Why Does Early Sexual Intercourse Predict Subsequent Maladjustment? Exploring Potential Familial Confounds

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Objective: Previous studies have found an association between early age at first sexual intercourse and subsequent psychosocial maladjustment. Using a quasi-experimental approach, we examined the extent to which this observed association may be due to familial confounds not explored in prior research.

Methods: Using a population-based cohort of Swedish adult twins (ages 19–47; \( N = 12,126 \)), we examined the nature of the association between early sexual intercourse (i.e., first intercourse occurring before age 16) and various outcomes reflecting psychosocial health, including substance use, depression, criminal convictions, and adolescent childbearing. We used two methods—discordant-twin analyses and bivariate twin modeling—to estimate the extent to which genetic and environmental confounds explained observed associations. Results: Individuals who engaged in early intercourse were at greater risk for most of the adverse psychosocial health outcomes measured in this study. However, twin pairs discordant for engaging in early intercourse did not differ significantly in their risk for psychosocial maladjustment. Our results indicated that early age at first sexual intercourse and subsequent psychosocial maladjustment may be associated because of familial factors shared by twins. Conclusions: Early intercourse may be associated with poor psychosocial health largely due to shared familial influences rather than through a direct causal connection. Therefore, effective and efficient interventions should address other risk factors common to early intercourse and poor psychosocial health.

Keywords: adolescence, sexual behavior, sex-education programs, behavior genetics

The formation of sexual identity and healthy relationships are important aspects of adolescent development (Fortenberry, 2003; Halpern, 2010; Tolman & McLelland, 2011), and most individuals report that they have had sexual intercourse by the age of 18 (Danielsson, Rogala, & Sundström, 2001; Eaton et al., 2010; Guttmacher Institute, 2001). However, younger sexually active adolescents are more likely to engage in behaviors that increase their risk of unintended pregnancy and the contraction and spread of sexually transmitted diseases (Buston, Williamson, & Hart, 2007; Danielsson et al., 2001; Hollander, 2009; Kaeble, Halpern, Miller, & Ford, 2005; Sandfort, Orr, Hirsch, & Santelli, 2008). The reduction of adolescent sexual risk behaviors—including delaying the onset of sexual activity—could significantly reduce the public health burden associated with these outcomes (Chesson, Blandford, Gift, Tao, & Irwin, 2004; Hoffman, 2006; Sonfield, Kost, Gold, & Finer, 2011).

Public policy focused on delaying sexual activity has posited that adolescent intercourse may have also negative effects on psychological health (Social Security Act, 1996). Previous research indicates that intercourse in early adolescence (hereafter referred to as “early sex”) tends to co-occur with early adolescent alcohol, tobacco, and illicit substance use (Kotchick, Shaffer, Forehand, & Miller, 2001; Siebenbruner, Zimmer-Gembeck, & Egeland, 2007; Zimmer-Gembeck & Collins, 2004), depressive symptoms (Lehrer, Shrier, Gortmaker, & Buka, 2006; Zimmer-Gembeck & Hefand, 2008), and general delinquency (Zimmer-Gembeck & Hefland, 2008). Early sex is also associated with young-adult delinquency (Armour & Haynie, 2007) and substance use disorders (Cornelius, Clark, Reynolds, Kirisci, & Tarter, 2007; McGue & Iacono, 2005), but it has less consistent associations with young-adult depressive symptoms (McGue & Iacono, 2005; Meier, 2007; Rector, Johnson, Noyes, & Martin, 2003; Spriggs & Halpern, 2008).

However, individuals do not engage in early sex or experience adverse psychosocial health outcomes at random. Twin and family studies indicate that familial influences—genetic and/or environmental influences shared by individuals within a family that make siblings or twins similar—contribute to variability in age at first...
intercourse (Mustanski, Viken, Kaprio, Winter, & Rose, 2007; Rodgers, Rowe, & Buster, 1999); young-adult substance use, abuse, and dependence (Kendler et al., 1999; McGue, Pickens, & Sviks, 1992; van den Bree, Johnson, Neale, & Pickens, 1998); lifetime major depression (Kendler, Gardner, & Pedersen, 2006); current depressive symptoms in adulthood (Kendler et al., 1994); criminal activity (Frisell, Lichtenstein, & Långström, 2011); and adolescent childbearing (Waldron et al., 2007).

It is possible that familial influences contributing to the likelihood of engaging in early sex overlap with familial influences that increase vulnerability to adverse psychosocial outcomes. These common influences could include genes related to personality traits such as sensation seeking and impulsivity (Verweij, Zietsch, Bailey, & Martin, 2009) or a predisposition for disinhibited behavior (McGue & Iacono, 2005). Environmental factors shared by family members (Dick, Johnson, Viken, & Rose, 2000), such as living in a disadvantaged or unstable household or neighborhood or being raised by parents with low education, poor psychological health, or certain attitudes about engaging in risk-taking behavior, could also contribute to early sex and psychosocial maladjustment (Kirby, 2003; Roche et al., 2005).

Almost all existing research regarding the psychosocial consequences of early sex has compared unrelated individuals to each other (Zimmer-Gembeck & Helfand, 2008). Such studies are unable to account for between-family differences that could be driving the observed associations (Rutter, 2007; Rutter et al., 2010; Shardish, Cook, & Campbell, 2002). Quasi-experimental methods (Shadish et al., 2002), such as the comparison of twins discordant for risk-factor exposure, can help address the limitations of previous research by controlling for possible familial confounds (Lahey, D’Onofrio, & Waldman, 2009; McGue, Osler, & Christensen, 2010; Rutter, 2007). Discordant-twin analysis provides an estimate of the association between early sex and each outcome that remains after controlling for genetic and environmental confounds.

A more complete understanding of the association between early sex and young-adult adjustment has important implications for public health intervention. If early sex causes adverse psychosocial health outcomes, delaying intercourse onset would reduce the likelihood that these outcomes occur. However, if early sex and adverse psychosocial health outcomes are associated because they are both influenced by a third, unmeasured variable (e.g., familial confounds), then delaying intercourse onset would not effectively reduce the likelihood of these negative outcomes. Recent studies using the discordant-twin approach have found that early sex is no longer associated with young-adult delinquency (Harden, Mendle, Hill, Turkheimer, & Emery, 2008) or young-adult sexual risk behaviors, including adolescent pregnancy (Huibregtse, Bonvalova, Hicks, McGue, & Iacono, 2011), after controlling for genetic and environmental influences shared by twins. These results suggest that the observed associations are entirely due to familial confounding.

In the current study, we examined the nature of the association between early age at first intercourse and several psychosocial health outcomes in young adulthood, including cigarette use, cannabis use, alcohol abuse/dependence, a major depressive episode, current depressive symptoms, criminal conviction, and adolescent childbearing. Using a genetically informative, population-based sample of adult Swedish twins, we tested whether these associations were explained by unmeasured familial confounds. We then used bivariate twin modeling to estimate the degree to which genetic and environmental confounds were responsible for the covariation between early sex and these outcomes.

**Methods**

**Sample**

The Study of Twin Adults: Genes and Environment (STAGE) is a registry of adult Swedish twins. The original purpose of STAGE was to assess lifetime history of medical illness, mental disorders, and other health-related behaviors (Lichtenstein et al., 2006). All twin pairs born in Sweden between 1959 and 1985 in which both twins lived past 1 year of age (n = 42,582) were invited to complete a Web-based survey or a telephone interview in November 2005 through March 2006. The Regional Ethics Committee at the Karolinska Institutet approved data collection, and participants provided consent while completing the assessment. Participants completing the phone interview were also mailed a separate paper questionnaire assessing potentially sensitive information, including sexual behavior. The total response rate was 59.6%, with 25,381 individuals completing assessments (including 11,235 complete twin pairs plus 2,911 individuals whose co-twin did not participate). The 25,381 STAGE responders did not differ from STAGE nonresponders (n = 17,201) with regard to age, birth weight, or lifetime diagnosis of any neurological condition. Responders were more likely to be female and have Swedish-born parents and less likely to have a history of criminal offending or any psychiatric disorder. Responders were more educated, and responding males had higher intellectual performance scores at the time of conscription (Furberg et al., 2008). We also compared STAGE responders to all individuals born in Sweden between 1959 and 1985 (n = 2,859,123) using data from the Swedish National Crime Register (Fazel & Grann, 2006) and Education Register (Statistics Sweden, 2011). Compared to all individuals born in Sweden between 1959 and 1985, STAGE responders had a comparable mean level of education, prevalence rate of criminal conviction (violent and nonviolent, including driving-related offenses), and parental education and parental criminal history (Donahue, D’Onofrio, Lichtenstein, & Långström, in press).

In the current analyses, we examined only monozygotic (MZ) twin pairs and same-sex dizygotic (DZ) twin pairs and only included pairs in which both twins participated in assessments. These inclusion criteria resulted in a subsample of 12,126 individuals from 6,063 twin pairs (3,548 MZ and 2,515 DZ pairs). Of these individuals, 60% were female, and the average age at assessment was 33 years.

**Measures**

**Early sex.** Participants were asked whether they had ever engaged in voluntary sexual intercourse, and, if so, at what age they first had intercourse. First intercourse occurring before the age 16 was categorized as early sex, in keeping with previous studies of adolescent sexual behavior (Zimmer-Gembeck & Helfand, 2008). Reported age at first intercourse younger than 10 was coded as missing because of low prevalence rates and the possibility of nonconsensual intercourse.
**Psychosocial health outcomes.** Participants who reported ever smoking and who smoked their first cigarette before age 25 were considered to have engaged in adolescent/young-adult cigarette use. Participants who reported ever using marijuana or hash and who reported such use before age 25 were considered to have engaged in adolescent/young-adult cannabis use.

History of alcohol abuse, alcohol dependence, and major depressive episode were measured using a questionnaire that was based on the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision* (DSM-IV-TR) criteria (American Psychiatric Association, 2000) and the Structured Clinical Interview for DSM-IV-TR (First, Spitzer, Gibbon, & Williams, 2002). Because of the low prevalence rates of alcohol abuse and alcohol dependence, individuals who met diagnostic criteria for alcohol abuse or alcohol dependence (diagnoses were mutually exclusive) were combined into one group representing individuals who had experienced clinically significant problems with alcohol. In keeping with DSM-IV-TR criteria, the symptoms of a major depressive episode had to occur within the same 2-week period, and individuals were asked to report their age during the 12-month period in question. Participants meeting criteria for alcohol abuse or dependence by age 25 were considered to have experienced adolescent/young-adult alcohol abuse/dependence.

In keeping with DSM-IV-TR criteria, the symptoms of a major depressive episode had to occur within the same 2-week period, and individuals were asked to report their age the first time they experienced a 2-week period of depressed mood or anhedonia in conjunction with other symptoms. Individuals meeting diagnostic criteria for a major depressive episode before the age of 25 were considered to have experienced an adolescent/young-adult major depressive episode.

Participants also responded to 11 items measuring their level of current depressive symptoms using the abbreviated Iowa form (Carpenter et al., 1998) of the Center for Epidemiologic Studies Depression (CESD) scale (Radloff, 1977). Participants indicated how often they had experienced any of 11 symptoms in the past week using a 4-point scale (0 = never or almost never, 1 = seldom, 2 = often, 3 = always or almost always). Responses across all items were summed, and participants with current symptom levels exceeding 8 points (24% of points possible, consistent with percentage thresholds used in the original CESD scale) were considered to have high current depressive symptoms.

Criminal history was obtained using the Swedish National Crime Register (Fazel & Grann, 2006). The crime register is maintained by the Swedish National Council for Crime Prevention and documents all registered violent, nonviolent, and drug-related convictions in lower courts that occurred in Sweden from 1973 to 2004, including date of conviction and type of offense, for individuals aged 15 years (i.e., the age of criminal liability in Sweden) and older. Age at first offense was calculated using the participant’s birthdate and the earliest date of criminal conviction reported in the crime register. Participants convicted of any criminal offense before age 25 were considered to have engaged in adolescent/young-adult criminal offending.

For female participants, childbirth history was obtained using the Swedish Medical Birth Registry. This registry is maintained by the National Board of Health and Welfare and includes information on more than 99% of all births occurring in Sweden since 1973, including maternal age at childbirth. Maternal age at first childbirth was calculated, and any participants giving birth before age 20 were categorized as having experienced adolescent childbirth.

Participants reporting outcome onset before age 16 were excluded (i.e., deleted listwise) in models for that outcome to ensure that early sex preceded outcome onset (see Table 1).

### Statistical Analyses

First, a series of logistic regression models was run for each outcome using Mplus version 6.1 software (Muthén & Muthén,

### Table 1

**Descriptive Statistics for a Population-Based Cohort of Adult Swedish Twins**

<table>
<thead>
<tr>
<th>Measure</th>
<th>M (SD), range</th>
<th>Prevalence, %</th>
<th>Total n providing data</th>
<th>Percentage missinga</th>
<th>n with outcome onset &lt; 16b</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>—</td>
<td>60.4</td>
<td>12,126</td>
<td>0.0</td>
<td>—</td>
</tr>
<tr>
<td>Age at assessment</td>
<td>32.6 (7.6), 19–47</td>
<td>—</td>
<td>12,126</td>
<td>0.0</td>
<td>—</td>
</tr>
<tr>
<td><strong>Sexual behavior</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age at first sexual intercourse</td>
<td>17.5 (3.1), 10–46</td>
<td>—</td>
<td>8,877</td>
<td>26.8</td>
<td>—</td>
</tr>
<tr>
<td>Ever had intercourse</td>
<td>—</td>
<td>95.1</td>
<td>9,480</td>
<td>21.8</td>
<td>—</td>
</tr>
<tr>
<td>First intercourse in early adolescence (&lt;16)</td>
<td>—</td>
<td>24.0</td>
<td>9,338</td>
<td>23.0</td>
<td>—</td>
</tr>
<tr>
<td>First intercourse in later adolescence (16–19)</td>
<td>—</td>
<td>53.4</td>
<td>9,338</td>
<td>23.0</td>
<td>—</td>
</tr>
<tr>
<td><strong>Outcomes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cigarette use by age 25</td>
<td>—</td>
<td>54.8</td>
<td>8,549</td>
<td>29.5</td>
<td>2,975</td>
</tr>
<tr>
<td>Cannabis use by age 25</td>
<td>—</td>
<td>14.0</td>
<td>11,654</td>
<td>3.9</td>
<td>243</td>
</tr>
<tr>
<td>Alcohol abuse/dependence by age 25</td>
<td>—</td>
<td>3.9</td>
<td>11,263</td>
<td>7.1</td>
<td>26</td>
</tr>
<tr>
<td>Major depressive episode by age 25</td>
<td>—</td>
<td>9.0</td>
<td>10,584</td>
<td>12.7</td>
<td>303</td>
</tr>
<tr>
<td>High current depressive symptoms</td>
<td>—</td>
<td>32.3</td>
<td>11,135</td>
<td>8.2</td>
<td>314</td>
</tr>
<tr>
<td>Criminal offending by age 25</td>
<td>—</td>
<td>9.9</td>
<td>12,102</td>
<td>0.2</td>
<td>158</td>
</tr>
<tr>
<td>Childbearing by age 20 (females only)</td>
<td>—</td>
<td>2.7</td>
<td>7,322</td>
<td>0.0</td>
<td>4</td>
</tr>
</tbody>
</table>

*Note. n = number of individuals.*

a Missing data did not systematically vary by age or gender. b Number of individuals with data available for each outcome but with a reported age of onset younger than 16 years. These individuals were excluded from further analyses for that outcome.
2010). We began by estimating the magnitude of the association between early sex and the outcome across the overall sample while controlling for individual gender and age (but without considering the co-twin’s status). This initial model accounted for the clustered nature of the data using robust standard errors.

**Discordant-twin analyses.** Next, we compared twins discordant for early sex. We first ran models including all twin pairs ignoring zygosity and then separately by zygosity. We used a two-level logistic regression model composed of a within-pair effect and a between-pair effect to account for the clustered nature of the data (i.e., individuals nested within twin pairs) (Snijders & Bosker, 1999). We calculated a mean early sex score for each twin pair (0.0 = concordant for no early sex, 0.5 = discordant, 1.0 = concordant for early sex) and calculated a deviation score for each individual twin from their pair-mean score. A deviation score greater than 0 indicated a twin who engaged in early sex but whose co-twin did not. The within-pair effect corresponded to the regression of the outcome on the participant’s deviation score with the intercept treated as a random effect varying across twin pairs (i.e., individuals were nested within twin pairs with varying levels of mean early sex exposure). The slope was treated as a fixed effect because twin pairs do not have enough cluster members to allow the slopes to vary across clusters (Kenny, Kashy, & Cook, 2006). Fitting the within-pair parameter is equivalent to fitting econometric fixed-effects models (Neuhaus & McCulloch, 2006), and the estimate for the within-pair parameter corresponds to the change in outcome risk predicted by early sex relative to no early sex, controlling for familial characteristics shared within a twin pair (Enders & Tofghi, 2007). For this reason, the within-pair effect is the parameter of interest in these models (Begg & Parides, 2003). Gender and age were also included as covariates in these models.

We also calculated a zygosity × deviation score interaction term to test whether the within-twin effect of early sex differed between discordant DZ and MZ pairs (coded as DZ = 1 and MZ = 0). The interaction term was added as a within-pair parameter with all twins included in this model. A nonsignificant interaction term would indicate no difference in outcome risk in DZ versus MZ pairs. Discordant DZ and MZ twins at equivalent risk would suggest confounding due to shared environmental influences because both twins in discordant DZ and MZ pairs were exposed to these shared environmental factors. A significant interaction parameter would indicate a significant difference in the within-pair estimates for MZ versus DZ twins. A significantly smaller effect in MZ twins than in DZ twins would indicate genetic confounding of the association between early sex and the outcome because MZ pairs share a greater amount of additive genetic influences (100% vs. an average of 50% for DZ pairs).

To be included in the models described above comparing unrelated individuals and discordant twins, a participant had to have provided data on the outcome in question and belong to a twin pair in which both twins reported on early sex. (For $r$ values, see Table 2 footnote.)

**Bivariate twin model.** For those outcomes for which (a) there was a significant unadjusted association between early sex and the outcome and (b) discordant-twin analyses suggested familial confounding of that association, bivariate twin modeling was used to obtain more precise estimates of the degree to which these confounds were genetic versus environmental in nature.

### Table 2
**Estimated Effects of Early Sexual Intercourse on Adverse Psychosocial Health Outcomes From Analyses of Unrelated Individuals and Discordant Twin Pairs**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Effect of early intercourse$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unrelated individuals</td>
</tr>
<tr>
<td>Cigarette use</td>
<td><strong>OR</strong> [95% CI]</td>
</tr>
<tr>
<td>$(r_{MZ} = .63, r_{DZ} = .28)$</td>
<td>1.00 [0.78, 1.29]</td>
</tr>
<tr>
<td>Cannabis use</td>
<td>1.76$^*$ [1.49, 2.09]</td>
</tr>
<tr>
<td>$(r_{MZ} = .69, r_{DZ} = .41)$</td>
<td>—</td>
</tr>
<tr>
<td>Alcohol abuse/dependence</td>
<td>1.78$^*$ [1.33, 2.38]</td>
</tr>
<tr>
<td>$(r_{MZ} = .57, r_{DZ} = .22)$</td>
<td>—</td>
</tr>
<tr>
<td>Major depressive episode</td>
<td>1.04 [0.83, 1.31]</td>
</tr>
<tr>
<td>$(r_{MZ} = .44, r_{DZ} = .36)$</td>
<td>—</td>
</tr>
<tr>
<td>High current depressive symptoms</td>
<td>1.22$^*$ [1.07, 1.39]</td>
</tr>
<tr>
<td>$(r_{MZ} = .40, r_{DZ} = .16)$</td>
<td>—</td>
</tr>
<tr>
<td>Criminal offending</td>
<td>2.04$^*$ [1.67, 2.48]</td>
</tr>
<tr>
<td>$(r_{MZ} = .61, r_{DZ} = .44)$</td>
<td>—</td>
</tr>
<tr>
<td>Adolescent childbearing</td>
<td>4.11$^*$ [2.67, 6.36]</td>
</tr>
<tr>
<td>$(r_{MZ} = .58, r_{DZ} = .48)$</td>
<td>—</td>
</tr>
</tbody>
</table>

*Note.* $B$, unstandardized logit coefficient. Gender and age are included as covariates in all models (effects not shown). To be included in models for each outcome, participants had to provide data on outcome exposure and have an early sex pair-mean score available (cigarette use: available $n = 2,092$; cannabis use: $n = 7,215$; alcohol abuse/dependence: $n = 7,176$; major depressive episode: $n = 6,574$; high current depressive symptoms: $n = 6,903$; criminal offending: $n = 7,490$; adolescent childbearing: $n = 3,654$). $^a$Early intercourse (< age 16) vs. reference category of later intercourse onset (age 16+) used in analyses for all outcome variables except for adolescent childbearing. For models predicting adolescent childbearing (by age 20), the reference category was restricted to intercourse onset at ages 16–19. $^p < .05$. 

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These structural twin models provided estimates of variance due to additive genetic influences (A), common environmental influences shared by twins (C), or nonshared environmental influences unique to each participant (E). We used a “common-and-specific-factors” model (Figure 1; Loehlin, 1996; Young-Wolff, Kendler, Ericson, & Prescott, 2011), allowing us to estimate A, C, and E contributing (a) specifically to early sex (latent factors Anet; Cnet; and Enet), (b) specifically to the outcome (Anet; Cnet; and Enet), and (c) to the covariance between early sex and the outcome (Acov; Ccov; and Ecov). Structural equation modeling was completed in Mplus version 6.1 software (Muthén & Muthén, 2010) using methods recommended by Prescott (2004) for bivariate twin modeling with categorical variables. For each outcome, a twin pair had to have at least one available data point (i.e., information on early sex for either twin or outcome for either twin) to be included in the bivariate models for that outcome. As many as 6,063 twin pairs (the full sample) could be included in each bivariate model. Those missing data on all variables were excluded from models for that outcome (cannabis, excluded missing data on all variables were excluded from models for that outcome. As many as 6,063 twin pairs (the full sample) could be included in each bivariate model. Those missing data on all variables were excluded from models for that outcome (cannabis, excluded missing data on all variables were excluded from models for that outcome. As many as 6,063 twin pairs (the full sample) could be included in each bivariate model. Those missing data on all variables were excluded from models for that outcome (cannabis, excluded missing data on all variables were excluded from models for that outcome. As many as 6,063 twin pairs (the full sample) could be included in each bivariate model. Those missing data on all variables were excluded from models for that outcome (cannabis, excluded missing data on all variables were excluded from models for that outcome. As many as 6,063 twin pairs (the full sample) could be included in each bivariate model. Those missing data on all variables were excluded from models for that outcome (cannabis, excluded missing data on all variables were excluded from models for that outcome. As many as 6,063 twin pairs (the full sample) could be included in each bivariate model. Those missing data on all variables were excluded from models for that outcome (cannabis, excluded 13; high current depressive symptoms, 30; criminal offending, 6; adolescent childbearing, 299).

**Results**

Descriptive statistics for all measured variables are shown in Table 1. Early sex was reported by 24% of individuals, consistent with data from other Swedish samples (Danielsson et al., 2001). Seven-hundred eighty MZ pairs (17%) and 746 DZ pairs (25%) were discordant for early sex. The within-pair tetrachoric correlation for early sex was higher in MZ pairs ($r = .78$) than in DZ pairs ($r = .53$). Within-pair correlations for each measured outcome are included in Table 2 and indicate significant within-pair similarity for each outcome. MZ correlations were stronger than DZ correlations for all outcomes, suggesting that genetic factors may influence variability in these behaviors.

In Table 2, the column labeled “Unrelated Individuals” shows the estimated effects of early sex on each outcome among all individuals in the sample. As described previously, these effects are comparable to effects estimated in traditional studies comparing unrelated individuals to one another. Early sex was associated with significantly higher risk of all outcomes, except for cigarette smoking and major depressive episode.

**Discordant-Twin Analyses**

For each outcome significantly associated with early sex, we then conducted discordant-twin analyses to estimate the effects of early sex on that outcome after controlling for potential familial confounds. (Discordant-twin analyses were not conducted for cigarette use or major depressive episode because of a lack of association in the initial model.) The remaining columns of Table 2 show the estimated within-pair effects of early sex among discordant-twin pairs, within-pair effects separated by zygosity, and estimates of the zygosity × deviation score interaction. The odds ratios (OR) corresponding to the association of early sex with each outcome from comparisons of unrelated individuals and the within-twin effect of early sex on each outcome among discordant DZ and MZ twin pairs are also displayed graphically in Figure 2.

For almost every measured outcome, the within-twin effect of early sex was attenuated relative to the effect found among unrelated individuals and was no longer significant. This was true when examining all discordant twins and when separately examining each zygosity type. These results suggest that a twin who engaged in early sex and their co-twin who did not engage in early sex did not differ significantly with regard to subsequent cannabis use, alcohol abuse/dependence, high current depressive symptoms, or criminal offending.

The one exception was that females who engaged in early sex were more than 4 times as likely to report giving birth by the age of 20 when compared with females who had sex later in adolescence. When examining all discordant twins, affected twins were significantly more likely to report adolescent childbearing than their unaffected co-twins, OR = 2.27, 95% 95% confidence interval (CI) [1.06, 4.87]. Because the effect was significant when comparing all discordant twins ($n = 3,654$) but not when comparing twins separately by zygosity ($n_{DZ} = 1.418$; $n_{MZ} = 2.236$), we may lack power to detect significant effects given the low base rate of adolescent childbearing (2.7%) plus the low prevalence of female twins discordant for adolescent childbearing ($n = 166$, or 4.5%).

For nearly all measured outcomes, there was not a significant interaction of zygosity × exposure (see Table 2, far-right column), suggesting that shared environmental influences explain part of the association between early sex and each of these outcomes. The one exception was for high current depressive symptoms—the within-twin effect of early sex on high current depressive symptoms.
symptoms was significantly higher among DZ pairs than among MZ pairs ($B = 0.51$, $SE = 0.26$), suggesting that genetic influences may explain the association between early sex and current depressive symptoms.

**Bivariate Twin Models**

Because the results from the discordant-twin analyses suggested that familial confounding may contribute to the association between early sex and cannabis use, alcohol abuse/dependence, high current depressive symptoms, criminal offending, and adolescent childbearing, we used a common-and-specific–factors twin model to estimate the degree to which the overlap between early sex and each of these outcomes was due to A, C, and E. Figure 3 presents the standardized path estimates for A, C, and E, representing influences on the covariation between early sex and the outcome as well as influences specific to early sex and to the outcome. The relative contribution of A ($r_A$), C ($r_C$), and E ($r_E$) to the total correlation ($r_{cov}$) between early sex and each outcome are also shown in Figure 3. Because there was no association between early sex and subsequent cigarette use or major depressive episode in the

![Figure 2](image-url). Odds ratios for experiencing outcome based on differential engagement in early intercourse, by degree of relatedness. A logarithmic scale was used on the y-axis to illustrate the relative magnitude of effect of odd ratios less/greater than 1.0. Significant effects ($p < .05$) are denoted by *.

![Figure 3](image-url). Standardized path estimates ($\beta$) from bivariate twin models estimating the covariation between early intercourse and each outcome: A, additive genetic influence; C, shared environmental influence; and E, nonshared environmental influence. Relative contributions of A, C, and E to overlap ($r_{cov}$) between early sex and outcome represented by $r_A$, $r_C$, and $r_E$. Significant effects ($p < .05$) are denoted by *. 

---

**Table 1**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Early sex (specific)</th>
<th>Outcome (specific)</th>
<th>Early sex and outcome (common)</th>
<th>$r_{cov}$</th>
<th>$r_A$, $r_C$, or $r_E$</th>
<th>Proportion of $r_{cov}$ due to all $r_A$, $r_C$, or $r_E$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cannabis use</td>
<td>A 0.70* C 0.39* E 0.45*</td>
<td>0.74* 0.17 0.52*</td>
<td>0.06 0.36* 0.16</td>
<td>0.16*</td>
<td>0.00 0.13 0.03</td>
<td></td>
</tr>
<tr>
<td>Alcohol abuse &amp; dependence</td>
<td>A 0.67* C 0.48* E 0.44*</td>
<td>0.69* 0.18 0.60*</td>
<td>0.22 0.22 0.19</td>
<td>0.14*</td>
<td>0.05 0.05 0.04</td>
<td></td>
</tr>
<tr>
<td>High current depressive symptoms</td>
<td>A 0.64* C 0.53* E 0.47*</td>
<td>0.50* 0.20 0.79*</td>
<td>0.29* 0.00 0.00</td>
<td>0.08*</td>
<td>0.00 0.00 0.00</td>
<td></td>
</tr>
<tr>
<td>Criminal offending</td>
<td>A 0.68* C 0.41* E 0.43*</td>
<td>0.48* 0.50* 0.58*</td>
<td>0.21 0.33* 0.20*</td>
<td>0.19*</td>
<td>0.04 0.11 0.04</td>
<td></td>
</tr>
<tr>
<td>Adolescent childbearing</td>
<td>A 0.58* C 0.24* E 0.40*</td>
<td>0.26 0.45* 0.53*</td>
<td>0.41* 0.47* 0.26*</td>
<td>0.45*</td>
<td>0.17 0.22 0.07</td>
<td></td>
</tr>
</tbody>
</table>
initial models, and these outcomes were not included in the discordant-twin analyses, bivariate twin models were not conducted for either of these outcomes.

There was significant, albeit small to moderate, covariation between early sex and each outcome (see column corresponding to $r_{cov}$ in Figure 3). The highest covariation was found between early sex and adolescent childbearing ($r_{cov} = 0.45$).

Shared environmental influences significantly contributed to the covariation between early sex and cannabis use, accounting for 0.13 (81%) of the total correlation between early sex and cannabis use ($r_{cov} = 0.16$) and suggesting that environmental influences shared within families primarily explain the observed association between engaging in early sex and subsequent cannabis use by young adulthood.

Although there was significant covariation ($r_{cov} = 0.14$) between early sex and alcohol abuse/dependence, path estimates for genetic, shared environmental, and nonshared environmental covariance were all nonsignificant, suggesting that familial confounding may contribute to the association between early sex and subsequent alcohol abuse/dependence, but we may have lacked the power to partition the source of confounding into specific sources.

Genetic influences significantly contributed to the covariation between early sex and high current depressive symptoms, accounting for 100% of the total correlation between early sex and high current symptoms ($r_{cov} = 0.08$) and suggesting that common genetic influences account for the observed association between engaging in early sex and endorsing a high number of depressive symptoms in adulthood.

Shared and nonshared environmental influences significantly contributed to the covariation between early sex and criminal offending, accounting for 57 and 21%, respectively, of the total covariation between early sex and criminal offending ($r_{cov} = 0.19$). These results suggest that environmental influences shared within families account for most of the observed association between early sex and subsequent criminal offending. However, nonshared environmental factors, or environmental influences not shared by both twins, contributing to early sex and criminal offending also play a role in this observed association.

Genetic and environmental familial influences, as well as nonshared environmental influences, significantly contributed to the covariation between early sex and adolescent childbearing. Genetic, shared environmental, and nonshared environmental influences accounted for 37, 49, and 14% of the total covariation between early sex and adolescent childbearing ($r_{cov} = 0.45$), suggesting that influences shared within a family account for a large portion (86%) of the observed association between early sex and childbearing in adolescence. However, nonshared environmental factors that contribute to early sex and adolescent childbearing may also play a role.

**Sensitivity analyses.** In the analyses presented here, participants who reported having experienced an outcome by the age of 25 were considered to have experienced that outcome by young adulthood. However, some participants (19.5%) had not reached age 25 by the time of assessment but were included in the analyses presented here. These participants had not lived through the entire risk period for experiencing an outcome, but it is possible that they could still experience the outcome after the time of assessment but before reaching age 25. To address the possibility that including these individuals could bias results, discordant-twin analyses and bivariate twin models for each outcome were run excluding all individuals who had not yet reached the age of 25. Similar results were found for all outcomes (results not shown).

The discordant-twin analyses presented here, with the exception of adolescent childbearing, compared individuals reporting first intercourse in early adolescence (before age 16) to individuals delaying intercourse onset until age 16 or later. Analyses comparing individuals reporting early sex to individuals reporting intercourse onset restricted to later in adolescence (ages 16–19) resulted in a similar pattern of results for all outcomes (not shown).

Given the retrospective nature of the measured outcome data and the potential for recall bias or inaccurate reporting of age at onset for psychiatric disorders (Simon & Von Korff, 1995), we also ran additional analyses to explore whether potential recall bias or onset inaccuracies could affect our reported results. We first ran discordant-twin analyses for each of the psychiatric outcomes (i.e., a major depressive episode and each substance-related outcome) without restricting outcome exposure to before the age of 25. In other words, any individual in the sample reporting that they experienced the outcome at any point in their lifetime before assessment was included, regardless of reported age at onset, to reduce potential bias introduced by constructing the outcome measure using a potentially inaccurate reported age of onset. Again, similar results were found for all outcomes (not shown). Additionally, we tested whether the age of the participant at assessment (in effect, the amount of time elapsed since early sex or the outcome occurred) affected the association between early sex and the outcome by adding an interaction term (early sex × age) to models estimating the association between early sex and outcome among unrelated individuals. This interaction term was nonsignificant in models for every outcome (results not shown).

**Discussion**

Using a population-based registry of 20- to 47-year-old Swedish twins, we explored whether familial confounding accounted for the associations between early sex and several measures of psychosocial health, including substance use, depression, criminal activity, and adolescent childbearing. This approach has rarely been applied in studies examining consequences of early sex despite numerous calls for studies testing alternative explanations (Harden et al., 2008; Huibregts et al., 2011; Sandfort et al., 2008; Udell, Sandfort, Reitz, Bos, & Dekovic, 2010).

Compared with unrelated individuals with later intercourse onset, STAGE respondents who reported voluntary sexual intercourse before age 16 were more likely to use cannabis, experience alcohol abuse or dependence, and to be convicted of a criminal offense by the age of 25. They were also more likely to endorse high levels of depressive symptoms as adults and, among females, to have children as adolescents. However, twins discordant for early sex did not differ significantly in their risk of experiencing any of these outcomes (with the exception of adolescent childbearing), and bivariate twin models suggested that familial confounds contributed to the covariation between early sex and these outcomes.

This study adds to an emerging line of research investigating the contribution of genetic and environmental confounds to consequences attributed to adolescent sexual behavior by using a powerful quasi-experimental approach (Lahey et al., 2009; McGue et
al., 2010). In combination with previous studies using different samples and varying measures of psychosocial health (Harden et al., 2008; Huibregtse et al., 2011), our results provide converging evidence that familial background factors play an important role in the association between early sex and psychosocial health outcomes. Sexual education programs aimed solely at delaying intercourse onset may be unlikely to greatly reduce an individual’s risk of “psychological harm” (Social Security Act, 1996) as measured in the study presented here. Instead, the associations between early sex and adverse psychosocial health outcomes may be the result of other risk factors common to both variables. These risk factors may include environmental influences, such as general adversity faced by the adolescent’s family (Kirby, 2003; Roche et al., 2005), including neighborhood safety or disadvantage, parenting style, and parents’ own psychological health and emotional stability (Brook, Brook, Rubenstone, Zhang, & Finch, 2010).

Although the associations of early sex with adolescent childbearing and criminal offending were predominantly explained by familial factors, the bivariate twin models also pointed toward the importance of nonshared environmental influences. Twins who engage in early sex could be at increased risk for adolescent childbearing because of environmental influences not shared with their twin; a twin who engages in early sex may also use contraceptives less effectively, have more sexual partners, or simply have increased opportunity for unintended pregnancy because of a greater number of years spent sexually active. A twin who engages in early sex may also spend more time with deviant peers, which could contribute to the association between early sex and later criminal behavior. However, variables such as contraceptive use, number of partners during adolescence, and peer deviance were not measured in STAGE. Future research should explore these variables as possible mediators of the association between early sex and these adverse outcomes.

Although the association between early sex and outcome risk was reduced in the comparison of discordant twins relative to the comparison of unrelated individuals, the ORs for several outcomes (e.g., cannabis use, alcohol abuse/dependence, and criminal offending) were not trivial, although they were nonsignificant. Because models comparing unrelated individuals and discordant twins for an outcome were run using the same sample of individuals, this should not be due to a reduction in power related to diminished sample size. Instead, this may be due to somewhat low prevalence rates for the outcome and a decreased ability to estimate outcome risk precisely when comparing discordant twins. However, in combination with results from the bivariate twin models, these results do suggest that there is significant familial confounding of the association between early sex and these outcomes.

Several other limitations of the study should also be considered. The STAGE survey relied upon retrospective reports of lifetime behaviors assessed during adulthood, and recall bias may have contributed to measurement error in the reported age at initiation. However, the prevalence of early sexual activity reported retrospectively in this sample is similar to rates of early sex reported prospectively in adolescent samples, suggesting minimal recall bias. The prevalence rates of psychiatric disorders occurring by age 25, on the basis of retrospective reports regarding onset, were also similar to rates reported in prospective samples of young adults (Kessler et al., 2005). Sensitivity analyses exploring the potential effects of recall error also resulted in findings similar to those in the original analyses. In future research, prospective reporting of mood, substance use, and sexual behavior would provide a more precise assessment of the link between these risk factors and sexual risk behavior. However, few population-based datasets meet these criteria while also allowing for the use of quasi-experimental designs.

The discordant-twin analyses could be completed only when both twins provided information for all relevant variables. Despite an overall response of approximately 60% among eligible participants, the basic demographic characteristics of STAGE participants were comparable to those of all individuals belonging to the same birth cohort in Sweden. This suggests that missing data in STAGE are likely missing at random and results may be generalizable to the broader Swedish population. However, our results may not be readily generalizable to U.S. adolescents because of national differences in attitudes toward adolescent sexuality (Guttmacher Institute, 2001). Relative to adolescents in the United States, Swedish adolescents have easier access to contraceptives and other reproductive health services and have higher rates of contraceptive use (Guttmacher Institute, 2001; Santelli, Sandfort, & Orr, 2009). Swedish adolescents are also provided with more comprehensive sexual education in combination with greater societal acceptance of adolescent sexuality. It is possible that early sex may be more strongly associated with adverse psychosocial health outcomes for U.S. adolescents given the less-supportive climate surrounding adolescent sexuality in the United States. However, it is worth noting that previous studies finding support for familial confounding have been conducted using data from U.S. samples (Harden et al., 2008; Huibregtse et al., 2011).

Low base rates of some outcomes included in the study presented here—particularly alcohol abuse/dependence and adolescent childbearing—may have limited our power to detect significant effects. In this case, the results of discordant-twin analyses would overstate the importance of familial confounds in the association between early sex and each outcome at the expense of possible causal influences. However, it is again noted that analyses comparing affected and unaffected individuals (treating all twins as individuals) and analyses comparing discordant twins (not accounting for zygosity) were conducted using data from the same number of individuals. Nevertheless, given this limitation, the results of our analyses should be interpreted cautiously, and a causal role of early sex on these measures of psychosocial adjustment cannot be definitively ruled out. On the other hand, the bivariate twin models do not rely solely on the analysis of discordant-twin pairs and did indicate that familial influences contributed substantially to the covariation between early sex and each outcome. Our results suggest that previous research not accounting for these unmeasured familial confounds may have overstated the relationship between early sex and subsequent health outcomes, and future research should consider such confounding factors.

Conclusions

The study presented here represents an underutilized but powerful approach to investigating the relationship between early sex and subsequent psychosocial health. Our results suggest that familial confounds may play an important role in increasing the likelihood of undesirable outcomes among individuals who are...
also at greater risk for engaging in sex during early adolescence. This means that preventive interventions designed to reduce these negative psychosocial health outcomes should not solely target delaying age at first intercourse if they hope to reduce the prevalence of these adverse outcomes. Future research incorporating genetically informative designs is needed to provide additional insight into the mechanisms connecting sexual and mental health, particularly among adolescents.

References


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