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 years to 17–18 years. In addition, 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) before 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 of 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 that 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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Sexual risk behaviors in adolescence expose youth to risk for early parenthood and sexually transmitted infections (STIs) [1,2]. According to the 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, Elkinghton 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 youths. 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 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 six were longitudinal and thus had adequate designs for examining prediction from risk factors. Of these, only three predicted to sexual onset, and all 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 [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 Dynamic Developmental Systems approach builds on lifespan approaches emphasizing the interaction between the individual’s prior dispositions and learning and the environments in which she/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]. Mid-adolescents 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]. Furthermore, 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 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 effect 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 years 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 at elevated risk for earlier sexual onset.
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