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Intergenerational Associations in Sexual Onset: Mediating Influences of Parental and Peer Sexual Teasing and Youth Substance Use

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Abstract

Purpose—The prospective intergenerational association between fathers' age of onset of sexual intercourse and their son's or daughter's age of onset of oral sex or intercourse up to 30 years later was examined using survival analyses across child ages 11–12 to 17–18 years. Additionally, novel constructs of parental and peer sexual teasing (at ages 11–12 years) and general risk factors of child substance use onset (alcohol and marijuana) prior to or concurrent with sexual onset were assessed as predictors of children's sexual onset and mediators of intergenerational associations.

Methods—Hypotheses were tested using the Oregon Youth Study Intergenerational sample, including 100 fathers and 176 children (46% girls).

Results—Univariate findings indicated that children were at risk for earlier sexual onset, provided their fathers had onset on sex at younger ages; in addition, child alcohol and marijuana onset and parental, but not peer, sexual teasing predicted earlier age of sexual onset for children. Multivariate findings indicated child alcohol onset fully mediated the intergenerational association in age of sexual onset, whereas parental teasing and child marijuana onset did not explain the intergenerational association.

Conclusions—Findings of intergenerational associations in risk of sexual onset indicate that mechanisms of this association should be further examined. Substance use onset also confers risk for earlier child sexual onset, with alcohol use onset accounting for intergenerational associations; thus, substance use onset should be a prime target for prevention. Parental sexual teasing warrants further study as a mechanism related to possibly unintended encouragement of youth's early sexual onset.

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Keywords

adolescence; alcohol use; fathers; intergenerational transmission; marijuana use; parent sexual teasing; peer sexual teasing; sexual onset

Sexual risk behaviors in adolescence expose youth to risk for early parenthood and sexually transmitted infections (STIs).^[1,2] According to recently released surveillance data by the Centers for Disease Control,^[3] youth aged 19 years or younger accounted for 19% of gonorrhea cases and 27% of chlamydia cases in 2015. Most research on the etiology of risky sexual behavior in adolescence has focused on risk factors in adolescence.^[4] The relatively few prospective studies that have examined risk factors from childhood have generally focused on factors related to the development of problem behaviors, in general, including substance use. It is well established that conduct problems and relatedly substance use are risk factors for early onset of intercourse (hereafter referred to as sexual onset) and for risky sexual behavior more generally.^[5–7]

Familial risk factors examined for sexual onset often have been factors also related to the development of conduct problems—particularly parenting, including monitoring of the child’s behavior. Parent-child communication has generally been examined as a protective rather than as a risk factor, considering aspects such as communications about sexuality.^[8] For example, Elkington et al.^[9] conducted qualitative interviews with youth on probation and their probation officers. Interview probes regarding communication referred to how much parents’ listened to the youth, and whether they talked about dating and sex with their parent(s). The chief negative communication risks identified involved conflict between parents and youth. However, parental verbal behaviors that are outcome specific and might actually encourage sexual involvement, such as sexual teasing, have been little considered as a possible risk factor for sexual onset.

Regarding family influences, effects of fathers’ behaviors on the sexual risk behavior of offspring are less often examined than are those of mothers.^[10] For example, Guilamo-Ramos et al.^[11] conducted a systematic review of paternal influences on adolescent sexual risk behaviors and found just 13 studies, of which only 6 were longitudinal and thus had adequate designs for examining prediction from risk factors. Of these, only three predicted to sexual onset, and all of these studies used the same data set (National Survey of Adolescent Health), with prediction over relatively short time frames within adolescence (the longest being about 7 years). Overall longitudinal findings indicated that positive emotional qualities of father-adolescent relationships were associated with later sexual onset.

The purpose of the present study was to test a model integrating general pathway risk for sexual onset, assessed by alcohol and marijuana use, and outcome-specific risk from parental and peer influences, assessed by parent and peer sexual teasing, in explaining the intergenerational association of fathers’ age of sexual onset with that of their child. Models tested using survival analysis^[12] were fully prospective over up to a 30-year period.

Conceptual model

The model underlying the present study is the Dynamic Developmental Systems model (DDS^[13,14]) that emphasizes interplay among systems affecting an individual's behavior—including individual developmental history—and social influences, which vary by developmental stage (e.g., the importance of peer influences in adolescence). The DDS approach builds on lifespan approaches emphasizing the interaction between the individual's prior dispositions and learning and the environments in which s/he is placed or selects.^[15,16] A key aspect of the model is the importance of both general pathway risk and outcome-specific risk^[17,18] that helps bring conceptual clarity to questions regarding the development of risk behaviors.

In the present study, general pathway risk for sexual onset is assessed by substance use, which is strongly predictive of sexual risk behavior.^[19,20] Midadolescents who report substance use also report earlier ages at which a variety of sexual behaviors are acceptable, including kissing, touching breasts and genitals, oral sex, and vaginal intercourse.^[21] When the Oregon Youth Study (OYS) fathers were adolescents, trajectories of substance use indicated an upturn at about the time of first intercourse.^[5] Further, in a study through ages 22–23 years for the OYS fathers, substance use was predictive of lifetime average sexual risk behavior and with contraction of an STI. It is thus possible that substance use by the child may partially explain and, therefore, mediate intergenerational associations in sexual risk behavior and early onset.

Regarding outcome-specific risk factors for sexual onset, a father's age at first sex, which may be communicated to the child and also affect parental permissive attitudes toward early sexual involvement^[22] is expected to predict a younger age at first sex for their child.^[5] Study of the outcome-specific risk factor of parental history of sexual risk behaviors has mainly focused on the observation that being the child of a teenage mother—indicative of early sexual involvement of the mother—predicts earlier sexual onset.^[23,24] We have found in prior work on alcohol that parents may be observed to communicate permissive attitudes via joking and talk that may be considered encouragement of a risk behavior.^[25] Parental use of alcohol was related both to encouragement of alcohol use during parent-child interactions (at child age about 11 years) and to child early onset of alcohol use. In the present study, we propose to test whether similar outcome-specific family influences may relate directly to an earlier age at sexual onset and mediate the intergenerational transmission of early sexual involvement. Possible greater tolerance for early sexual onset due to prior early onset by the parent themselves may be communicated to the child in ways, such as by talking with the child about the age at which the parent became sexually involved or talk about sexual behaviors in front of or with the children (e.g., joking and telling anecdotes about past sexual escapades; teasing the child about boyfriends/girlfriends, body development, or sexual involvement).

Peer risk factors also are important social influences at adolescence and, for sexual risk behaviors, include the general risk pathway factor of association with antisocial and delinquent peers^[5,26] and outcome-specific risk from association with peers who are sexually active.^[27] Interactional processes that may be encouraging of sexual engagement

have been less considered. Peers can encourage or reinforce engagement in antisocial or rule-breaking behaviors through “deviancy training”, whereby shared laughter and positive affect during mention of such behaviors in discussions predicted higher levels of antisocial behavior.^[28] We posit that an outcome-specific peer interaction process that may predict early sexual involvement is peer teasing related to sexual issues, similar to the parental teasing already described.

Hypotheses

It was hypothesized that the father’s age at sexual onset would be predictive of their child’s sexual onset (assessed across ages 11–12 to 17–18 years) and that both parent and peer sexual teasing would add significantly to prediction. Also, it was expected that the more proximal outcome specific risks (teasing by both parents and peers) and general risk factors (child onset of alcohol and/or marijuana use) for sexual involvement would partially account for (mediate) any intergenerational association. Finally, all multivariate models controlled for boys elevated risk for earlier sexual onset relative to girls.^[29] For the present study, sexual onset was defined as youth report of engagement in either sexual intercourse or in oral sex. Onset of each of these behaviors increases rapidly across adolescence. As oral sex has risk for STI transmission,^[30–32] oral sex was included in the definition of sexual onset of the child.

Method

Participants

The study involved children ($N = 176$) of 100 fathers and 117 mothers. Fathers were originally recruited as boys to the OYS (from 1984 to 1985), a community-based sample that involved recruiting all boys in the fourth-grade classes (aged 9–10 years) of schools in neighborhoods (74%; $n = 206$ participation) with higher-than-average rates of delinquency in a medium-sized metropolitan area in the Pacific Northwest. Participants were representative of the neighborhoods at the time; 90% of the boys were White, and most were from families of lower socioeconomic status, predominantly below college level and in manual jobs.^[33] Participation rates were 89% or better at each assessment following the boys then men from ages 9–10 to 37–38 years.

The Three Generational Study (3GS) recruited the offspring of the OYS men and these children’s mothers. After initial assessments, recruitment was limited to the first two biological children per woman (i.e., OYS men who fathered children with more than one woman could have more than two children followed in 3GS). Child assessments occur at ages 21 months (T1) and ages 3, 5, 7, 9, 11–12, 13–14, 15–16, and 17–18 years (T2–T9). The study is ongoing as children are still being born to the OYS men, with a 90% retention rate of Generation Three children. However, the N available for each wave is determined by timing of the children’s births. For the present study, the sample was limited to those offspring who were old enough to have completed at least the age 11–12 years assessment. As of spring 2016, this resulted in an N of 176 (46% male, $n = 81$) children of 100 OYS fathers (44 with 1 child, 37 with 2 children, 18 with 3 children, and 1 with 4 children) with 117 different biological mothers. Analytic adjustment for dependence among siblings’

scores is discussed below. Of the 176 participating children, 174 were biologically related to the OYS men, 2 were stepchildren. At 3GS T1, the children were age 1.7 (0.3 *SD*) years, whereas the fathers were age 26.0 years (2.9 *SD*) and the mothers were age 24.9 years (3.8 *SD*), on average. Of the children, all 176 participated at T6 (ages 11–12 years), 142 at T7 (ages 13–14 years), 95 at T8 (ages 15–16 years), and 24 at T9 (17–18 years). Twenty-four of the children participated in all 4 waves, 70 in 3 waves, 49 in 2 waves, and 33 participated in 1 wave.

Procedures

Assessments of the OYS boys/men included interviews and questionnaires. Assessments of the men's children in the 3GS included child- and parent-report questionnaires, interviews, and staff coding and ratings of father- and mother-child interactions. All measures and procedures were approved by the Oregon Social Learning Center Internal Review Board.

Measures

Father age at onset of intercourse—This was assessed by self-reports on a sexual behavior questionnaire^[34] administered yearly from ages 13–14 to 29–30 years. The age at the study wave when he first reported engaging in sexual intercourse was used to calculate his age at onset of intercourse.

Parent and peer sexual teasing of child—These were assessed (separately) by child report on teasing related to sexual issues, including four items at ages 11–12 years using the Teasing Questionnaire.^[35] Sample items are as follows: *How often do your friends (parents) tease you about: Having a boy/girlfriend, Whether you have kissed a boy/girl, Whether you have gone farther than kissing, and Body changes*. Response scales ranged from 1 = *never* to 4 = *a lot*. Reliable scales of parental sexual teasing ($\alpha = .66$) and peer sexual teasing ($\alpha = .71$) were formed. Of the children, 64.8% reported some parental teasing and 63% some peer teasing.

Child alcohol and marijuana onset—This was assessed by child reports in the interview regarding whether they had tried one whole drink of alcohol (beer, wine coolers, wine, and hard liquor) or had used any marijuana. Each of these onsets was coded “1” if the youth had started using the substance in any wave prior to or concurrent with the one where they reported first oral sex or intercourse or “0” otherwise.

Child sexual onset—At ages 11–12, 13–14, 15–16, and 17–18 years, the adolescents were asked if they had ever given or received oral sex (defined) and if they had ever had sexual intercourse (defined), and the first affirmative responses to either were used to define age of onset. Once a child had onset, all scores at subsequent ages were set to missing values, thus modeling only those children who remained at risk for sexual onset across each successive time period across adolescence from ages 11–12 to 17–18 years.

Data Analyses

Children's sexual onset assessed at four time points across adolescence (ages 11–12, 13–14, 15–16, and 17–18 years) was modeled using discrete-time survival analyses (DTSA^[12]). in

Mplus version 7.3.^[36] Dependence among siblings' scores was accounted for by adjusting the standard errors using a sandwich estimator. Discrete-time categories were defined by the child ages of 0–11, 11–12, 13–14, 15–16, and 17–18 years. Models were estimated assuming proportional odds, which imply that the effects of the predictors and covariates are equal across children's adolescence. Indirect (mediational) effects involving child sexual teasing and substance use onset were simultaneously estimated using the multiple mediator model.^[37] The predictors of parent and peer teasing were significantly skewed and thus transformed to approach a normal distribution and reduce the influence of outliers. Prior to modeling, these variables and father age of onset were standardized to obtain standardized regression coefficients from the DTSA models. For child alcohol and marijuana use onset, predictors were coded as *yes* = 1 and *no* = 0.

Results

Descriptive Statistics

Shown in Table 1 are the percentage of children who experienced sexual onset by each age category, and the proportion with alcohol or marijuana use onset prior to or concurrent with sexual onset. The correlations of father intercourse onset age with parental and peer teasing were $r = -.046$ ($p > .10$) and $r = -.121$ ($p > .10$), respectively. Thus, father age of intercourse onset was not predictive of his later sexual teasing of his child.

Survival Analyses

Univariate survival models—Six DTSA models were conducted to examine separately whether each of the five predictors and the control variable of child gender would relate to sexual onset. Findings are shown in Table 2 (Panel I). With the exception of peer sexual teasing, all predictors were associated with children's earlier age of sexual onset. As hypothesized, a younger age at onset for the father predicted an earlier age of sexual onset for their offspring. In addition, alcohol and marijuana use onset and parent sexual teasing of their child at ages 11–12 years predicted an earlier age of sexual onset. Finally, boys were likely to experience sexual onset earlier than girls.

Survival model of father age of onset of intercourse and parental teasing—In order to test whether parental teasing added to the prediction of child age of sexual onset over prediction from the father's age of onset and child gender, a survival analysis was conducted including only those three main effects. The findings (Table 1, Panel II) indicated that parental sexual teasing was marginally significant.

Multivariate Survival Model of Direct and Indirect Effects

The hypotheses that risk from fathers' earlier age of onset of intercourse on their children's earlier sexual onset would be mediated by parental teasing, alcohol onset, and marijuana onset were tested in a multivariate survival model that included simultaneous estimation of all direct and indirect effects (Figure 1). First, the direct effect of father age of sexual onset on child age of onset was nonsignificant in the multiple mediator model ($b[se] = -.29[.18]$, $p = .119$). Second, fathers' earlier age of onset significantly predicted child onset of alcohol and marijuana use, but not parental sexual teasing. Likewise, child onset of alcohol and

marijuana use, but not parental sexual teasing, were predictive of earlier sexual onset in children. Third, examination of the individual indirect effects revealed that child alcohol onset fully mediated the association between fathers' and children's earlier sexual onset ($b[se] = -.09[.045]$, $p < .05$), whereas child marijuana use onset and parental sexual teasing did not ($b[se] = -.08[.04]$, $p = .064$ and $b[se] = -.01[.02]$, $p = .636$), respectively; however, the indirect effect via marijuana onset was marginally significant). Finally, the multiple mediator model allows for the estimation of the total indirect effect (denoting the aggregate effect of parental teasing, alcohol onset, and marijuana onset), which was significant ($b[se] = -.19[.06]$, $p < .01$). Thus, intergenerational transmission for early sexual onset was fully mediated by child alcohol use onset; whereas evidence for child marijuana use onset and parental sexual teasing as mediating mechanisms was limited and only supported when all three risk factors for child sexual onset were considered.

Discussion

In this study, we found support for the intergenerational transmission of sexual onset risk in adolescence from fathers to children when examined over approximately a 30-year period using survival models with prospective data. Further, the effect was fully mediated by child alcohol use. New conceptualizations of specific social influence factors—namely parent and peer sexual teasing—were examined, in addition to mediational effects via the general risk pathways of child onset of alcohol and marijuana use. Findings indicated that reliable constructs of parental and peer sexual teasing—according to the child's reports—could be identified, but only parental teasing was associated with sexual involvement. As the parental teasing was assessed at child ages 11–12 years, it was prior to sexual onset (in all but one case); thus, it was not an outcome of onset. Interestingly, parental sexual teasing was also associated with child marijuana use. Perhaps sexual teasing by the parents indicates a general tolerance of the child's engagement in adult behaviors at an early age and a more deviant home environment. However, fathers' age at onset of intercourse was not predictive of such teasing, indicating that these were relatively independent risk factors. Contrary to the hypothesis, peer teasing was not predictive of sexual onset. This was unexpected, given that deviant talk with peers is predictive of problem behavior in adolescence.^[28] It may be that prediction would have been stronger if sexual teasing by parents and peers had been assessed closer in time to age of sexual onset—for example at age 14 years. Further, both parent and peer sexual teasing were assessed by a relatively small number of items. Findings for prediction from parent sexual teasing in the univariate model indicate that these constructs should be given further consideration as potentially encouraging of early sexual onset.

Findings of the present study confirm the strong association of substance use with sexual involvement in adolescence. Both alcohol and marijuana use onset were predictive of children's earlier sexual onset in a multivariate model, but only alcohol use onset was a significant mediator of the effect of fathers' age of onset on child onset. This may indicate that child alcohol use onset plays a more salient role than does marijuana use onset in explaining intergenerational transmission of sexual behaviors. However, as the mediating effect of marijuana onset was marginally significant, it is possible that effects of onset of these two substances were relatively similar. Father risk, indicated by his sexual onset in his

own adolescence, was related to transmission of individual or environmental risk conducive to adolescent onset for at least these two forms of substances at adolescence, and onset for these two substances by the child showed additive risk for their sexual onset (even though only alcohol was a significant mediator). These intergenerational findings support the linked nature of substance use and sexual involvement at adolescence. This indicates a need for parents, policy makers, and service providers to tackle these issues together.

The present study had a number of strengths, including prospective data on fathers and their children assessed across up to a 30-year period; examination of hypotheses regarding novel outcome-specific social influence processes from key social sources (parents and peers); and consideration of the role of general pathway risk factors for youth problem behaviors more broadly, assessed by onset of alcohol and marijuana use. One limitation was that the constructs were more heavily reliant on self-reports than is desirable. Second, the sample size was relatively small, and gender differences were not examined. Finally, the sample was predominantly, although not exclusively, White and of lower socioeconomic status. Generalizability to other populations should be examined.

In sum, findings from this study indicate that father earlier sexual onset is a risk factor for their children's earlier age of sexual onset. Parental sexual teasing in early adolescence was associated with child sexual onset, but was not a significant predictor in multivariate models. The findings also confirmed the strong connection between substance use and sexual involvement in adolescence. Such an association starts early in adolescence for some children and should be a primary prevention target.^[38,39] The findings for the novel construct of parent sexual teasing and child sexual onset indicate that this process is worthy of further examination as possibly being involved in encouraging sexual involvement that leads to risk for STIs and pregnancy at adolescence. Overall, both parental and peer sexual teasing should be further examined, because—if they do play a role in early sexual onset and other forms of sexual risk behavior in adolescence—they may be malleable intervention targets.

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Abbreviations

DDS	Dynamic Developmental Systems
OYS	Oregon Youth Study
3GS	Three Generational Study

DTSA	discrete-time survival analyses
STIs	sexually transmitted infections

References

1. Campa M, Eckenrode J. Pathways to intergenerational adolescent childbearing in a high-risk sample. *J Marriage Fam.* 2006; 68:558–572.
2. Westrom, L., Eschenbach, D. Pelvic inflammatory disease. In: Holmes, KK, Mardh, P., Sparling, PF., editors. *Sexually Transmitted Diseases*. New York: McGraw Publishers; 1999. p. 783-810.
3. Centers for Disease Control. Sexually Transmitted Disease Surveillance. 2015. Available at <http://www.cdc.gov/std/stats15/std-surveillance-2015-print.pdf2015>
4. Price MN, Hyde JS. Perceived and observed maternal relationship quality predict sexual debut by age 15. *J Youth Adolesc.* 2011; 40:1595–1606. [PubMed: 21384101]
5. Capaldi DM, Crosby L, Stoolmiller M. Predicting the timing of first sexual intercourse for at-risk adolescent males. *Child Dev.* 1996; 67:344–359. [PubMed: 8625717]
6. Elliott, DS., Morse, BJ. Delinquency and Drug Use as Risk Factors in Teenage Sexual Activity. Vol. 21. Newbury Park, CA: Sage; 1989.
7. Fergusson DM, Woodward LJ. Educational, psychosocial, and sexual outcomes of girls with conduct problems in early adolescence. *J Child Psychol Psychiatry.* 2000; 41:779–792. [PubMed: 11039690]
8. Lehr ST, Demi AS, DiIorio C, Factiveau J. Predictors of father-son communication about sexuality. *J Sex Res.* 2005; 42:119–129. [PubMed: 16123842]
9. Elkington KS, Belmonte K, Latack JA, et al. An exploration of family and juvenile justice systems to reduce youth HIV/STI risk. *J Res Adolesc.* 2015; 25:700–716. [PubMed: 26539022]
10. Ellis BJ, Schlomer GL, Tilley EH, Butle EA. Impact of fathers on risky sexual behavior in daughters: A genetically and environmentally controlled sibling study. *Dev Psychopathol.* 2012; 24:317–332. [PubMed: 22293012]
11. Guilamo-Ramos V, Bouris A, Lee J, et al. Paternal influences on adolescent sexual risk behaviors: A structured literature review. *Pediatr.* 2012; 130:e1313–e1235.
12. Allison, PD. *Event History Analysis: Regression for Longitudinal Event Data*. Sage University Paper Series: Quantitative Applications in the Social Sciences. Beverly Hills, CA: Sage; 1984.
13. Capaldi DM, Kim HK, Owen LD. Romantic partners' influence on men's likelihood of arrest in early adulthood. *Criminology.* 2008; 46:401–433.
14. Capaldi, DM., Kim, HK., Pears, KC. The association between partner violence and child maltreatment: A common conceptual framework. In: Whitaker, D., Lutzker, J., editors. *Preventing Partner Violence: Research and Evidence-Based Intervention Strategies*. Washington, DC: American Psychological Association; 2009. p. 93-111.
15. Baltes, PB. Life-span developmental psychology: Observations on history and theory revisited. In: Lerner, RM., editor. *Developmental Psychology: Historical and Philosophical Perspectives*. Hillsdale, NJ: Lawrence Erlbaum; 1983. p. 79-11.
16. Elder, GH, Jr. Perspectives on the life course. In: Elder, GH., Jr, editor. *Life Course Dynamics: Trajectories and Transitions*. Ithaca, NY: Cornell University Press; 1985. p. 23-49.
17. Capaldi DM, Stoolmiller M, Kim HK, Yoerger K. Growth in alcohol use in at-risk adolescent boys: Two-part random effects prediction models drug and alcohol dependence. *Drug Alcohol Depend.* 2009; 105:109–117. [PubMed: 19625141]
18. Kerr DCR, Tiberio SS, Capaldi DM. Contextual risks linking parents' adolescent marijuana use to offspring onset. *Drug Alcohol Depend.* 2015; 154:222–228. [PubMed: 26166667]
19. Elkington KS, Bauermeister JA, Zimmerman MA. Do parents and peers matter? a prospective socio-ecological examination of substance use and sexual risk among African American youth. *J Adolesc.* 2011; 34:1035–1047. [PubMed: 21159374]

20. Guo J, Chung I-J, Hicks KA, Hawkins JD, Catalano RF, Abbott RD. Developmental relationships between adolescent substance use and risky sexual behavior in young adulthood. *J Adolesc Health*. 2002; 31:354–362. [PubMed: 12359381]
21. Rosenthal DA, Smith AM. Adolescent sexual timetables. *J Youth Adolesc*. 1997; 26:619–636.
22. Dittus PJ, Jaccard J, Gordon VV. The impact of African American fathers on adolescent sexual behavior. *J Youth Adolesc*. 1997; 26:445–465.
23. Cavazos-Rehg PA, Spitznagel EL, Bucholz KK, et al. Predictors of sexual debut at age 16 or younger. *Arch Sex Behav*. 2010; 39:664–673. [PubMed: 18846417]
24. Wilson EK, Dalberth B, Koo H. We're the heroes!': Fathers' perspectives on their role in protecting their preteenage children from sexual risk. *Perspect Sex Reprod Health*. 2010; 42:117–124. [PubMed: 20618751]
25. Kerr DCR, Capaldi DM, Pears KC, Owen LD. Intergenerational influences on early alcohol use: Independence from the problem behavior pathway. *Dev Psychopathol*. 2012; 24:889–906. [PubMed: 22781861]
26. French D, Dishion TJ. Predictors of early initiation of sexual intercourse among high-risk adolescents. *J Early Adolesc*. 2003; 23:1–21.
27. Jessor R, Costa F, Jessor L, Donovan JE. Time of first intercourse: A prospective study. *J Pers Soc Psychol*. 1983; 44:608–628.
28. Dishion TJ, Spracklen KM, Andrews DW, Patterson GR. Deviancy training in male adolescent friendships. *Behav Ther*. 1996; 27:373–390.
29. Santelli JS, Lindberg LD, Abma J, McNeely CS, Resnick M. Adolescent sexual behavior: Estimates and trends from four nationally representative surveys. *Fam Plan Perspect*. 2000; 32:156–165.
30. Boekeloo BO, Howard DE. Oral sexual experience among young adolescents receiving general health examinations. *Am J Health Behav*. 2002; 26:306–314. [PubMed: 12081363]
31. Edwards S, Carne C. Oral sex and transmission of non-viral STIs. *Sex Transm Infect*. 1998; 74:95–100. [PubMed: 9634339]
32. Hawkins DA. Oral sex and HIV transmission. *Sex Transm Infect*. 2001; 74:307–308.
33. Hollingshead, AB. Four Factor Index of Social Status. Department of Sociology, Yale University; New Haven, CT: 1975. Unpublished manuscript
34. Capaldi, DM., Metzler, C., Ary, D., Noell, J. Sex Survey. OSLC; 10 Shelton McMurphey Blvd., Eugene, OR 97401: 1991. p. 1-6. Unpublished instrument
35. Pears KC, Capaldi DM. Intergenerational transmission of abuse: a two-generation, prospective study of an at-risk sample. *Child Abuse and Negl*. 2001; 25:1439–1461.
36. Muthén, LK., Muthén, BO. Mplus User's Guide. 7. Los Angeles, CA: Muthén & Muthén; 1998–2012.
37. MacKinnon, DP. Introduction to statistical mediation analysis. New York, NY: Erlbaum; 2008.
38. Botvin GJ, Griffin KW. Life skills training: preventing substance misuse by enhancing individual and social competence. *New Dir Youth Dev*. 2014; 142:57–65.
39. Kellam SG, Wang W, Mackenzie ACL, et al. The impact of the good behavior game, a universal classroom-based prevention intervention in first and second grades, on high-risk sexual behaviors and drug abuse and dependence disorders into young adulthood. *Prev Sci*. 2014; 15:S6–S18. [PubMed: 23070695]

Implications and Contribution

A prospective intergenerational association of fathers' age of onset of sexual intercourse with their offspring's age of sexual onset was found. Effects of parental and peer sexual teasing and child alcohol and marijuana use onset were also tested, but only child alcohol use onset mediated the intergenerational association.

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Figure 1.

Discrete time survival to onset of sexual behaviors.

Note. Numbers denote $b(se)$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 1

Sexual and substance use onset for all children and by gender

	Children	Boys	Girls
Sample size (<i>n</i>)	176	81	95
Adolescent sexual onset (<i>n</i> who onset / <i>n</i> at-risk to onset, % who onset)			
Ages 11–12 years	1/176 (1%)	1/81 (1%)	0/95 (0%)
Ages 13–14 years	15/142 (11%)	10/62 (16%)	5/80 (6%)
Ages 15–16 years	41/95 (43%)	21/42 (50%)	20/53 (38%)
Ages 17–18 years	13/24 (54%)	6/9 (67%)	7/15 (47%)
Alcohol use onset prior to or concurrent with sexual onset	45/176 (26%)	25/81 (31%)	20/95 (21%)
Marijuana use onset prior to or concurrent with sexual onset	39/176 (22%)	22/81 (27%)	17/95 (18%)

Table 2

Direct effects survival analysis models

Panel I: Association of each predictor with child age of sexual onset (run in separate models)				
	Estimate	SE	Est./SE	p-value
Father age of onset of intercourse	−0.354	0.179	−1.979	0.048
Parent sexual teasing	0.365	0.166	2.202	0.028
Peers sexual teasing	−0.224	0.148	−1.515	0.130
Child use of alcohol	1.703	0.327	5.212	<0.001
Child use of marijuana	1.771	0.329	5.375	<0.001
Child is male	0.751	0.329	2.285	0.022
Panel II: Prediction to child age of sexual onset from both father age of onset and parental sexual teasing				
Father age of onset of intercourse	−0.371	0.188	−1.974	0.048
Parent sexual teasing	0.321	0.166	1.936	0.053
Child is male	0.571	0.354	1.610	0.107