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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

number of assessments from age 18 to 24 years during which participants reported having sex under the influence (i.e., chronicity) was used as a risk score and modeled as a categorical variable.

Adolescent predictors. Past-year sexual behavior was self-reported and collected prospectively. In addition, in SSDP, age at first sexual initiation was self-reported retrospectively at ages 18 and 24 years. Sexual initiation was coded as 1 (early) if participants reported age of debut earlier than 15, a definition used in previous studies [11], and 0 if sex was initiated at age 15 or later. Initiation of sexual activity before age 10 was coded as missing because of concerns about nonconsensual sexual activity. The variable was modeled as binary. Early alcohol use reflected the chronicity (number of years) of alcohol use between ages 10 and 14 years in SSDP and 10 and 15 years in RHC and modeled as a categorical risk index.

Childhood predictors (ages 10–14 years). A behavioral disinhibition measure (five items per wave in SSDP and three in RHC) assessed impulse-control problems and was modeled as a continuous variable. Examples include "How often do you do what feels good, regardless of the consequences?" and "How often do you do something dangerous because someone dared you to do it?" Items were scored on a 5-point scale (1 = "never" to 6 = "once a week or more") and summed for a single risk score. Family management was assessed in the parent interview and included items on parental monitoring, rules, discipline, and rewards (six in SSDP and five in RHC). Items were summed and modeled as continuous. Response options were 1 = "NO!"; 2 = "no"; 3 = "yes"; and 4 = "YES!" where greater values correspond to more monitoring of child activities. School bonding (eight items in SSDP and five in RHC) reflected liking school, classes, teachers, and school assignments, and was modeled as a continuous variable. Response options ranged from 1 = "NO!" to 4 = "YES!" with higher values reflecting greater school bonding. Participants' self-report of antisocial peers included five items per wave measuring friends being in trouble with teachers, police, school suspension/expulsion, or gang activities (close friends and other peers). Response options included "yes/no," count of antisocial peers, and a 4-point scale (1 = "NO!" to 4 = "YES!"). Because of the differently scaled items, variables were standardized at each age before averaging; higher values represented more antisocial peers. Age of pubertal onset was self-reported retrospectively (ages 18 and 24 years in SSDP and 17–18 years in RHC).

Demographics

Gender and ethnicity were self-reported. Ethnicity in SSDP was categorized as black, Asian, and Native American (reference: white). In RHC, the racial/ethnic categories are white (vs. nonwhite) because individual minority groups were too small to permit generalization. Eligibility for the National School Lunch/Breakfast program from school records was used as a proxy for childhood socioeconomic status. Parent age at birth of target child determined whether participants were born to teenage parents.

Statistical analyses

We first evaluated the relationship between STI and the potential predictors using probit regression to generate "baseline" beta coefficients (adjusted for demographics). We then tested three models of specific mechanisms by which early sexual initiation and STI diagnosis may be linked using path modeling [33]. Path modeling, which is a type of structural equation modeling, is a powerful methodology that examines how multiple predictors are related to STI as well as to each other, by estimating multiple simultaneous regressions between variables in the model. We chose this methodology over traditional regression analysis to explicitly test mediators, such as number of sexual partners, which may explain the relationships between early sex and STI. Path model coefficients are interpreted as standardized regression betas ranging from −1 to 1 and represent change in units of an outcome per 1 standard deviation change in the predictor. We also calculated the change in beta ($\Delta\beta$), defined as the "baseline" beta for a predictor minus the beta for that same predictor in a given path model, to determine how much of the effect was accounted for by the other variables in the model.

Models were estimated using Mplus 7 [34]. Hypotheses 1–3 were tested in the SSDP dataset; the omnibus model was first estimated in SSDP and then estimated and assessed for potential replication in RHC. Appendix 1 (which can be found in the online edition of this article) contains unstandardized and standardized coefficients and standard errors for the omnibus models. Bivariate correlations between all variables are presented in Appendix 2, which can be found in the online edition of this article. Each model was saturated, meaning that direct paths were estimated between each predictor and each outcome variable. Because both studies included an intervention in early childhood (for study designs, see [35,36]), intervention status was included as a covariate in all analyses. All models were adjusted for demographic variables (gender, ethnicity, being a child of a teenage parent, and childhood socioeconomic status) and cohort membership (in RHC) to control for differences in mean levels of the variables, such as gender differences in STI prevalence. Checks of model comparability showed no differences in structural paths by treatment condition, gender, or ethnicity, meaning that associations between variables did not vary by these groups and analyses stratified by these groups were not necessary.

Results

Both study samples were gender balanced, but participants in SSDP were racially and ethnically more diverse (47% Caucasian vs. 75% in RHC) (Table 1). Over half of participants (52%) in SSDP received free or reduced-price lunch in fifth to seventh grade, whereas this was true for 38% of RHC participants. Approximately one third (36.7%) of SSDP participants, but only 17.9% of RHC youth reported early sexual initiation (before age 15 years), but by ages 24 to 25 years, a fifth of each sample had self-reported an STI. In SSDP, the STI rate was 32.5% for early initiators of sex compared with 16.6% later initiators (Pearson χ^2, $p < .001$); rates in RHC were comparable (34.4% vs. 15.8%; $p < .001$).

In probit regression analyses, early sexual debut was associated with increased risk of STI, with "baseline" betas of .33 in SSDP and .37 in RHC (Table 2). In both samples, lifetime number of sex partners, behavioral disinhibition, early alcohol use, sex under the influence, and antisocial peers were also associated with increased risk of STI. Older pubertal age and higher levels of family management and school bonding were associated with decreased risk.

Table 1
Characteristics of study samples

Self-report measure	Seattle Social Development Project (N = 808), n (%) reporting >0 behaviors or mean (standard deviation)	Raising Healthy Children (N = 1,040), n (%) reporting >0 behaviors or mean (standard deviation)
Demographic variables		
Women	39 (49.0%)	492 (47.3%)
White	381 (47.2%)	783 (75.3%)
African American	207 (25.6%)	36 (3.5%)
Native American	43 (5.3%)	24 (2.3%)
Asian/Pacific Islander	177 (21.9%)	70 (6.7%)
Mixed		127 (12.2%)
Treatment condition	156 (19.3%)	562 (54.0%)
Received free lunch	423 (52.4%)	395 (38.0%)
Child of teen parent	127 (15.7%)	96 (9.2%)
Model variables		
Sexually transmitted infection	176 (21.8%)	199 (19.1%)
Sex under influence	446 (32.9%)	442 (42.5%)
Early sexual initiation	295 (36.5%)	186 (17.9%)
Early alcohol use[b]	2.04 (1.68)	1.31 (1.41%)
Lifetime sexual partners, n[a,b]	7.93 (6.61)	7.94 (6.58)
Pubertal age, years[b]	12.48 (1.63)	12.76 (1.45)
Behavioral disinhibition[b]	1.81 (.86)	2.90 (1.21)
Family management[b]	3.50 (.38)	3.31 (.42)
School bonding[b]	2.93 (.50)	3.24 (.36)
Antisocial peers[b]	.01 (.45)	.00 (.59)

[a] Number of lifetime sexual partners capped at 20. At age 24, 94% of all participants in the Seattle Social Development Project and Raising Healthy Children reported at least one sexual partner.
[b] Mean (standard deviation).

Figure 1A shows the model testing Hypothesis 1 using SSDP data. Results indicated that, consistent with expectations, earlier puberty predicted earlier sexual debut ($\beta = -.30$), which in turn predicted a higher number of partners ($\beta = .41$). The effect of early sexual debut on STI diagnosis ($\beta = .20$) remained, but was substantially attenuated from the "baseline" relationship in Table 2 ($\Delta\beta = .13$) by number of sexual partners, suggesting partial but not complete mediation.

Table 2
Baseline beta coefficients reflecting association of individual predictors of sexually transmitted infections, adjusted for demographics[a]

	Seattle Social Development Project β[b]	Raising Healthy Children β[b]
Pubertal age	−.10*	−.08+
Early sex	.33***	.37***
Lifetime number of sexual partners	.39***	.36***
Behavioral disinhibition	.14**	.23***
Early alcohol use	.20**	.20***
Sex under influence	.27***	.30***
Family management	−.17***	−.14**
School bonding	−.17**	−.07
Antisocial peers	.25***	.20***

[a] Analyses were adjusted for gender, ethnicity, socioeconomic status, treatment condition, being the child of a teen parent, and cohort (Raising Healthy Children only).
[b] Unexponentiated standardized beta coefficients from probit regression analyses.
* $p < .05$.
** $p < .01$.
*** $p < .001$.
+ $p < .10$.

The second hypothesis examined whether behavioral disinhibition drives risky behaviors such as early sex, early alcohol use, and sex under the influence, which then results in STI acquisition (Figure 1B). Although we expected overlap between early sex and early alcohol use (bivariate correlation, $r = .15$; $p < .01$) (Appendix 2), after including behavioral disinhibition in the model, the two were no longer significantly related. Behavioral disinhibition predicted greater likelihood of both early sexual debut ($\beta = .34$) and early alcohol use ($\beta = .22$), and was directly related to having more sex while under the influence ($\beta = .17$). However, behavioral disinhibition did not predict STI directly, nor did it completely explain the relationship between early sex or early alcohol use and STI, as demonstrated by the continued presence of direct links between these factors ($\beta_{\text{early sex}} = .26$; $\beta_{\text{alcohol use}} = .16$) and STI. Early sex predicted more sex under the influence ($\beta = .27$). Both early sex and early alcohol use were related to greater likelihood of STI, although the effect of both was somewhat attenuated from "baseline" effects ($\Delta\beta = .07$ and .04, respectively). Thus, results were somewhat consistent with Hypothesis 2 in showing that behavioral disinhibition increased risk-taking behavior; however, behavioral disinhibition did not appear to explain much of the link between early sexual initiation and STI.

The model for Hypothesis 3 (Figure 1C) showed that environmental antecedents had a role in increasing the likelihood of early sex and early alcohol use, but did not directly predict STI. Having antisocial peers had the strongest effect on increased risk of early sex ($\beta = .28$) and alcohol use ($\beta = .17$). Family management appeared to buffer engagement in early sex ($\beta = -.12$) and early alcohol ($\beta = -.11$), and school bonding protected against early alcohol use ($\beta = -.14$). Partially consistent with Hypothesis 3, having antisocial peers was linked with greater likelihood of having sex under the influence ($\beta = .15$). Notably, after accounting for environmental antecedents, early alcohol use no longer predicted STI. The relationship between early sexual debut and STI remained ($\beta = .21$), although it was moderately attenuated from the "baseline" relationship ($\Delta\beta = .12$).

A final model examining the relative contribution of all of the predictors was tested in SSDP and replicated in the RHC dataset (Figures 2 and 3). Overall, the omnibus models integrated and supported findings from Hypotheses 1, 2, and 3. The strongest predictor of STI diagnosis was the number of sexual partners, a finding that was replicated in both SSDP ($\beta = .27$) and RHC ($\beta = .21$) studies. Consistent with Hypothesis 1, the effect of early sex was completely mediated through number of sexual partners in SSDP, whereas in RHC, a significant yet attenuated effect remained ($\beta = .18$; $\Delta\beta = .19$). The effect of behavioral disinhibition on increased risky behavior in adolescence and in young adulthood tested by Hypothesis 2 was replicated in both models. Consistent with Hypothesis 2, the effect of behavioral disinhibition on STI was indirect through early sexual initiation, sex under the influence, and number of lifetime partners. The alcohol-specific pathway from early alcohol use to sex under the influence, and to STI was evident only in RHC. Finally, Hypothesis 3 regarding early environmental predictors' effects on adolescent risk was replicated in both samples. In addition, in SSDP, the presence of antisocial peers directly predicted STI diagnosis.

Discussion

The current study examined three mechanisms that may explain the link between early sexual initiation and STI: number

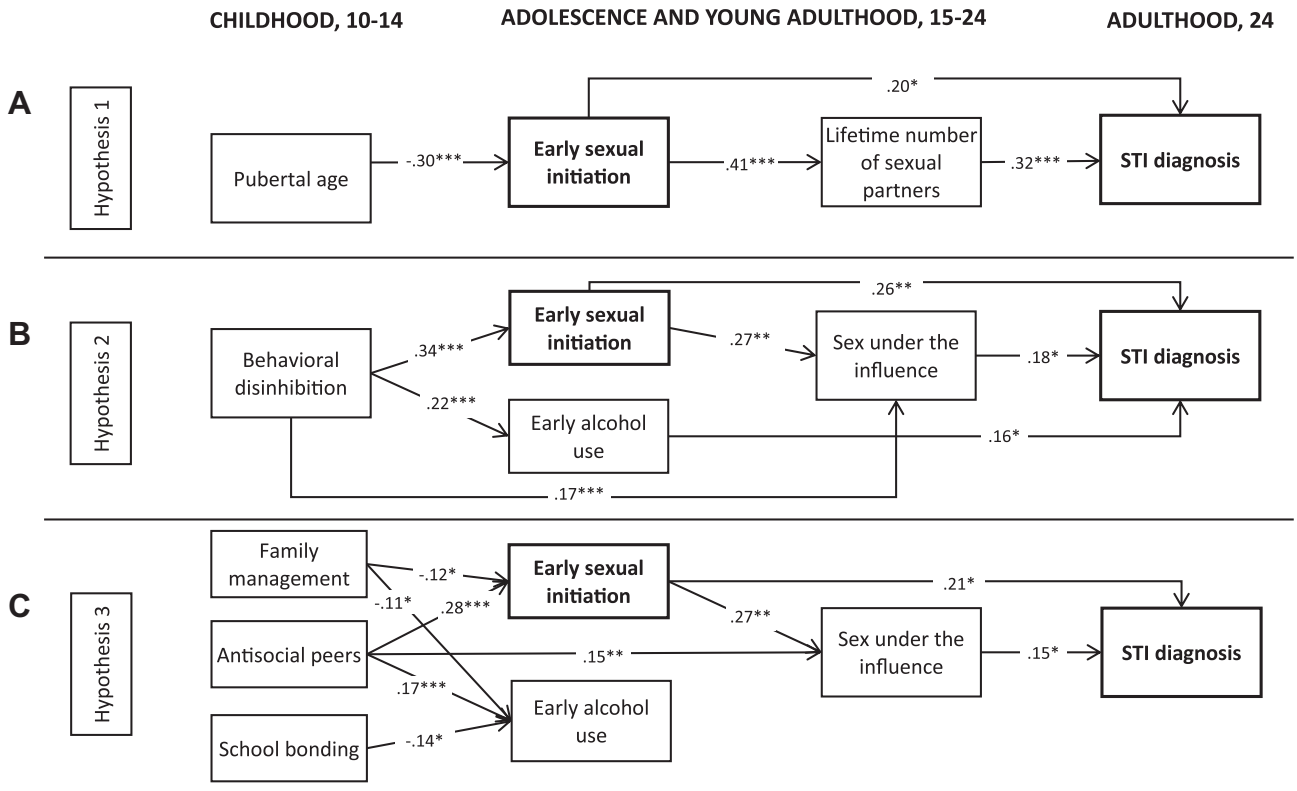

Figure 1. Models testing Hypotheses 1–3 in Seattle Social Development Project dataset. $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$. Coefficients in the models are partial standardized betas. All models are saturated such that all dependent variables are regressed onto all model predictors. Where lines are not drawn, the relationships are nonsignificant at $p < .05$. All variables controlled for gender, childhood socioeconomic status, ethnicity, and being the child of a teen parent. In (C), correlations between family management (X), antisocial peers (Y), and school bonding (Z) are as follows: XY = –.29***; YZ = –.22***; XZ = .36***. STI = sexually transmitted infection.

of sexual partners, behavioral disinhibition, and environmental antecedents. In accordance with the first mechanism, accounting for lifetime number of sexual partners completely explained the relationship between early sexual debut and STI in one dataset and significantly attenuated the relationship in the other. In the second mechanism, behavioral disinhibition, although not directly related to STI diagnosis, predicted all the other risk factors for STI, which suggests that it is a common driver of risk. Similarly, family, school, and peer environments were linked to risk behaviors, and (in SSDP) antisocial peers had a direct effect on STI diagnoses in addition to the effect on the risk behaviors.

Our observation that behavioral disinhibition appeared to drive sexual and alcohol-related risk behavior is consistent with prior findings showing that childhood behavioral disinhibition is related to adolescent risk behaviors, including those that are and are not sexual in nature [37,38]. The absence of a direct relationship between behavioral disinhibition and STI diagnosis suggests that individual risk traits operate through intervening behaviors, such as having sex with multiple partners or under the influence of drugs and alcohol. Behavioral disinhibition has been linked to a number of other risk behaviors (e.g., delinquency [22]), and interventions that successfully moderate the influence of this trait, if incorporated into STI prevention strategies, could have broad effects on risk behaviors in general, beyond risky sex. For example, some broad universal interventions have been shown to have STI effects, especially among higher risk youths [28].

Results also supported the hypothesis that environmental antecedents in family, peer, and school domains are related to early sex and other risk behaviors leading to STI acquisition. The consistent links between having antisocial peers in childhood and each of the risk behaviors, with paths following through to STI diagnoses in young adulthood in SSDP, is consistent with the observation of Henry and colleagues [39] that adolescent peer attitudes influenced sexual risk behavior in young adulthood. However, the pathways for family and school influences were not as consistently strong and not all links were observed in both studies. These effects may operate indirectly through behavioral disinhibition and antisocial peers. Altogether, findings suggest that factors earlier in life strongly influence STI risk, and interventions at younger ages may have long-term effects in preventing sexual risk behaviors and subsequent STI.

Over and above individual or environmental factors, lifetime number of sex partners remained the strongest and most consistent predictor of STI. However, given the complex influences on this behavior, prevention messages focusing solely on partner reduction are unlikely to be successful. Nevertheless, lifetime number of sex partners may be an excellent measure of the efficacy of interventions designed to mitigate the effect of behavioral disinhibition or antisocial peer influences on risky sexual practices.

The strengths of the current study include the longitudinal design and the ability to test mediators at different developmental stages. Testing the model for replication in another longitudinal study further strengthened the findings. Although the samples differed in ethnic and socioeconomic diversity and were

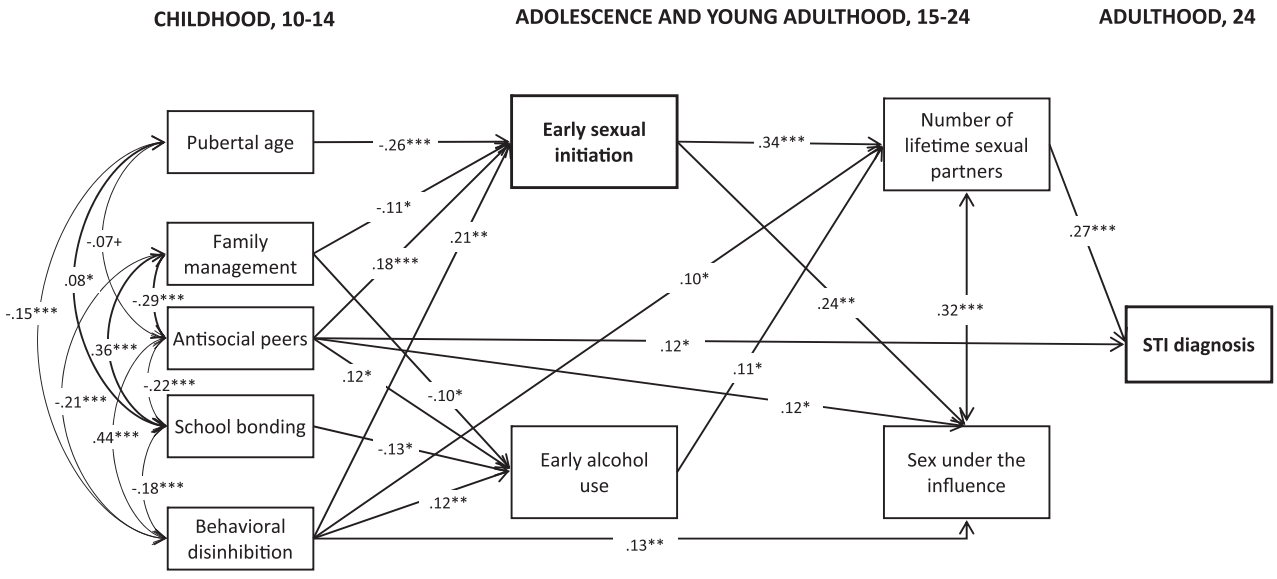

Figure 2. Primary omnibus model depicting childhood and adolescent predictors of STI infection at age 24 in Seattle Social Development Project. $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$; $^{+}p < .10$. Coefficients in the models are partial standardized betas. The model is saturated such that all dependent variables are regressed onto all model predictors. All variables controlled for gender, childhood socioeconomic status, ethnicity, and being the child of a teen parent. STI = sexually transmitted infection.

over a decade apart, the uniformity of results speaks to the stability of the findings. Nevertheless, there are also limitations. First, STI diagnosis was self-reported; respondents may have elected not to report diagnoses, and youth who were never tested may have had undetected STI. Second, we could not determine with precision the proximity of some risk factors to STI acquisition. Although this limits determination of temporal sequence for some risk factors (e.g., sex under the influence, factors measured after sexual debut), the temporal sequence between behavioral disinhibition and environmental influences in childhood is clear. Finally, the influences on sexual risk behavior and STI are numerous and our examination was not comprehensive. Future studies should examine other mediators, such as peer sexual behavior and family attitudes toward sex, among others, which are likely to affect sexual behavior and STI acquisition.

In conclusion, our results suggest that programs aimed at delaying sexual initiation through promoting abstinence until marriage may miss important intervention targets that can be addressed through a broader, social-developmental approach to

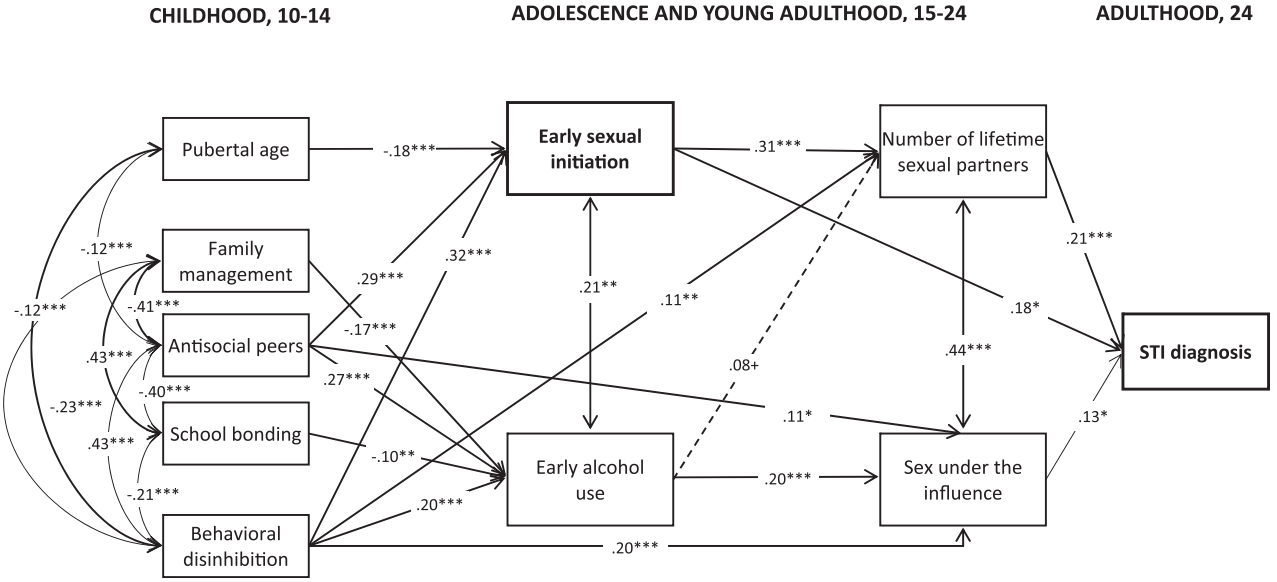

Figure 3. Replication model. Childhood and adolescent predictors of sexually transmitted infection (STI) at age 24 in Raising Healthy Children. $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$; $^{+}p < .10$. Coefficients in the models are partial standardized betas. The model is saturated such that all dependent variables are regressed onto all model predictors. Paths that are marginally significant at $p < .10$ are shown (as dashed lines) only when significant in Figure 2. All variables controlled for gender, childhood socioeconomic status, ethnicity, and being the child of a teen parent.

preventive intervention. For example, self-regulation training may decrease early sexual initiation, as may strengthening family management. Indeed, prevention programs that have been most successful at reducing risky sexual practices have taken multi-pronged approaches, focusing on contraception and sexually transmitted disease education, and also addressed other risk behaviors, such as substance use [40]. The current study shows that the sexual risk behaviors that lead to STI are affected by multiple childhood and adolescent processes. Prevention programs must address the larger social-developmental risk and protective factors in addition to the proximal causes to successfully reduce sexual risk behavior and STI.

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Appendix 1

Unstandardized and standardized parameters for Model 4: Seattle Social Development Project (SSDP) and Raising Healthy Children (RHC) samples

Parameter	SSDP sample			RHC sample		
	Unstandardized estimate	Standard error	Standardized estimate	Unstandardized estimate	Standard error	Standardized estimate
Structural paths						
Pubertal age → early sex	−.21	.04	−.26***	−.16	.04	−.18***
Behavioral disinhibition → early sex	.33	.10	.21**	.33	.05	.32***
Family management → early sex	−.41	.19	−.11*	−.14	.14	−.05
School bonding → early sex	.11	.16	.04	−.08	.16	−.02
Antisocial peers → early sex	.52	.16	.18***	.62	.11	.29***
Child of teen parent → early sex	.15	.15	.04	.30	.18	.07****
Male → early sex	.68	.14	.25***	−.48	.12	−.19***
White → early sex				−.04	.12	−.01
Black → early sex	.27	.44	.09			
Asian → early sex	−.56	.55	−.17***			
Native → early sex	.48	.78	.08			
Free lunch → early sex	.19	.19	.07	.36	.11	.14**
Older cohort → early sex				−.36	.11	−.14**
Treatment → early sex	−.45	.18	−.16**	−.02	.11	−.01
Pubertal age → early alcohol use	−.03	.03	−.05	−.03	.02	−.04
Behavioral disinhibition → early alcohol use	.16	.05	.12**	.20	.04	.20***
Family management → early alcohol use	−.33	.16	−.10*	−.50	.10	−.17***
School bonding → early alcohol use	−.30	.14	−.13*	−.34	.12	−.10**
Antisocial peers → early alcohol use	.31	.13	.12*	.56	.08	.27***
Child of teen parent → early alcohol use	−.13	.13	−.04	−.25	.13	−.06*
Male → early alcohol use	−.10	.10	−.04	−.16	.08	−.07*
White → early alcohol use				−.05	.09	−.02
Black → early alcohol use	−.44	.75	−.17			
Asian → early alcohol use	−.75	.85	−.27			
Native → early alcohol use	−.26	3.26	−.05			
Free lunch → early alcohol use	−.28	.35	−.12	.19	.08	.08*
Older cohort → early alcohol use				−.20	.07	−.08**
Treatment → early alcohol use	.04	.15	.02	.04	.08	.02
Early sex → lifetime sex partners	1.65	.34	.34***	1.62	.33	.31***
Early alcohol use → lifetime sex partners	.65	.29	.11*	.41	.25	.08****
Pubertal age → lifetime sex partners	−.04	.17	−.01	−.10	.15	−.02
Behavioral disinhibition → lifetime sex partners	.76	.33	.10*	.61	.22	.11**
Family management → lifetime sex partners	−.16	.77	.01	.28	.56	.02
School bonding → lifetime sex partners	−.34	.57	−.03	.29	.64	.02
Antisocial peers → lifetime sex partners	.62	.66	.04	.67	.46	.06
Child of teen parent → lifetime sex partners	.69	.63	.04	1.69	.74	.07*
Male → lifetime sex partners	1.47	.55	.11**	.12	.46	.01
White → lifetime sex partners				−.46	.45	−.03
Black → lifetime sex partners	−.52	1.18	−.04			
Asian → lifetime sex partners	−.77	1.36	−.05			
Native → lifetime sex partners	−.42	3.78	−.01			
Free lunch → lifetime sex partners	−.43	.66	−.03	.31	.42	.02
Older cohort → lifetime sex partners				−.99	.44	−.08*
Treatment → lifetime sex partners	.59	.82	.04	.03	.41	.00
Early sex → sex under the influence	.21	.09	.24**	.05	.07	.06
Early alcohol use → sex under the influence	.01	.08	.01	.19	.05	.20***
Pubertal age → sex under the influence	.00	.04	.00	−.02	.03	−.03
Behavioral disinhibition → sex under the influence	.18	.07	.13**	.18	.04	.20***
Family management → sex under the influence	−.19	.15	−.05	.08	.10	.03
School bonding → sex under the influence	.10	.13	.04	−.06	.13	−.02
Antisocial peers → sex under the influence	.30	.14	.12*	.21	.09	.11*
Child of teen parent → sex under the influence	−.04	.14	−.01	.19	.14	.05
Male → sex under the influence	.39	.13	.16**	−.14	.09	−.06
White → sex under the influence				.08	.09	.03
Black → sex under the influence	−.11	.57	−.04			
Asian → sex under the influence	−.77	.64	−.26			
Native → sex under the influence	−.16	2.56	−.03			
Free lunch → sex under the influence	.00	.26	.00	−.11	.08	−.05
Older cohort → sex under the influence				−.25	.08	−.11**
Treatment → sex under the influence	.08	.18	.03	−.11	.08	−.05
Lifetime sex partners → STI diagnosis	.05	.01	.27***	.04	.01	.21***
Sex under the influence → STI diagnosis	.09	.08	.09	.14	.06	.13*
Early sex → STI diagnosis	.14	.09	.15	.17	.09	.18*
Early alcohol use → STI diagnosis	.10	.07	.09	.02	.06	.02
Pubertal age → STI diagnosis	−.01	.04	−.01	.00	.04	.00
Behavioral disinhibition → STI diagnosis	−.15	.09	−.10****	.03	.06	.03
Family management → STI diagnosis	−.17	.19	−.05	−.18	.14	−.06

Appendix 1
Continued

Parameter	SSDP sample			RHC sample		
	Unstandardized estimate	Standard error	Standardized estimate	Unstandardized estimate	Standard error	Standardized estimate
School bonding → STI diagnosis	−.19	.13	−.08	.17	.17	.05
Antisocial peers → STI diagnosis	.31	.15	.12*	−.02	.12	−.01
Child of teen parent → STI diagnosis	−.05	.15	−.02	.12	.17	.03
Male → STI diagnosis	−.71	.15	−.29***	−.92	.12	−.37***
White → STI diagnosis				−.07	.12	−.03
Black → STI diagnosis	.41	.15	.15**			
Asian → STI diagnosis	−.01	.23	.00			
Native → STI diagnosis	−.10	.89	−.02			
Free lunch → STI diagnosis	.25	.14	.10****	.10	.11	.04
Older cohort → STI diagnosis				.21	.11	.09*
Treatment → STI diagnosis	−.24	.20	−.10	−.11	.10	−.04
Correlational paths						
Early alcohol use ↔ early sex	−.01	.08	−.01	.21	.06	.21***
Sex under the influence ↔ lifetime sex partners	1.56	.29	.27***	2.53	.24	.44***

STI = sexually transmitted infection.
*$p < .05$; **$p < .01$; ***$p < .001$; ****$p < .10$.

Appendix 2
Model variable intercorrelations

	Lifetime STI diagnosis	Lifetime partners	Sex under influence	Early alcohol use	Early sex	Family management	School bonding	Antisocial peers	Pubertal age	BD
1. Lifetime STI diagnosis		.36***	.26***	.17**	.35***	−.16**	−.19**	.30***	−.16**	.14*
2. Lifetime partners	.36***		.47***	.21***	.45***	−.13***	−.18***	.28***	−.14***	.33***
3. Sex under influence	.29***	.60***		.18***	.45***	−.15**	−.16***	.34***	−.11*	.36***
4. Early alcohol use	.16***	.25***	.30***		.15**	−.15***	−.26***	.21***	−.09**	.26***
5. Early sex	.35***	.44***	.29***	.38***		−.22***	−.20***	.44***	−.31***	.42***
6. Family management	−.11*	−.13***	−.15***	−.35***	−.24***		.34***	−.28***	−.01	−.20***
7. School bonding	−.01	−.11***	−.17***	−.30***	−.20***	.43***		−.24***	.09*	−.23***
8. Antisocial peers	.09*	.28***	.31***	.44***	.44***	−.41***	−.40***		−.08*	.46***
9. Pubertal age	−.11*	−.14***	−.10*	−.09**	−.26***	.03	.03	−.11***		−.15***
10. BD	.13**	.32***	.32***	.35***	.46***	−.24***	−.26***	.49***	−.11***	
11. Male	−.37***	.03	.02	.08*	−.05	−.06****	−.13***	.27***	.04	.26***
12. Teen parent	.09*	.11***	.06	−.04	.11*	.06****	.02	.02	−.04	.03
13. White	−.05	−.05	.03	−.03	−.04	.03	−.06****	−.04	.06****	.05****
14. Black										
15. Asian										
16. Native										
17. Free lunch	.11*	.12***	.01	.08*	.19***	.01	.09**	.09**	−.04	.01
18. Older cohort	.01	−.11**	−.10**	−.07*	−.12**	.03	.02	.02	.02	.07*
19. Treatment	−.08****	−.01	−.04	.03	−.03	−.05	−.07*	.01	.02	−.02

	Male	Teen Parent	White	Black	Asian	Native	Free Lunch	Older Cohort	Treatment
1. Lifetime STI diagnosis	−.15***	.12*		.27***	−.22***	.00	.14**		−.19*
2. Lifetime partners	.23***	.10**		.07****	−.21***	.03	−.05		−.05
3. Sex under influence	.26***	.08****		.13**	−.35***	.04	−.04		−.05
4. Early alcohol use	.01	−.04		−.07****	−.28***	.01	−.19***		−.02
5. Early sex	.28***	.19***		.24***	−.30***	.09****	.08****		−.22**
6. Family management	−.11***	−.02		.04	−.08*	.00	−.08*		.14**
7. School bonding	−.13***	−.02		−.01	.21***	.01	.08*		.20***
8. Antisocial peers	.17***	.16***		.26***	−.19***	.00	.09*		−.10*
9. Pubertal age	.12**	−.11**		−.07*	.14***	−.01	−.01		.01
10. BD	.15***	.05		.04	−.22***	.05	−.06		.00
11. Male		.02		.00	.04	−.07****	−.05		−.03
12. Teen parent	−.04	−		.22***	−.11**	−.03	.13***		−.16**
13. White	.03	−.01							
14. Black					−.31***	−.14	.27***		−.10*
15. Asian						−.13	.16***		.07
16. Native							.06		.07
17. Free lunch	.00	.11***	−.19***						−.01
18. Older cohort	.04	−.08*	.01				−.08*		
19. Treatment	.04	−.03	.00				−.10**	−.04	

Correlations for the Seattle Social Development Project dataset are presented above the diagonal. Correlations for Raising Healthy Children are below the diagonal.
BD = behavioral disinhibition; STI = sexually transmitted infection.
*$p < .05$; **$p < .01$; ***$p < .001$; ****$p < .10$.