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Long-term consequences of early sexual initiation on young adult health: A causal inference approach

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Abstract

Although early sexual initiation has been linked to negative outcomes, it is unknown whether these effects are causal. In this study, we use propensity score methods to estimate the causal effect of early sexual initiation on young adult sexual risk behaviors and health outcomes using data from the National Longitudinal Study of Adolescent to Adult Health. We found that early sexual initiation predicted having 2 or more partners (for both males and females) and having a sexually transmitted infection in the past year (females only) but did not predict depressive symptoms in the past week (for either gender). These results underscore the importance of continued programmatic efforts to delay age of sexual initiation, particularly for females.

Introduction

Many adolescent sexuality education programs aim to delay the onset of sexual initiation to improve behavioral, developmental, and health-related outcomes of individuals (Chin et al., 2012; Kirby, 2007). However, to provide evidence that this focus is warranted, it is important to establish that early sexual behavior indeed *causes* negative outcomes. Studies have linked early sexual initiation to negative physical (Kaestle, Halpern, Miller, & Ford, 2005; Kaplan, Jones, Olson, & Yunzal-Butler, 2013) and mental health (Jamieson & Wade, 2011) outcomes, but determining whether the effects are causal is complicated by the presence of many third variables (i.e., confounders) that represent alternative causal explanations (e.g., the association between depression and early sexual initiation may be explained by pubertal timing). Understanding causal effects is critically important in creating efficacious programs: if third-variable explanations are the true cause of associations between early sexual initiation and later behavioral and health-related outcomes, then programs designed to

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Author's Contributions

KCK conceived of the study, participated in its design and coordination and drafted the manuscript; SAV participated in the design and interpretation of the data and helped to draft the manuscript; NAB performed the statistical analysis; and DLC participated in the design and assisted with the statistical analyses and interpretation of the results. All authors read and approved the final manuscript.

delay initiation will not have an effect on later outcomes. Causal inference techniques such as propensity score methods (Rosenbaum & Rubin, 1983) hold potential for strengthening causal inferences about the effect of early sexual initiation on later sexual behaviors and health outcomes, but they have not yet been widely used to answer this question. In this study, we use longitudinal data from a nationally representative sample of adolescents to estimate the long-term causal effects of early sexual initiation.

Early sexual initiation is associated with a number of negative risk behaviors and negative health outcomes, including having more sexual partners (Coker et al., 1994; Kaplan et al., 2013; O'Donnell, O'Donnell, & Stueve, 2001; Sandfort, Orr, Hirsch, & Santelli, 2008), inconsistent condom use (Coker et al., 1994; Kaplan et al., 2013), and STIs (Coker et al., 1994; Kaestle et al., 2005). In addition, early sexual initiation is associated with an increased likelihood of depression one year later for adolescent girls (Coker et al., 1994; Spriggs & Halpern, 2008). However, not all studies have found such negative effects (Sabia, 2006), and longitudinal research suggests that the impact of early sexual initiation either decreases or disappears by young adulthood (Kaestle et al., 2005; Spriggs & Halpern, 2008).

Prior studies are limited in their ability to estimate the *causal* effect of early initiation on later outcomes. One approach to rule out third variable explanations is random assignment, which is not possible in the case of early sexual initiation. Another approach is regression adjustment by including the confounders as covariates; however, this approach is prohibitively complex and could result in unstable estimation due to all of the potential confounders. Yet another approach is the use of propensity score methods (Rosenbaum & Rubin, 1983), which attempt to mimic randomization by balancing measured confounders across each exposure group, (i.e., groups experiencing and not experiencing early sexual initiation), thus strengthening the causal inference.

Little extant research has used propensity score methods to estimate the effect of early sexual initiation on later sexual behaviors or health outcomes. One study used propensity score methods among a sample of same-sex identical twin pairs (which helps control for genetic factors) and found that among young adult males, early sexual initiation was associated with a greater number of sexual partners in the past year and in a lifetime, and among males and females it was associated with past-year intercourse under the influence of alcohol or drugs (Huibregtse, Bornovalova, Hicks, McGue, & Iacono, 2011). A limitation of this study was the relatively late cutoff for early sexual initiation (≤ 16 years of age), which may influence the limited findings, especially for females, because very early sexual initiation (e.g., 13 years old) may place an individual on a more negative trajectory than initiating at more normative ages (i.e., 15 or 16; Finer & Philbin, 2013; Sandfort et al., 2008). Furthermore, this study focused primarily on predicting sexual risk behaviors and not other health outcomes, such as STIs or mental health. Another study using propensity score matching found that early initiation was associated with increased depression for adolescent girls who broke up with their first sexual partner (Meier, 2007). However, this study did not examine whether the effects were long-lasting.

Using data from a nationally representative sample of adolescents from the National Longitudinal Study of Adolescent to Adult Health (Add Health), the current study uses

propensity score methods to estimate the long-term causal effects of early sexual initiation (defined as age 14 or younger) on number of past-year sexual partners, STIs, and depressive symptoms in young adulthood (when youth are at heightened risk for negative health outcomes; Weinstock, Berman, & Cates, 2004) and to test for gender differences in these associations.

Methods

Study Population

Data for the current study are from the contractual use dataset of Add Health, a nationally representative sample of adolescents in 7th–12th grade at study entry. Eighty high schools and 52 middle schools in the US participated in the study. Systematic sampling methods and implicit stratification were used to ensure that the sample was representative of all United States (US) schools at the time (Harris, 2009). The current study used data from Wave I (collected in 1994–1995) and Wave III (collected in 2002–2003). To ensure that the temporal ordering of events was maintained (i.e., that the potential confounders were measured before sexual initiation and that the outcomes happened after initiation), we restricted the analytic sample to adolescents who (a) were 11 to 13 years old at Wave I ($N=2,865$), (b) had not initiated sexual intercourse at Wave I ($N=2,530$), (c) were present at Wave III, and (d) had complete data on the outcome measures ($N=1,902$; mean age at Wave I = 12.8; 58% female; 62% White, 19% Black, 11% Hispanic, 4% Asian, and 3% other race/ethnicity).

Measures

Early Sexual Initiation (Measured Retrospectively at Wave III)—Early sexual initiation was assessed by taking the response to the question, “How old were you the first time you had vaginal intercourse?” and dichotomizing it into two groups: *Early sexual initiation* (age 14 or younger, coded 1; 13.5% of the sample) and *Not early initiation* (age 15 or older, coded 0). Individuals who had not initiated intercourse by Wave III ($n = 433$ or 22%) were coded as 0.

Outcomes (Measured at Wave III)—*Multiple partners* in the past year was assessed using the response to the question, “With how many sexual partners have you had vaginal intercourse in the past 12 months?” This item was dichotomized into 0–1 partners (coded as 0) versus 2 or more partners (coded as 1). At Wave III, 34% of males and 27% of females had 2 or more partners in the past year. A measure of any *STI* in the past year was created by summing the “Yes” responses to the question, “In the past 12 months, have you been told by a doctor or nurse that you had the following sexually transmitted diseases?”, followed by the names of 13 STIs plus an “other” category, then dichotomizing into 1= one or more STIs (8%), or 0=no STIs in the past year. This self-report measure was used because it had less missingness, captured a longer time duration, and included a comprehensive list of STIs (compared to the biological STI measures). A 9-item version of the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) was used to assess psychological and somatic *depressive symptoms* ($\alpha=.81$). Individuals responded to statements asking them to rate the frequency of each symptom in the past week. Responses were on a 4-point scale ranging from 0=*rarely* to 3=*most of the time*. We dichotomized

depressive symptoms using a cut-off that correlates to a diagnosis of clinical depression, similar to other studies using Add Health (10 for boys and 11 for girls; see Lehrer, Shrier, Gortmaker, & Buka, 2006; Roberts, Lewinsohn, & Seeley, 1991). Based on these cut points, 7% of males and 11% of females had symptoms consistent with clinical depression.

Potential Confounders (Measured at Wave I)—Based on previous research showing associations between demographic, biological, family structure and process, peer, school, mental health, religion, problem behavior, (see Zimmer-Gembeck & Helfand, 2008 for review) and neighborhood (Browning, Burrington, Leventhal, & Brooks-Gunn, 2008) factors and sexual behavior we selected 59 Wave I variables as potential confounders. The details of the confounders, organized by these categories can be found in the Supplemental Table. Note that there is no increased bias by including variables that are associated with only the exposure or the outcome, and thus it is beneficial to select a broad range of potential confounders (Brookhart et al., 2006).

Statistical Analysis

First, we computed propensity scores, which were defined as an individual's probability of being exposed (i.e., initiating sex age 14 or younger) versus not exposed (i.e., initiating sex at age 15 or older) given the measured values of the confounders (Rosenbaum & Rubin, 1983). Generalized boosted modeling (GBM; McCaffrey, Ridgeway, & Morral, 2004) was used to obtain the predicted probabilities, which are estimates of the propensity scores. GBM is a non-parametric method that has been shown to out-perform logistic regression because of its ability to permit missing data on the confounders and include higher-order interaction terms (Lee, Lessler, & Stuart, 2009). Wave III sample weights were included in the propensity score model to control for any confounding introduced by the differential probability of selection into the baseline sample. Because sufficient overlap between groups is necessary to justify the use of propensity score methods for strengthening causal inference (Harder, Stuart, & Anthony, 2010), we assessed overlap of the propensity score distributions for the exposure groups (i.e., early sexual initiation vs. not early initiation) using boxplots. Figure 1 shows that although the mean propensity scores were different for each group, there was sufficient evidence (visual assessment) of overlap to proceed with analyses.

Next, inverse propensity weights (IPWs; Robins, Hernan, & Brumback, 2000) were used to weight the data to ensure balance on the measured confounders across the exposure groups (similar to the goal of randomization). The IPWs were calculated as the inverse of the estimated propensity score for the early sexual initiation group and the inverse of one minus the estimated propensity score for the not early initiation group. In models where gender was considered as a moderator, the IPWs were conditioned on gender (Robins, Hernan, & Brumback, 2000). Balance was assessed by calculating standardized mean differences (Stuart, 2010) between exposure groups for each of the confounders; an absolute standardized mean difference less than .25 is generally considered to be a small effect size (Cohen, 1988; Harder, Stuart, & Anthony, 2010). Estimation of the propensity scores, calculation of the IPWs, and calculation of standardized mean differences were computed using the *twang* package in R (Ridgeway, McCaffrey, Morral, Burgette, & Griffin, 2013).

Figure 2 shows that all of the potential confounders were balanced ($\leq .25$) across the initiation groups once the data set was weighted by the IPWs.

Finally, the average causal effect of early sexual initiation on each of the outcomes was estimated using a weighted logistic regression model using the IPW. Cluster and strata variables were included to adjust for the complex survey design (Chantala, 2006). Three regression models were run for each outcome: a model with early initiation only (Model 1), a model controlling for gender (Model 2), and a model with an interaction term between sexual initiation and gender (Model 3). In instances where the interaction term was statistically significant ($p < .05$), we present stratified analyses to facilitate interpretation.

Results

Table 1 shows the prevalence of young adult (i.e., age 19–21) behaviors and health outcomes, stratified by early initiation group. The prevalence of each outcome was statistically significantly higher for the early sexual initiation group compared to not early initiation.

The effect of early sexual initiation on having multiple sexual partners in the past year was positive and significant (see Table 2); adolescents who had initiated sexual intercourse early had 3.33 (95% CI: 2.48, 4.48) times greater odds of having 2 or more partners in young adulthood. There was no evidence to suggest that this effect differed by gender ($p = .24$).

The effect of early initiation on having an STI in the past year was also positive and significant (see Table 2). A significant interaction term between early initiation and gender suggested that the effects may vary across gender ($p = .04$). Upon stratification, the effect of early initiation on having an STI was not significant for males ($p = .44$) but was significant for females ($p < .01$). Females who initiated early had 3.12 (95% CI: 1.93, 5.26) times greater odds of having an STI than those who did not initiate early.

Finally, the effect of early initiation on depressive symptoms in young adulthood was not significant in the unadjusted model or a model controlling for gender (Models 1 and 2 in Table 2). However, there was a statistically significant interaction effect between gender and early initiation (Model 3; $p = .05$). Upon stratification by gender, neither of the effects were statistically significant (males: OR = .54; 95% CI: .23, 1.20; females: OR = 1.43; 95% CI: .85, 2.43); however, since the coefficients were in opposite directions, a significant interaction term emerged in the full model, (see Figure 3) despite a non-significant main effect.

Discussion

The goal of the current study was to estimate the long-term causal effects of early sexual initiation on young adult sexual risk behaviors and health outcomes by using propensity score methods to strengthen causal inference. The results suggest that engaging in intercourse at a young age does have an impact on later outcomes that cannot be better explained by third variables such as biological factors (e.g., physical maturation), personality traits (e.g., impulsivity), and physical environment (e.g., neighborhood drug problems). Thus

our study provides some support for interventions that promote delaying sexual intercourse to achieve better health outcomes.

Results showing that early initiation predicts two or more past year partners in young adulthood for both male and females corroborates the findings from other studies using traditional regression analyses (Sandfort et al., 2008). However, this finding, combined with the finding that early initiation had a significant effect on past-year STIs only for females, suggests that factors other than number of sexual partners may explain the association between early sexual initiation and STIs for males. This may be due to women's greater biological susceptibility to many STIs (Coombs, Reichelderfer, & Landay, 2003), which may mean the magnitude of risk conferred by each additional sexual partner is greater for women than men. Alternatively, early initiation may increase the likelihood of choosing riskier partners or engaging in more risky sexual behaviors for women. Future research should examine these potential explanations.

Past research on the effect of early sexual initiation on later depressive symptoms has demonstrated short-term effects (Meier, 2007) but no significant effects in young adulthood (Spriggs & Halpern, 2008). Our results corroborate prior findings, as we found no significant association between early initiation and young adult depressive symptoms. However, a significant interaction effect by gender (i.e., coefficients were in opposite direction) suggests that males and females may experience different psychological processes related to early sexual behavior (Higgins, Trussell, Moore, & Davidson, 2010). This may be a result of sexual double standards (Crawford & Popp, 2003) that may make sex more distressing for female compared to male adolescents. The gender differences found in both sexual risk and mental health outcomes suggest that male adolescents may need different messaging than female adolescents, as is being addressed by emerging programs such as HoMBRes (Martinez, Roth, Kelle, Downs, & Rhodes, 2014). There also may be subgroups of individuals with different mental health outcomes of sexual behavior, and future research should examine this possibility.

There are some limitations to the current study. An assumption of propensity score methods is that there are no unmeasured confounders (Rosenbaum & Rubin, 1983), and it is possible that unmeasured confounders exist. For example, sexual abuse has been found to be a strong predictor of early sexual initiation (Senn, Carey, & Vanable, 2008); however, Add Health did not collect comprehensive data about abuse in a way that would allow us to determine whether abuse occurred prior to sexual initiation. In addition, all of the outcome measures were self-report, and underreporting may have influenced these findings. Further, there may be differences in these outcomes by race/ethnicity or socioeconomic status, and future research could examine this possibility.

The current study focuses solely on early vaginal sexual initiation. Although other sexual behaviors (i.e., oral or anal sex) confer STI risk (Baggaley, White, & Boily, 2010), there is evidence to suggest that oral sex often occurs in close proximity to vaginal sex and anal sex much later than first vaginal sex (Lindberg, Jones, & Santelli, 2008). Future studies could explore how initiation of these other sexual behaviors is related to later behavioral and health outcomes. In addition, future research could compare the effects of early, normative and late

initiation, as there may be further variability in outcomes among those with normative versus late initiation (Sandfort et al., 2008) and how factors like partner and relationship characteristics at the time of initiation (Sayegh, Fortenberry, Shew, & Orr, 2006) may explain how individuals may have less negative outcomes of first intercourse, as found in other developed countries (Tietler, 2002; Santelli, Lindberg, Finer, & Singh, 2007). A holistic, person-centered approach, such as latent class analysis (Lanza, Collins, Lemmon, & Schafer, 2007), that includes multiple aspects of sexual behavior, could provide more information about whom to target and how to tailor intervention messages around timing of sexual initiation.

This study extends literature on outcomes of early sexual behavior by strengthening causal inference and examining gender differences. Results of the current study provide evidence that early sexual initiation has long-lasting negative consequences on sexual risk behaviors and health outcomes. These findings suggest that current programmatic efforts such as the evidence-based interventions promoted by the CDC (2015) to delay sexual initiation are warranted, particularly for female adolescents (DiClemente et al., 2004). However, the lack of significant findings for males in terms of STIs and the suggestive, yet non-significant, finding that early initiation may be protective against depressive symptoms suggests that male and female adolescents may benefit from different messages in prevention programs.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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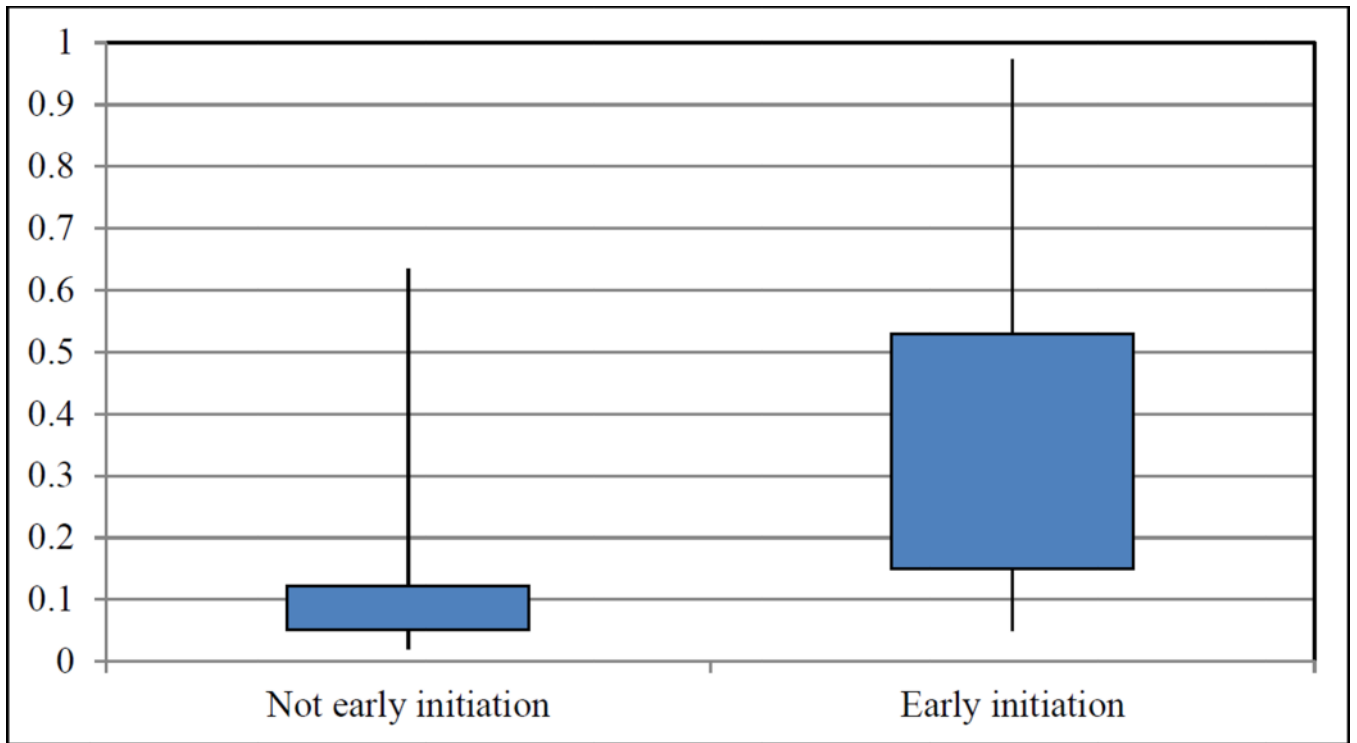


Figure 1. Boxplot showing the overlap in the distribution of the propensity scores between early and not early sexual initiation groups.

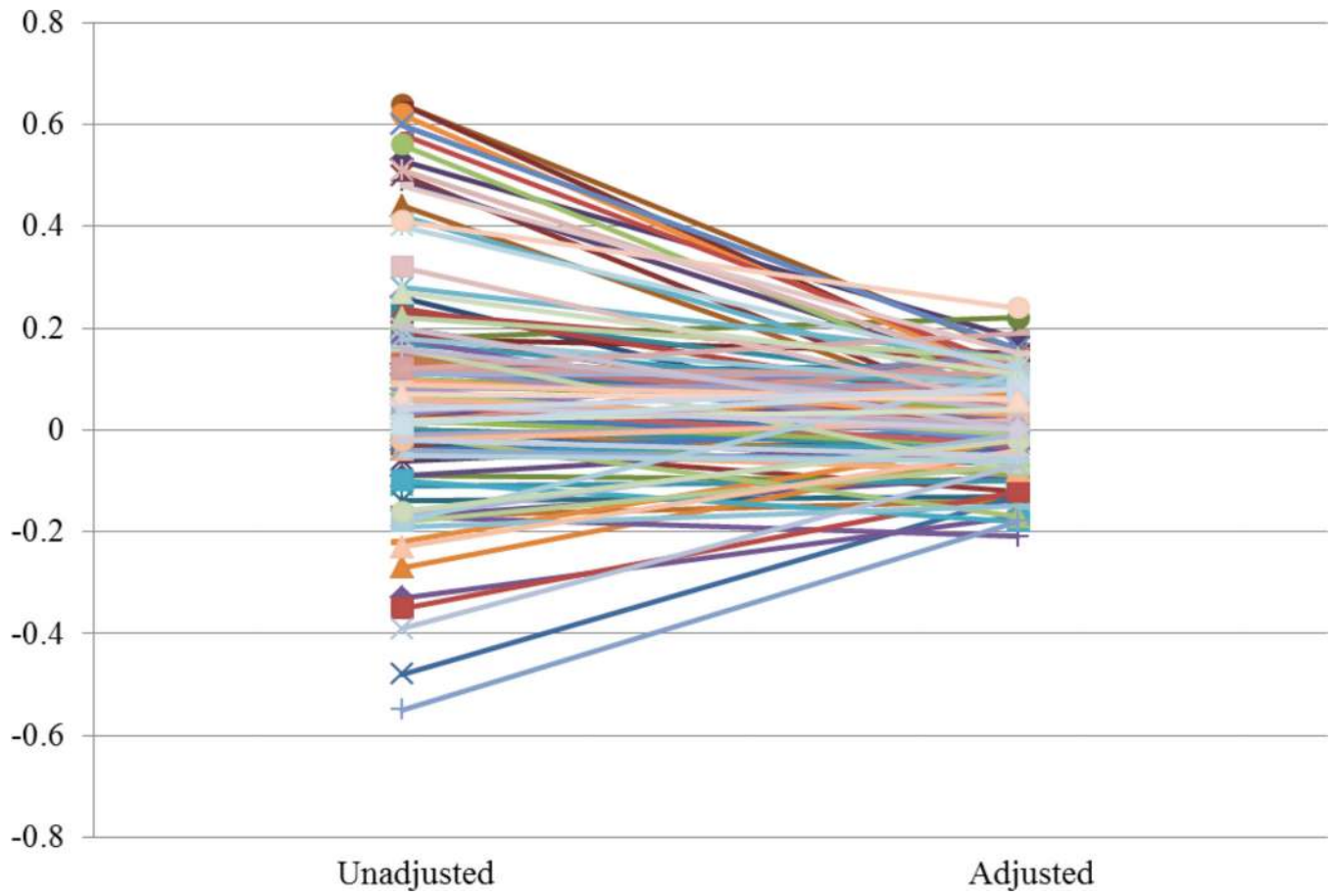


Figure 2. Standardized mean differences between early and not early sexual initiation groups for each confounder for the unadjusted and the propensity-score-adjusted data.

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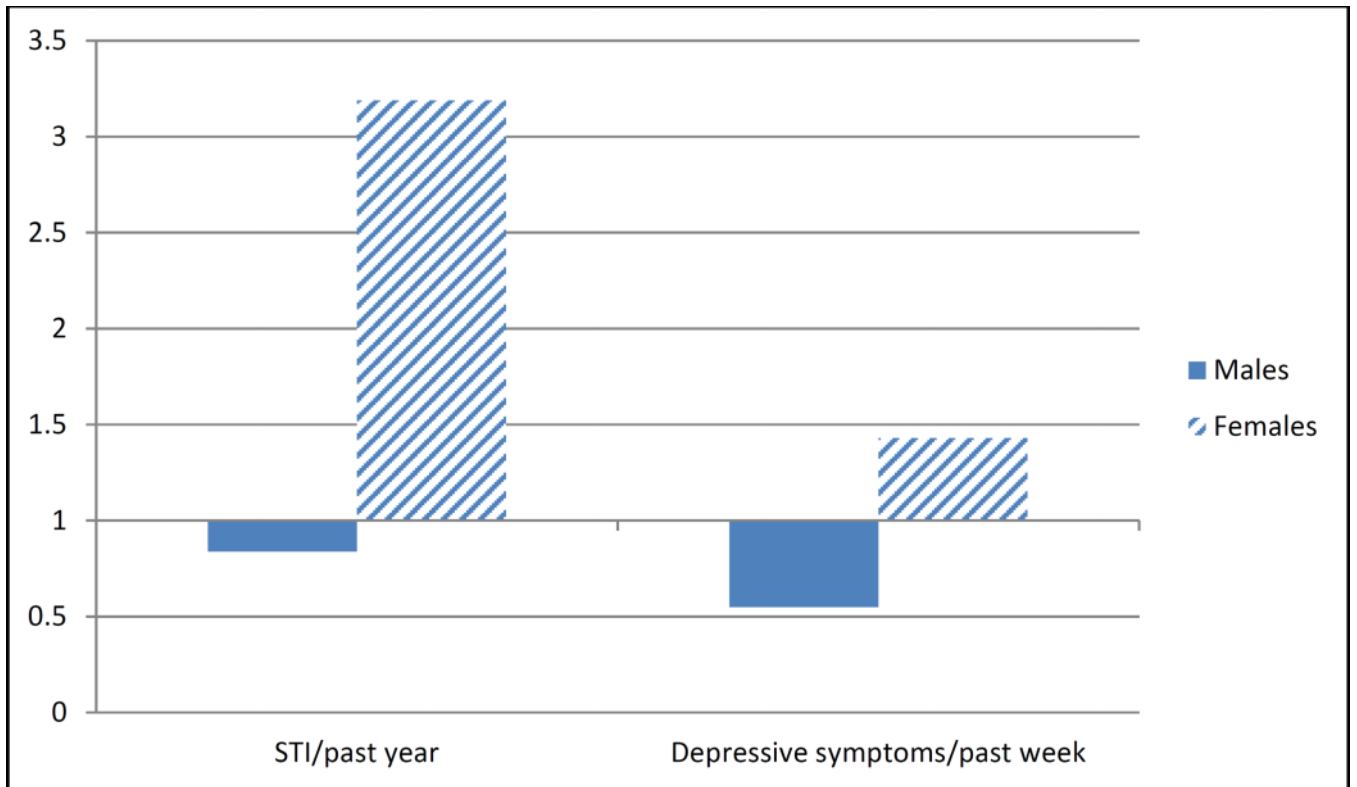


Figure 3. Propensity-score-adjusted odds ratios for the effect of early sexual initiation on past year STI and depressive symptoms in young adulthood, stratified by gender

Table 1

Prevalence of young adult sexual behaviors and health outcomes by early sexual initiation group

Outcome	Overall %	Early sexual initiation % (N= 433)	Not early initiation % (N=1469)	p-value
2 or more partners/past year	30.1	50.8	26.9	<.001
STI/ past year	7.6	18.8	5.9	<.001
Depressed symptoms/past week	9.5	14.5	8.8	<.001

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Odds ratios (and 95% confidence intervals) estimating the causal effect of early sexual initiation on young adult health behaviors and outcomes (n=1902)

Table 2

Predictor	2 or more partners/past year		STI/past year		Depressed symptoms/past week	
	OR	95 % CI	OR	95 % CI	OR	95 % CI
Model 1:						
Early initiation	3.25	(2.42,4.37)	2.71	(1.74,4.23)	1.15	(.73,1.80)
Model 2:						
Early initiation	3.33	(2.48,4.48)	2.70	(1.70,4.30)	1.13	(.72,1.77)
Female	.66	(.51,.85)	4.55	(2.64,7.82)	1.65	(1.22,2.24)
Model 3:						
Early initiation	4.31	(2.69,6.89)	.84	(.25,2.76)	.55	(.25,1.20)
Female	.69	(.52,.91)	3.78	(2.10,6.81)	1.52	(1.11,2.08)
Early*Female	.65	(.32,1.32)	3.82	(1.07,13.65)	2.61	(.98,6.96)