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Understanding the Link Between Early Sexual Initiation and Later Sexually Transmitted Infection: Test and Replication in Two Longitudinal Studies

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A B S T R A C T

Purpose: Age at sexual initiation is strongly associated with sexually transmitted infections (STI); yet, prevention programs aiming to delay sexual initiation have shown mixed results in reducing STI. This study tested three explanatory mechanisms for the relationship between early sexual debut and STI: number of sexual partners, individual characteristics, and environmental antecedents.

Methods: A test-and-replicate strategy was employed using two longitudinal studies: the Seattle Social Development Project (SSDP) and Raising Healthy Children (RHC). Childhood measures included pubertal age, behavioral disinhibition, and family, school, and peer influences. Alcohol use and age of sexual debut were measured during adolescence. Lifetime number of sexual partners and having sex under the influence were measured during young adulthood. Sexually transmitted infection diagnosis was self-reported at age 24. Early sex was defined as debut at <15 years. Path models were developed in SSDP evaluating relationships between measures, and were then tested in RHC.

Results: The relationship between early sex and STI was fully mediated by lifetime sex partners in SSDP, but only partially in RHC, after accounting for co-occurring factors. Behavioral disinhibition predicted early sex, early alcohol use, number of sexual partners, and sex under the influence, but had no direct effect on STI. Family management protected against early sex and early alcohol use, whereas antisocial peers exacerbated the risk.

Conclusions: Early sexual initiation, a key mediator of STI, is driven by antecedents that influence multiple risk behaviors. Targeting co-occurring individual and environmental factors may be more effective than discouraging early sexual debut and may concomitantly improve other risk behaviors.

IMPLICATIONS AND CONTRIBUTION

Early sexual initiation has been linked with sexual risk behavior and sexually transmitted infection (STI). In these analyses, behavioral disinhibition, family management, and antisocial peers influenced the relationship between early sex and STI. Addressing multiple early risk factors, rather than early sexual initiation, may more effectively reduce rates of STI.

Sexually transmitted infections (STI) are among the most commonly occurring infections in the United States. Approximately 20 million new cases occur every year, nearly half of which are among young adults aged 18–24 years [1]. Despite prevention efforts, there has been little reduction in rates of *Chlamydia trachomatis* and other common STI [2], which suggests that current prevention approaches are not sufficiently effective.

Early sexual initiation is one of the most robust predictors of STI among adolescents and young adults [3–6], making this an attractive target for prevention efforts. However, prevention programs promoting abstinence or delay of sexual activity among adolescents have had mixed results in reducing STI [7,8].

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Other prevention approaches, focused on decision making, proper condom use, and negotiation skills, have been shown to reduce sexual risk behavior [9], but have short-lived and moderate effects [10]. Given the strong links between early sexual debut and later STI, more robust and sustainable intervention targets may be identified by examining mechanisms for this relationship.

Three potential mechanisms for this link have shown promise. The first focuses exclusively on sexual behaviors and assumes a single causal pathway, from pubertal age to early sexual initiation, and subsequently to the number of lifetime sexual partners by young adulthood, assuming the effect of early sex is mediated by the number of sexual partners. Ample literature links pubertal timing, early sex, and number of sexual partners [4,11–15]. Studies have further linked number of sexual partners and STI [13,16,17]. James et al [14] used path modeling to show the causal chain between pubertal timing, age of initiation, and sexual risk behavior, but did not evaluate the effect on STI itself.

The second mechanism examines a role of behavioral disinhibition and alcohol use in exacerbating STI risk [17–19]. Adolescents who initiate intercourse early are more likely to use alcohol and to report alcohol problems compared with their peers who delay sex (e.g., [20]); conversely, adolescent alcohol use is associated with STI (for review, see [21]). A tendency toward behavioral disinhibition, indicated by impulsivity and sensation seeking, has commonly been theorized to explain the comorbidity in problem behaviors, such as the link between alcohol use, sexual risk behaviors, and STI [22]. This suggests a pathway from behavioral disinhibition to early sex and early alcohol use, followed by having sex under the influence, and subsequently, STI.

The third explanatory mechanism hypothesizes that early environmental antecedents common to early alcohol use and early sexual debut explain the increased risk for STI [23]. In the family domain, monitoring of child activities is an especially important factor in adolescent sexual risk taking and STI [24–28]. Peer delinquency and school bonding have also been found to predict risky sexual behavior and STI acquisition [26,29–31]. Because these same environmental factors have been linked to substance-related risk behaviors [32], they may account for the apparent relationship between early alcohol use and early sex, and also between early sex and STI.

Current study

The current study employed an innovative test-and-replicate strategy using two longitudinal datasets to test these hypothesized explanations of the early sexual debut–STI link. First, the three discrete mechanisms were tested in the Seattle Social Development Project (SSDP) longitudinal dataset. The first hypothesis posited that the cumulative exposure to multiple sexual partners, permitted by earlier sexual initiation, predicts STI risk. Thus, early sexual debut was hypothesized to be a marker for number of lifetime sexual partners, increasing risk for STI. Hypotheses 2 and 3 postulated that early sex and early alcohol use have common antecedents that explain their co-occurrence in adolescence. The same antecedents predict risky sexual practices (e.g., having sex under the influence) in young adulthood, and subsequent STI. Hypothesis 2 tested the effect of childhood behavioral disinhibition as a common individual-level antecedent, whereas Hypothesis 3 examined the effects of environmental antecedents in the family, peer, and school domains. Because Hypotheses 1, 2, and 3 are not mutually exclusive, a final step combined them into a single omnibus model using the SSDP dataset and tested the model's stability through replication in another longitudinal sample, the Raising Healthy Children (RHC) study.

Methods

Participants

The SSDP and RHC are two longitudinal studies of youth development. In 1985, the SSDP recruited 808 fifth graders (mean age, 10.70 years; standard deviation, .52 years) from 18 Seattle public schools, many of which served low-income households. Participant surveys used in this study were conducted annually from ages 10 to 16 years, with follow-up at ages 18, 21, and 24 years (collected in 1999). Interviews with parents were conducted annually when youth were aged 10–16 years. Data collection continues, and retention rates for the sample have remained high (>90%) since the age 14 interview in 1989.

The 1,040 participants of RHC were drawn from a suburban school district near Seattle. Participants were enrolled in first (younger cohort) or second grade (older cohort) in 1993 and 1994, and were then observed annually in the spring until 2011, when the younger cohort was 24 years old, and the older cohort was 25 years old. Additional interviews were conducted in fall 2004, 2005, and 2006 during the 2 years after high school. Parent interviews were conducted annually through age 18 years. Retention rates for the RHC sample have also remained high (≥85%) since study inception. Both studies were approved by the University of Washington Human Subjects Review Committee.

Measures

Lifetime sexually transmitted infection. Participants reported whether they were ever told by a doctor or nurse that they had an STI. In SSDP, the questionnaire included two items regarding diagnosis of human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) and "sexually transmitted disease (STD or VD [venereal disease], other than HIV/AIDS), such as gonorrhea, genital warts, chlamydia, trich, herpes, or syphilis" (ages 21–24 years). The RHC questionnaire included a question about HIV/AIDS and [12] additional items naming specific STI (ages 19–24 years). Only five participants reported HIV/AIDS diagnosis, so this was combined with other STI. Diagnosis was coded as 1 if a participant responded "yes" in any interviews and 0 if they responded "no" in all interviews (or had no sexual partners). The diagnosis of STI was modeled as a binary (categorical) variable in the analyses.

Young adult predictors (ages 18–24 years). Lifetime number of sexual partners was assessed at age 24 years in SSDP and 22–24 years in RHC (highest number across the assessments, capped at 20) and modeled as normally distributed. Sex under the influence assessed how often participants engaged in sexual intercourse after drinking alcohol or using drugs (1 = "never" to 5 = "every time"). Drinking alcohol before having sex more than half of the time and/or ever using illicit drugs before having sex was coded as 1 (otherwise coded as 0). The