we included all same-sex sibling pairs that shared both biological parents (Posthuma & Boomsma, 2000). Forty-nine percent of the sample was male, and the remainder (51%) was female.

Add Health is a nationally representative, longitudinal study targeting adolescent health and risk behaviors. Data were collected in four waves between 1994 and 2009 (Carolina Population Center, 1994–1996; 2001–2002; 2007–2009). Details of the study design and sampling procedure may be found in Bearman, Jones, and Udry (1997) and K. M. Harris et al. (2009). Add Health deliberately oversampled adolescent sibling pairs initially identified through school rosters and adolescent self-reports on an in-school questionnaire completed by 90,000 students just prior to Wave I. From this point, twin pair zygosity was diagnosed by matching 11 molecular genetic markers and by twins' responses to four questionnaire items concerning similarity of appearance (J. Harris, 2006). Similar self-report measures are widely used to determine zygosity in twin research and have been cross-validated with zygosity determinations based upon DNA samples (Loehlin & Nichols, 1970; Spitz et al., 1996). Jacobson and Rowe (1999) found negligible differences for sociodemographic variables (e.g., age, ethnicity, and maternal education) between Add Health sibling pairs and the full Add Health sample.

Measures

Age at first sex (AFS). At Waves I and II, participants reported whether they had ever had vaginal intercourse and, if so, in what month and year they had sex the first time. From these reports, AFS (in years) was calculated. At Waves III and IV, participants were asked whether they had ever had vaginal intercourse and, if so, their age (in years) when they first had sex. As in previous studies with this data set (e.g., Harden et al., 2008), analyses were conducted using the AFS from the earliest wave in which the participant reported having had sex in order to minimize telescoping. For example, if an adolescent reported having had sex at age 13 at Wave I and at age 14 at Wave II, the Wave I report was used. Because we were interested in voluntary first sex, when non-virgin participants reported an AFS that was likely prepubertal and possibly nonconsensual (<11 years), they were coded as missing ($n = 104$ individuals), resulting in a measure of AFS ranging from 11 to 30 years ($M = 17.16$, $SD = 2.88$). Participants who did not endorse an age at first sex by the last reporting wave were also coded as missing ($n = 336$ individuals). The correlation between AFS in the first and second sibling of each pair was 0.33 in DZ twins, 0.40 in non-twin full-sibling pairs, and 0.56 in MZ twins. The correlations in AFS across study waves ranged from .42 to .85. Reliability of reports of AFS across waves have been extensively studied in the Add Health data, and reporting errors tend to be largely random and have little impact on the conclusions drawn from the estimated ages at first sex (e.g., Upchurch, Lillard, Aneshensel, & Fang Li, 2002).¹

Biological father absence. At Wave I, participants were asked whether they were living with their biological father and, if not, to indicate at what age they had they last lived with him. From this information, a variable was created to index biological father absence at or before the age of 10. (This cutoff was chosen to ensure that father absence temporally preceded AFS; later, we report results from post hoc sensitivity analyses in which the cutoff age for father absence was varied.) In the rare instance in which siblings living in the same household were discordant in their endorsement of biological father absence, we coded the pair father as absent. Of the 1,244 sibling pairs, 361 (29%) reported father absence at or before the age of 10.

Socioeconomic status (SES). Socioeconomic status was measured using residential parent's mean level of educational attainment. Educational attainment is a commonly used index of socioeconomic status (Bradley & Corwyn, 2002), which might be more stable than family income (U.S. Treasury Department, 2008) and has been used in previous $G \times E$ analyses (e.g., Harden, Turkheimer, & Loehlin, 2007). Educational attainment was coded on a 9-point ordinal scale ranging from *eighth grade or less* to *professional training beyond a 4-year degree*. The median level of SES in the study sample was a score of 5 (equivalent to a general education degree [GED] or high school graduate), and the mean score was 5.25 ($SD = 2.14$).

Race/ethnicity. In terms of racial/ethnic identity, 38% of our sample identified as either African American or Hispanic (among this 38%, 56% identified as African American and 44% Hispanic) and the remainder (62%) identified as White. Race was dummy-coded such that 1 corresponded to either African American or Hispanic and 0 corresponded to White.

Table 1
Correlations Among Study Variables

| Variable | 1 | 2 | 3 | 4 | 5 |
|-------------------------|-------------|-------------|------------|------|------|
| 1. Age at first sex | 1.00 | | | | |
| 2. Socioeconomic status | .14 | 1.00 | | | |
| 3. Father absence | -.13 | -.05 | 1.00 | | |
| 4. Race | -.10 | -.28 | .06 | 1.00 | |
| 5. Gender | .04 | -.07 | -.02 | .03 | 1.00 |

Note. Correlations based on one twin per pair, selected at random. Pearson correlations are presented for continuous variables; phi coefficients for the associations among dichotomous variables. Values significantly different from zero at $p < .05$ are in bold.

Gender. Gender was coded such that 1 corresponded to males and 2 to females. Table 1 summarizes the relations among AFS and the four moderating variables. Consistent with prior epidemiological literature, adolescents from higher SES homes reported a later AFS, on average, whereas adolescents from father absent and racial/ethnic minority homes reported earlier AFS. Moreover, racial/ethnic minority adolescents were more likely to experience father absence and had lower SES.

Analyses

Data were analyzed using a series of structural equation models (SEM) with the software program Mplus (Muthén & Muthén, 1998–2007). Model fit was evaluated using differences in model log-likelihood and root-mean-square error of approximation (RMSEA). RMSEA values less than 0.05 indicate good model fit (Browne & Cudeck, 1993).

First, we estimated genetic and environmental influences on AFS using a univariate biometric model (Neale & Maes, 2007). This model partitions the variance of a phenotype (here, AFS) into additive genetic effects (A), shared environmental effects (C ; family-level experiences that serve to make siblings more similar), and nonshared environmental effects (E ; environmental experiences that are uncorrelated between twins, plus measurement error).² This methodology capitalizes on the difference in genetic similarity between MZ and DZ twins to make inferences about the relative contributions of genes and environments to a given phenotype. The correlation between the A components in the first and second sibling in each pair is fixed to 1.0 in MZ twins and 0.5 in DZ twins and non-twin full siblings. In the context of the model MZ and DZ twins and non-twin full siblings share 100% of their common, or shared, environment and 0% of their unique, or nonshared, environment. Thus the correlation between the C component in the first and second sibling is fixed to 1.0 in all pair types, whereas the E correlation is fixed to 0 in all pair types.

¹ Using the Add Health data, Upchurch et al. (2002) evaluated the conclusions from seven analyses of age at first sex, each based upon a separate assumption for coding reported age of first intercourse, and found that all seven analyses reached very similar conclusions.

² Although conventionally labeled the *nonshared environmental factor*, this factor represents variation due to factors that differ within MZ twin pairs. To the extent that MZ twins are not, in fact, perfectly genetically identical (Charney, 2012), the effects of that within-MZ variation will be reflected in E .

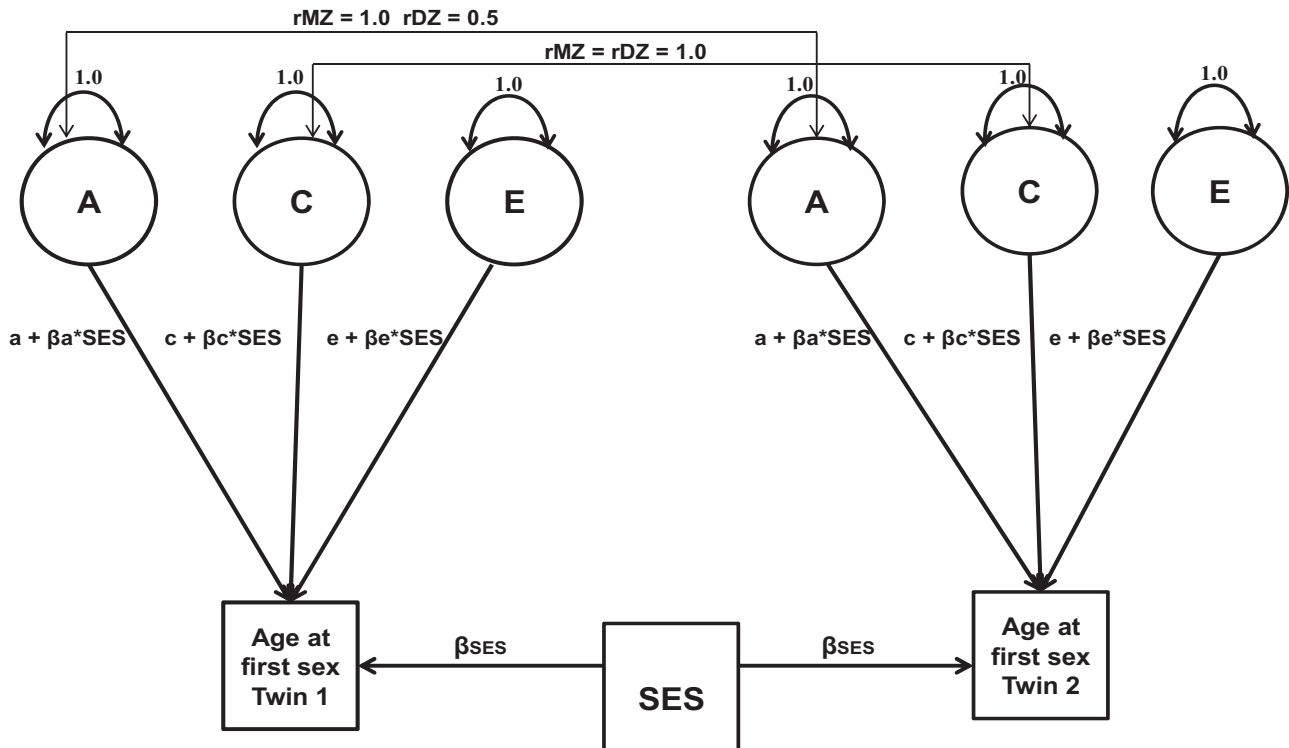


Figure 2. Path diagram of Gene × Environment interaction model. A, C, and E represent the univariate additive genetic, shared environmental, and nonshared environmental variance components, respectively; β_a , β_c , and β_e represent the moderated components of a, c, and e, respectively; and β_{SES} represents the main effect of socioeconomic status on age at first sex. MZ = monozygotic pairs, DX = dizygotic pairs.

To test for $G \times E$ effects, we used a model that was designed to test interactions between a measured environmental moderator and the paths from the latent genetic and environmental factors (Purcell, 2002); see Figure 2 for an example using SES as the moderator. First, the main effect of the moderator variable, SES, on AFS is estimated as β_{SES} . The variance in the outcome variable (i.e., AFS) that is *unique* of the moderator is divided into latent A, C, and E components. In addition, the paths from the A, C, and E components to AFS are allowed to interact with the moderator variable (e.g., path labeled $a + \beta_a * SES$). Thus, for the interaction model using SES as a moderator, AFS was modeled as follows:

$$AFS = \beta_{SES} + (a + \beta_a * SES)A + (c + \beta_c * SES)C + (e + \beta_e * SES)E \quad (1)$$

The presence of moderation can be inferred when an interaction term, β_a , β_c , or β_e is significantly different from zero. In the case of *gene*–environment interaction in particular, this would refer to a significant β_a term. For example, a significant and positive β_a term would indicate that as SES increases, the genetic variance in AFS also increases. Conversely, a negative β_a term would indicate that as SES increases, the genetic variance in AFS decreases. To address concerns about gene–environment correlation (e.g., Mendle et al., 2006; 2009), we controlled for gene–environment correlation by including the main effects of each moderator.

Results

Genetic and Environmental Influences on Age at First Sex: Population Averages

The first column in the top of Table 2 (“Main effects only”) shows the parameter estimates from the univariate ACE model for AFS, without any moderator effects. These results represent the average contribution of genetic and environmental variation in the sample as a whole. Additive genetic effects accounted for 38% of the variance in AFS, $1.74/(1.74 + 1.29 + 1.84)$, shared environmental effects for 21%, and unique environmental effects for 42%.

Moderation by SES

Model 2 tested whether SES moderated the magnitude of genetic and environmental influences on AFS. Parameter estimates for Model 2 are summarized in Table 2 (“SES interaction” under “Univariate models”). Overall, the interaction model fit the data better than a reduced “main-effect-only” model in which all the interaction effects were fixed to zero ($\Delta\chi^2 = 52.88$, $\Delta df = 3$, $p < .001$). There was a significant main effect of SES, whereby each unit increase in SES corresponded to just under a 2.5-month increase in AFS. Neither C nor E showed any significant interaction effects with SES, but there was a significant $G \times E$ interaction, illustrated in Figure 3A. Visual representation of

Table 2

Unstandardized Additive Genetic (A) and Shared (C) and Nonshared (E) Environmental Variance Components for Age at First Sex

| Parameters | Main effects only | SES interaction | Father absence interaction | Race/ethnicity interaction | Gender interaction |
|----------------------|--------------------|---------------------|----------------------------|----------------------------|---------------------|
| Univariate models | | | | | |
| <i>a</i> | 1.74 (0.22) | 0.40 (0.33) | 1.90 (0.22) | 2.09 (0.23) | 1.97 (0.77) |
| <i>c</i> | 1.29 (0.22) | 1.44 (0.10) | 1.18 (0.26) | 0.79 (0.45) | .64 (1.08) |
| <i>e</i> | 1.84 (0.08) | 1.98 (0.05) | 1.83 (0.10) | 1.69 (0.10) | 2.46 (0.29) |
| <i>b_M</i> | — | 0.20 (0.04) | -0.74 (0.16) | -0.64 (0.14) | 0.19 (0.14) |
| <i>b_a</i> | — | 0.57 (0.05) | -1.35 (0.45) | -1.34 (1.02) | -0.09 (0.44) |
| <i>b_c</i> | — | 0.07 (0.10) | 0.35 (0.31) | 0.87 (0.54) | 0.32 (0.59) |
| <i>b_e</i> | — | -0.07 (0.04) | -0.09 (0.15) | 0.41 (0.18) | -0.41 (0.17) |
| Multivariate models | | | | | |
| <i>a</i> | 0.41 (0.61) | | | | |
| <i>c</i> | 1.55 (0.40) | | | | |
| <i>e</i> | 2.56 (0.21) | | | | |
| <i>b_M</i> | — | 0.19 (0.04) | -0.57 (0.16) | -0.30 (0.15) | 0.17 (0.14) |
| <i>b_a</i> | — | 0.44 (0.08) | 0.21 (0.29) | -0.74 (0.35) | 0.52 (0.24) |
| <i>b_c</i> | — | -0.05 (0.11) | -0.63 (0.59) | 0.47 (0.44) | -0.31 (0.34) |
| <i>b_e</i> | — | -0.07 (0.03) | -0.28 (0.17) | 0.12 (0.14) | -0.40 (0.12) |

Note. All estimates unstandardized (in units of years). Univariate models estimated interactions with each moderator separately; multivariate model estimated interactions with all moderators simultaneously. Abbreviations: SES = socioeconomic status; *a* = additive genetic effects, *c* = shared environmental effects, *e* = nonshared environmental effects; *b_M* = main effect of moderator on age at first sex; *b_a*, *b_c*, and *b_e* = interactions between the moderator and the A, C, and E components, respectively. Parameters significantly different than zero at $p < .05$ are in bold.

the interaction reveals a U-shaped curve suggestive of a differential-susceptibility effect. Among adolescents whose parents had only a high school education, additive genetic effects accounted for no variation in AFS, whereas among adolescents whose parents had graduated from college, additive genetic effects accounted for 43%.

In addition, there also appeared to be an uptick in genetic variance at very low levels of SES. Based on the 95% confidence intervals around the estimated genetic variance at each level of parental education, however, genetic influences on AFS at the low end of parental education were significantly different from zero only at the very lowest level of parental education (less than eighth grade). Only 3.4% of twin pairs had this level of parental education. Roisman et al. (2012) argued that the *proportion affected* “offers a pragmatic way of evaluating evidence for differential susceptibility,” as “the model is of limited use if only a small number of individuals experience the theorized [effects]” (p. 396). They suggested a cutoff of proportion affected $> 16\%$; based on this recommendation, we “question whether [our] data are consistent with differential susceptibility theory” (p. 396).

An alternative way to represent this interaction, more directly parallel to how results from candidate Gene \times Environment studies are typically presented, is to plot the predicted relationship between SES and AFS for two values on the latent *A* factor, which represents genetic predispositions for later versus earlier age at first sex (shown in Figure 3B). Higher socioeconomic advantage was positively associated with later AFS for youth with higher scores on the latent *A* factor (+1 *SD* above the mean). In addition, consistent with the U-shaped curve for genetic variance, there was a crossover effect potentially suggestive of differential susceptibility, as youth with higher scores on the latent *A* factor showed the earliest AFS at low levels of SES. As discussed earlier, however, the difference between genotypes (i.e., the genetic variance) was not significant at the low end of SES except for the few pairs whose parents had less than an eighth grade education.

Overall, results from the SES moderation models suggest that genetic influences on AFS are actuated in high-SES environments but minimal in low-SES environments. Put differently, high-SES environments facilitate a later AFS, but only in those individuals with particular genetic predispositions.

Moderation by Biological Father Absence

Model 3 tested for moderation effects of father absence. Parameter estimates from Model 3 are summarized in the third column of Table 2 (“Father absence interaction” under “Univariate Model”). There was a significant main effect of father absence, whereby children who experienced father absence at or before age 10 experienced AFS nearly 9 months earlier, on average, than their father-present counterparts. There was also a significant $G \times E$ interaction. For individuals who did not experience father absence at or before age 10, additive genetic effects accounted for 43% of the variation in AFS; in contrast, for individuals whose biological fathers were absent at or prior to age 10, additive genetic effects accounted for only 5% of the variation in AFS. Neither *C* nor *E* showed any significant interaction effects with early father absence. Overall, the interaction model fit the data better than a reduced “main-effect-only” model in which all the interaction effects were fixed to zero ($\Delta\chi^2 = 20.98$, $\Delta df = 3$, $p < .001$).

Moderation by Race/Ethnicity

Model 4 tested for moderation effects of race/ethnicity. Parameter estimates for Model 4 are summarized in the fourth column of Table 2 (“Race/ethnicity interaction” under “Univariate models”). There was a significant main effect of race/ethnicity. Children who identified as African American or Hispanic tended, on average, to experience AFS just over 7.5 months earlier than Whites. In terms of the moderation model, neither *A* nor *C* showed any significant moderation. There was, however, a significant *E* by race/ethnicity

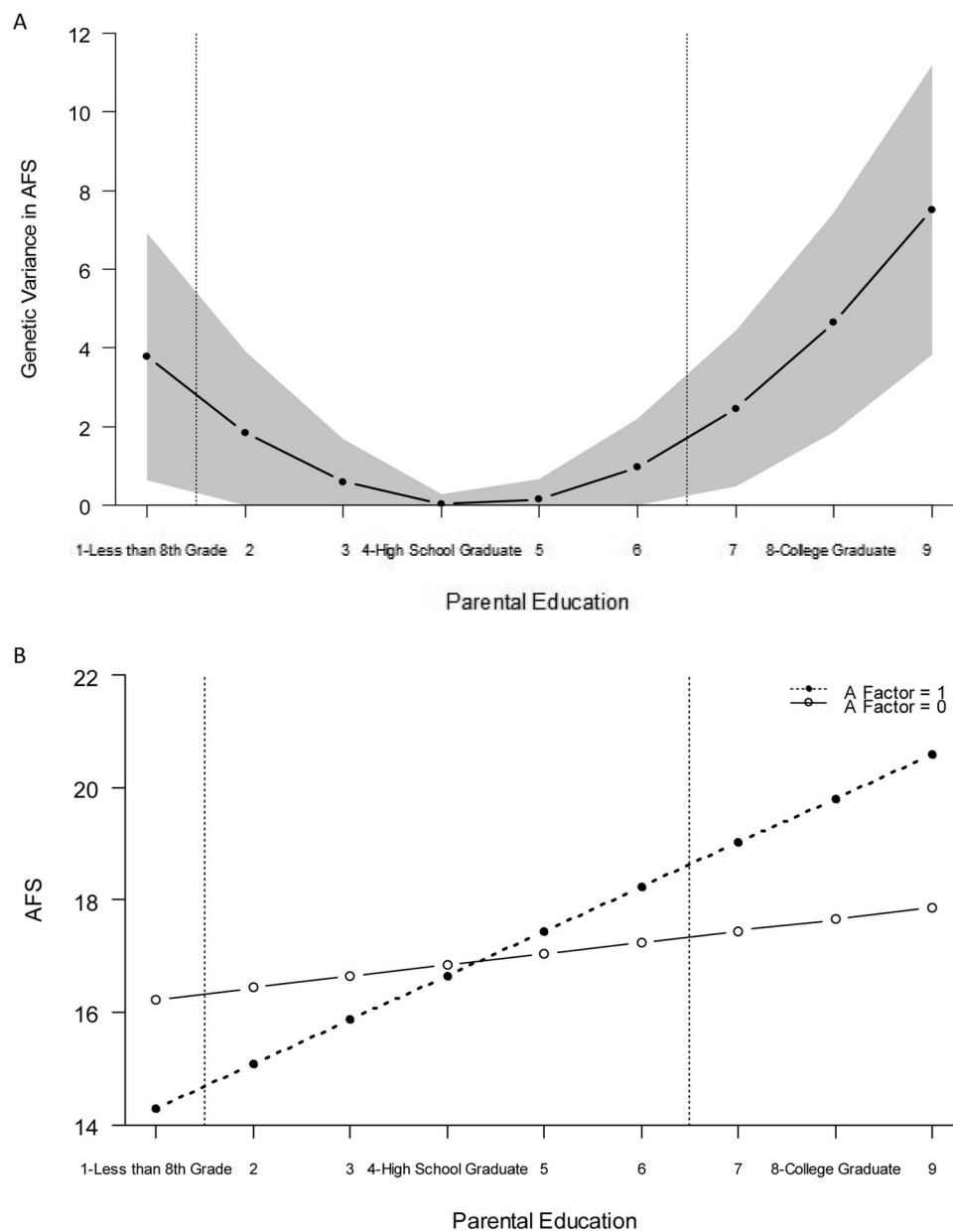


Figure 3. Gene × Socioeconomic Status (SES) interaction on age at first sex (AFS). Figures 3A and 3B are based on parameters from univariate model of SES interaction (Table 2). Vertical dashed lines delineate regions of significance; genetic variance (i.e., differences between genotypes) is significantly different from zero to the left of the first line and the right of the second line. Panel A: 95% confidence interval around estimate for genetic variance is shown in shaded section. Genetic variance is plotted in unstandardized form (in units of years). Panel B: A factor = additive genetic factor illustrated in Figure 2. Values represent the mean and +1 standard deviation above the mean on the latent factor.

interaction. Unique environmental variance accounted for roughly 36% of the variance in AFS for White youth, compared with 60% for African American or Hispanic youth. The pattern for additive genetic variance mirrored the $G \times E$ effects observed for SES and father absence in that the additive genetic variance in AFS tended to be suppressed for African American and Hispanic individuals and amplified for White individuals, although this interaction did not reach customary significance thresholds ($p < .05$). In addition,

the interaction model overall did not fit the data significantly better than a reduced “main-effect-only” model in which all the interaction effects were fixed to zero ($\Delta\chi^2 = 5.82$, $\Delta df = 3$, $p = .12$).

Moderation by Gender

Model 5 tested for moderation effects of gender, as summarized in the final column of Table 2 (“Gender interaction” under “Uni-

variate models”). There was no significant main effect for gender. Neither *A* nor *C* showed any significant moderation; however, there was a significant *E* by gender interaction. For females, unique environmental variance accounted for 51% of the variance in AFS, whereas for males, unique environmental effects accounted for 60% of the variance. Overall, the interaction model fit the data better than a reduced “main-effect-only” model in which all the interaction effects were fixed to zero ($\Delta\chi^2 = 16.82$, $\Delta df = 3$, $p < .001$).

Multivariate Interaction Model

Because of the overlap among SES, racial/ethnic minority status, and father absence, our final model tested all interactions simultaneously in a multivariate interaction model. This model tests whether each moderator uniquely interacts with genetic and environmental influences on AFS above and beyond its relation with the other moderators. The bottom half of Table 2 summarizes the parameter estimates from the multivariate interaction model. Six results were notable. First, SES, father absence, and race/ethnicity all had significant unique main effects on AFS. Second, the interaction between SES and additive genetic variance in AFS remained significant in the full model. As was observed when SES was entered as the only moderator, additive genetic effects accounted for greater variation in AFS among youth from more advantaged backgrounds. Third, a nonshared environmental interaction with SES emerged as significant, suggesting that as SES increased, the nonshared environment became less influential on AFS. Fourth, the interaction between father absence and genetic variance in AFS was no longer statistically significant in the full model. Fifth, the interaction between race/ethnicity and nonshared environmental variance was also no longer significant once entered into the full model. However, race/ethnicity did moderate the additive genetic variance for AFS in the full model, with genetic variation suppressed among Black and Hispanic youth compared with White youth. Sixth, the interaction between gender and nonshared environmental variance maintained its significance once entered into the full model. In addition, the interaction between gender and the latent additive genetic variance became statistically significant, with females showing greater additive genetic influence on AFS than males. Overall, the fit of the full multivariate interaction model was significantly better than the fit of a reduced model, in which all interaction effects were fixed to zero ($\Delta\chi^2 = 75.06$, $\Delta df = 12$, $p < .0001$).

Post Hoc Sensitivity Analyses

Because evolutionary-developmental theory emphasizes the first 5–7 years of life as particularly sensitive to environmental input, we conducted a series of post hoc sensitivity analyses assessing father absence using alternate age cutoffs (ages 5, 6, and 7) and as a continuous measure (number of years of father absence before age 10). In none of these scenarios was father absence a statistically significant moderator of genetic and environmental influences on AFS. Full results of these sensitivity analyses may be obtained upon request.

Discussion

Youth who experience environmental adversity tend to initiate sexual intercourse at an earlier age. The present study tested whether three broad markers of environmental risk—low SES, biological father absence in childhood, and racial/ethnic minority status—moderated the heritability of AFS. Our results suggest that genetic influences for age at first sex are greater in contexts of relative social advantage and suppressed in more adverse conditions. In particular, genes are a stronger predictor of timing of first sex among high-SES and White individuals and contribute negligibly to AFS among low-SES and ethnic-minority adolescents. These findings are consistent with previous $G \times E$ interaction studies of environmental adversity both for AFS (Waldron et al., 2008) and for other phenotypes (e.g., intelligence; Turkheimer et al., 2003). In addition, father absence did not uniquely moderate genetic influences on AFS in a full model that simultaneously controlled for SES and race/ethnicity, suggesting that father absence, per se, might not be the most potent environmental precursor to early sexual activity but rather a “proxy” for a larger matrix of social disadvantage.

In addition, although we obtained significant shared environmental variance in AFS in the sample as a whole (21% in a model with no moderation), it should be noted that mean differences between race/ethnic groups in age at first sex will lead to higher estimates of shared environmental variance. This finding might be attributable to the high levels of racial and ethnic diversity of the Add Health sample. In support of this interpretation, the estimate of shared environmental variance from a model that included the main effect of race/ethnicity (“Race interaction model” in Table 2) was smaller (0.79 vs. 1.29) and no longer significantly different than zero.

To make sense of our results, it is important to remember that there is not a single gene for age at first sex; rather, genetic influences on sexual timing are likely mediated through a complex constellation of physiological (e.g., pubertal timing and physical attractiveness), motivational (e.g., sexual drive), and behavioral (e.g., sensation seeking, substance use, and religiosity) traits. Therefore, the finding of higher genetic variance in advantaged populations indicates that these “embodied characteristics matter strongly and pervasively as causes” of individual sexual behavior (Freese, 2008, p. S20), but *only* for individuals who occupy positions of relative social privilege and economic security. The relevant question, then, is how these links between embodied characteristics and sexual behavior are disrupted under conditions of social disadvantage. One explanation that could account for both the main effects of adverse environments on the average age at first sex and the moderating effects on genetic variance in age at first sex is that individuals who would otherwise be genetically predisposed toward later sexual intercourse (via, for example, later pubertal timing, greater religiosity, reduced sensation seeking, or greater anxiety) are shaped by the social context in which they live to initiate sexual intercourse earlier. For example, several studies have shown that media consumption, which tends to correlate with riskier sexual attitudes, is greater among low-SES youth (e.g., Blosser, 1988; Ward et al., 2005). This might result both in reduced genetic variation in age at first sex and earlier mean ages at first sex—consistent with our findings.

Previous research on racial differences in the relation between pubertal timing and initiation of sexual activity in adolescent girls would also be consistent with this explanation. Large epidemiological samples have shown nontrivial heritability estimates for pubertal timing (e.g., Harden, Mendle, & Kretsch, 2012), and genetic influences on age at menarche have been found to overlap with genetic influences on age at first sex (Rowe, 2002). That is, part of the genetic influence on age at first sex—at least in girls—can be accounted for by heritable differences in the onset of puberty. However, after controlling for mean group differences in pubertal timing as a function of race, Cavanagh (2004) found that the phenotypic association between pubertal timing and age at first sex was moderated by race: later pubertal timing was associated with delayed sexual initiation among White but *not* among African American girls. In explaining her results, Cavanagh (2004) noted that “differences in the social construction of girlhood must be taken into account when examining the pathways that make up the human life course” (p. 306). Although puberty might be the time in which many Whites first become aware of themselves as sexually mature, African American girls tend to be overly sexualized in American culture (hooks, 1992). Consequently the pubertal transition might not hold the same significance for African American girls, both in terms of how they view themselves and how they are viewed by others (Cavanagh, 2004).

Earlier sexual intercourse has sometimes been conceptualized under a higher order domain of externalizing (or disinhibited) behavior (Jessor & Jessor, 1977). In contrast to our finding of decreased heritability of sexual behavior in disadvantaged contexts, Hicks, South, DiRago, Iacono, and McGue (2009) found that the heritability of adolescent antisocial behavior increased in the context of multiple indicators of environmental adversity (deviant peer relations, poor parent–child relations, and poor academic engagement). Although precocious sexual activity is correlated with externalizing behaviors, it is also qualitatively unique in important ways. For instance, although earlier intercourse in some individuals is likely influenced by the hallmark characteristics of externalizing behavior, such as sensation seeking and impulsivity, it might also be part of an integrated life-history strategy (Ellis et al., 2004). In addition, recent research has shown that early sexual activity within the context of a long-term monogamous relationship might actually be associated with decreased levels of delinquent behavior (Harden et al., 2008; McCarthy & Grodsky, 2011).

Limitations and Future Directions

There are a number of methodological considerations that are important to note. First, although we have interpreted SES as an index of differences in environmental advantage, parental educational attainment also reflects genetic differences between parents (Rowe, Vesterdal, & Rodgers, 1998). This occurs because educational attainment is partially contingent on heritable traits such as intelligence, conscientiousness, and attentional capacity. Although the biometric model controlled for genetic variance common to educational attainment and AFS, we were unable to rule out the possibility that increased heritability in AFS might not be better accounted for by a Gene × Gene interaction rather than a Gene × Environment interaction. In addition, the magnitude of the genetic correlation between AFS and SES remains unknown. Because raised-together biological sibling pairs are necessarily identical for

parental characteristics such as SES, however, twin modeling is not genetically informative in this regard.

Second, while we were interested in obtaining reports on voluntary AFS, the limitations of the AFS definition preclude our ability to ascertain with 100% certainty that all sexual experiences were indeed voluntary. Third, like many other researchers, we used father absence as an indicator of paternal investment. Although father absence is highly correlated with factors broadly related to low paternal investment, such as diminished relationship quality and emotional distance (e.g., Cooksey & Craig, 1998; Gorvine, 2010), recent studies have shown that alternative indicators of (low) paternal investment, such as paternal psychopathology, substance abuse, and legal troubles, might be better predictors of daughters’ development than a dichotomous father present–father absent distinction (e.g., Ellis et al., 2012; Ellis & Essex, 2007). It will be important for future studies to assess alternative indicators of paternal investment, including indicators indexing the positive end of the spectrum, before an unequivocal interpretation can be made for its role in moderating the heritability of AFS.

Fourth, although African Americans and Hispanics both show earlier age at first sex relative to Whites and both endure the effects of racism in American culture, there are important socio-cultural differences between these two groups. Unfortunately, due to sample size, we did not have adequate power to estimate differences between these minority groups. Finally, more generally, quantitative genetic models require very large numbers of participants to distinguish between different patterns of G × E (e.g., differential susceptibility vs. genetic suppression). In the case of the present study, the results of our SES moderation models and post hoc sensitivity analyses appear to be consistent with genetic suppression. However, because of the comparatively small number of families at the very low end of the SES spectrum (i.e., less than high school education), it is worth being cautious about whether our results reflect a differential susceptibility versus genetic suppression effect. This ambiguity underscores the need for behavioral genetic research to include adequate numbers of socioeconomically disadvantaged and minority families, who are currently underrepresented in the majority of twin samples.

Conclusion

In the present study, we used behavioral genetic methodology to investigate the genetic and environmental etiology of individual differences in AFS. We tested for the presence of Gene × Environment interaction using three broad indices of environmental risk. Individuals whose backgrounds were characterized by relative advantage showed greater genetic influence in AFS. Conversely, genetic effects were suppressed for individuals whose backgrounds were characterized by relative disadvantage. These results suggest that among adolescents who have fewer social and economic resources to draw upon, AFS is increasingly influenced by family-level environmental circumstances rather than genetic propensities.

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