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Effects of Fathers on Adolescent Daughters' Frequency of Substance Use and Risky Sexual Behavior

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This research: (1) implements a genetically informed design to examine the effects of fathers' presence–absence and quality of behavior during childhood/adolescence on daughters' frequency of substance use during adolescence; and (2) tests substance use frequency as mediating the relation between paternal behavior and daughters' sexual risk taking. Participants were 223 sister dyads from divorced/separated biological families. Sisters' developmental exposure to socially deviant paternal behavior predicted their frequency of tobacco, alcohol, and cannabis (TAC) use. Older sisters who co-resided with fathers who were more (vs. less) socially deviant reported more frequent TAC use during adolescence. More frequent TAC use predicted more risky sexual behavior for these daughters. No effects were found for younger sisters, who spent less time living with their fathers.

In modern contexts, the human developmental period of adolescence has become nearly synonymous with behavioral risk taking. Substance use and unprotected sex, for instance, constitute some of the most serious challenges to the health and well-being of American teenagers (Steinberg, 2015). Although such behaviors generally are considered normative during this time, there is wide variability in the extent to which individuals participate in these activities, and those who participate more tend to experience more physical and mental health problems, as well as higher rates of teen pregnancy and school dropout (e.g., Chassin, Hussong, & Beltran, 2009; Tapert, Aarons, Sedlar, & Brown, 2001; Trim, Meehan, King, & Chassin, 2007). Given the emotional and financial costs of these outcomes for teens, their families, and society, it is critical to understand the factors that promote risky behavior among youth.

A vast empirical literature documents statistical links between family contexts and adolescent risk taking in Western populations (e.g., Breivik, Olweus, & Endersen, 2009; Bronte-Tinkew, Moore, & Carrano, 2006; Coley, Votruba-Drzal, & Schindler, 2008; Doom, Vanzomeren-Dohm, & Simpson, 2016; Hartman, Sung, Simpson, Schlomer, & Belsky, 2018; Hemovich, Lac, & Crano, 2011). Within this broad literature, specific associations have been found between low paternal investment (e.g., father absence; harsh, conflictual, or disengaged fathering) and adolescent daughters' risky sexual behavior (Coley, Votruba-Drzal, & Schindler, 2009; Ellis et al., 2003; Ellis, Schlomer, Tilley, & Butler, 2012; James, Ellis, Schlomer, & Garber, 2012). According to paternal investment theory (PIT; Draper & Harpending, 1982; Ellis, 2004; Ellis et al., 2012), an evolutionary-developmental model of fathering, such associations reflect a unique effect of fathers on daughters' relationships with men. Paternal investment is posited to forecast the investment that daughters (and their future offspring) are likely to receive from male relationship partners in adulthood and to guide daughters' sexual and reproductive behaviors accordingly (Ellis et al., 2003). Because low paternal investment suggests that male investment in relationships will be minimal, this perspective predicts that such developmental exposures will shift daughters toward

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sexual behaviors considered to be high risk. This general prediction has received empirical support across a variety of methods and outcomes (e.g., Coley et al., 2009; DelPriore, Proffitt Leyva, Ellis, & Hill, 2018; Ellis et al., 2012; James et al., 2012).

In the following, we extend this body of work to consider the effects of fathers on daughters' frequency of substance (alcohol and/or drug) use, behavior linked with variation in family environments, on one hand, and sexual risk taking, on the other (e.g., Bronte-Tinkew et al., 2006; Bryan, Ray, & Cooper, 2007; Doom et al., 2016; Ritchwood, Ford, DeCoster, Sutton, & Lochman, 2015; Timmermans, van Lier, & Koot, 2008). The current study uses a genetically informed and environmentally controlled research method—the differential sibling-exposure design (described below)—to test how daughters' developmental experiences with their fathers affect daughters' own frequency of substance use during adolescence, independent of the many risk and protective factors that vary across families. This method can assess the relative effects of father presence–absence versus paternal behavior quality, as well as amounts of these exposures, on daughters. We also test whether more frequent substance use promotes daughters' risky sexual behavior in the context of low paternal investment.

Paternal Investment, Substance Use, and Risky Sexual Behavior in Adolescence

Paternal investment theory posits unique effects of experiences with fathers in shaping daughters' sexual and reproductive behaviors in addition to, or beyond, genetic transmission of these behaviors (see, e.g., McHale, Bissell, & Kim, 2009; Zietsch, Verweij, Bailey, Wright, & Martin, 2010). Specifically, PIT proposes that low paternal investment prompts psychosocial changes in daughters that promote short-term mating behaviors (e.g., more sexual risk taking), which function to increase daughters' reproductive success when long-term male investment is not reliably available. Such effects of fathers are predicted to be independent of the effects of mothers and qualitatively different for daughters compared to sons. Although PIT was originally developed in relation to father presence–absence, more recent theoretical refinements (e.g., Ellis, 2004) propose that paternal investment *quality* may be a more important influence on daughters' sexual behavior than is the father's mere presence or absence. This focus on quality has been supported by empirical work designed to test for the relative effects of paternal presence–absence versus variation in the quality of paternal behavior on daughters' outcomes (e.g., DelPriore, Schlomer, & Ellis, 2017; Ellis et al., 2012). This research found that when accounting for environmental and genetic risk factors that vary across families, low-quality father–daughter relationships was a stronger predictor of daughters' risky sexual behavior than was father absence, per se. Further, quality of fathering was found to exert its strongest influence on daughters' sexual behavior, for better and for worse, when this exposure was prolonged.

This theoretical perspective resides within a vast empirical literature illustrating a myriad of ways that parental behavior could promote or deter comorbid forms of risk taking among adolescents. For instance, low parental warmth and involvement predict reduced parental monitoring and thereby increased susceptibility to peer influence and more risk taking behaviors, including (though not limited to) substance use (e.g., Breivik et al., 2009; Bronte-Tinkew et al., 2006; DelPriore et al., 2017; Dishion, Ha, & Véronneau, 2012; Hemovich et al., 2011). It is well established that adolescents who regularly use or abuse substances also take more sexual risks, including becoming sexually active at younger ages, having more sexual partners, and being more likely to have unprotected sex and contract sexually transmitted infections (STIs) compared to those who use substances less frequently (e.g., Ritchwood et al., 2015; Tapert et al., 2001). There are many reasons why substance use and risky sexual behavior might co-occur. The disinhibiting effects of drugs and alcohol can impair judgment and decrease the perceived risks (i.e., pregnancy and STIs) associated with having unprotected sex (e.g., Coleman & Cater, 2005; Dir et al., 2018; Ritchwood et al., 2015). At the same time, engagement in casual sexual behavior can encourage substance use in an attempt to enhance the sensations of those experiences (Bryan et al., 2007; Hendershot, Magnan, & Bryan, 2010). Taken together with PIT, the extant literature suggests that low paternal investment may increase daughters' sexual risk taking by increasing their use of drugs and/or alcohol.

Here, we extend the PIT framework to examine the effects of fathers on adolescent daughters' frequency of substance use, behavior known to promote sexual risk taking (as noted above). From a functional perspective, daughters' use of drugs and alcohol may facilitate the short-term and/or risky sexual behavior commonly observed in contexts characterized by low male investment. This view is supported by research finding stronger relationships between substance use and risky sexual behavior for female than for male adolescents (Bryan et al., 2007; Ritchwood et al., 2015), perhaps due to the higher costs of unprotected sex for females compared to males.

Genetic Confounds and the Differential Sibling-Exposure Design

Though social contexts (i.e., environmental factors) are known to predict varied forms of risk taking among adolescents, observed associations between substance use and risky sexual behavior also may be explained by genetic third variables. Genetic variants linked with impulsivity and risk taking have been identified among individuals (e.g., Cleveland et al., 2018; Kendler et al., 2012; Kreek, Nielsen, Butelman, & LaForge, 2005). At the population level, behavioral genetic studies indicate considerable heritability of both adolescent substance use and risky sexual behavior (McGue, Elkins, & Iacono, 2000; McHale et al., 2009).

It is important to note, however, that such genetic influences are unlikely to unfold independent of environmental ones. For example, a parent with mental health issues (including, but not limited to, substance abuse) is likely to transmit this genetic risk to their biological child while also affecting the social (home) environment to which that child is exposed, thereby creating a "double dose" of risk (Blazei, Iacono, & McGue, 2008; Jaffee, Moffitt, Caspi, & Taylor, 2003). Further, genetic influences can moderate the effects of family environments on children's development (e.g., Dick et al., 2007).

Despite abundant research linking adolescent substance use and risky sexual behavior, and on the social contexts that predict both behaviors, relatively little research has examined the nature of these associations using designs that account for known genetic influences. We address this gap in the literature by testing our main predictions in the context of a differential sibling-exposure design (for an in-depth discussion of this design, see Ellis et al., 2012). This quasi-experimental approach accounts for genetic and environmental risks that naturally vary between families by comparing the outcomes of full biological sister pairs who experienced the divorce or separation of their parents while growing up together, followed by primary co-residence with the mother. The social and physical environments of sisters reared within these families are matched in important ways. For example, these sisters grow up together in the same neighborhood and with the same socioeconomic status. Further, a genetic tendency toward risk taking is randomized across birth order: Older sisters, as a group, are no more or less likely to inherit a given genetic variant than are younger sisters, as a group. The primary systematic difference between older and younger sisters in these families is their differential exposure to the fathers' presence–absence and behavior. By virtue of being younger when the parental divorce or separation occurred, younger sisters receive a larger dose of exposure to father absence while growing up compared to their older sisters. Therefore, if father absence has an effect on daughters, then younger sisters (who experience greater father absence) should engage in more risk taking than their older sisters (who experience less father absence). On the other hand, older sisters receive a larger dose of exposure to their fathers' presence and behavior while growing up compared to their younger sisters. If the quality of the father's behavior (e.g., warm, supportive as contrasted with harsh, deviant behaviors) has an effect on daughters, then these effects should be stronger for older versus younger sisters (for better and for worse).

THE CURRENT STUDY

The current study tests whether developmental exposure to fathers (their presence–absence and the quality of their behavior) affects daughters' frequency of substance use during adolescence, and whether frequent substance use promotes daughters' risky sexual behavior in the context of low paternal investment. We assessed *quality of paternal behavior* in two ways (see DelPriore, Shakiba, Schlomer, Hill, & Ellis, 2019): (1) the father's socially deviant behavior inside and outside of the home (e.g., issues with anger and violence); and (2) the father's warmth and direct involvement in his daughters' lives. We assessed daughters' *frequency of substance use* by asking women to report on their frequency of use of tobacco, alcohol, and cannabis (TAC) products when they were high school age, as well as their use of harder, illicit drugs (e.g., cocaine, heroin). Finally, to assess daughters' *risky sexual behavior*, we asked women to report the number of sexual partners they had prior to age 19 as well as the frequency with which they engaged in different high-risk sexual behaviors (e.g., having unprotected sex) during high school (Ellis et al., 2012).

As noted earlier, recent research employing the differential sibling-exposure design has found that it is not fathers' presence or absence (i.e., time spent living with/without one's father), per se, but rather the interaction between father presence–absence and quality of fathering, that drives behavioral differences commonly observed among girls between families (e.g., DelPriore et al., 2017; Ellis et al., 2012; Tither & Ellis, 2008). Therefore, we predicted that differences in sisters' frequency of substance use would be influenced by differential exposure within families to variation in paternal behavior quality during childhood/adolescence. Specifically, the effects of lower quality paternal behavior on more frequent substance use in daughters should be stronger for older (vs. younger) sisters given

older sisters' longer co-residence with the father. Based on previous research linking substance use and risky sexual behavior (Ritchwood et al., 2015), we predicted that frequency of substance use would statistically mediate the relationship between lower quality paternal behavior and higher levels of adolescent sexual risk taking in daughters. Based on past research using the differential sibling-exposure design, we did not predict a main effect within families of differential exposure to father presence–absence (independent of paternal behavior quality) on sisters' frequency of substance use.

METHOD

Procedures

This study was reviewed and approved by the University of Arizona Institutional Review Board (protocol number: 1300000470; title: The impact of fathers on risky sexual behavior and decision-making in daughters).

Recruitment. Participants were recruited online via Craigslist (www.craigslist.org) and Amazon's Mechanical Turk (www.mturk.com). We targeted full biological sister pairs who: (1) were separated by a minimum age gap of four years; (2) were between ages 18 and 36; and (3) whose parents divorced/separated[1] while the younger sister was age 14 or under. Prospective participants contacted the research team via telephone to confirm their eligibility. After passing this oral screening, each sister was electronically mailed a link to the online Qualtrics (Provo, UT, USA) survey. (See DelPriore et al., 2019, for additional details regarding recruitment and prescreening.)

Participants. The final analytic sample[2] included adult sister pairs from 223 families (N = 446 individuals). When they completed the study, older sisters were 29.50 years (SD = 3.94, range 22–38) and younger sisters were 23.85 years (SD = 3.78, range 18–32), on average. The average age difference between sisters was calculated to be 5.65 years (SD = 1.78, min = 3.45, max = 12.78)[3]. When their parents divorced or separated, older sisters were 11.34 years (SD = 4.13; range 3–23) and younger sisters were 5.74 years (SD = 3.71; range 0–14), on average. Most participants self-identified as non-Hispanic White (66.1%), Hispanic/Latinx (11.7%), non-Hispanic Black (11.0%), multiracial (9.9%), or Asian (1.3%). During the telephone screening, 53% of participants reported being poor or lower middle class while growing up, 36% middle class, and 11% upper middle class/wealthy. (See DelPriore et al., 2019, for additional details regarding the screening procedures and participant demographics.)

Measures

We assessed the quality of paternal behavior experienced by daughters during childhood/adolescence, as well as their frequency of substance use and risky sexual behavior in adolescence. (The measures assessing quality of paternal behavior are the same as those reported in DelPriore et al., 2019, with scoring modifications made herein based on reviewer feedback.) We report on all measures directly relevant to testing the current hypothesis.

Quality of paternal behavior (childhood/adolescence). In addition to assessing paternal presence–absence from the home (in years), we included measures of (1) paternal social deviance; and (2) the father's warmth and involvement in his daughters' lives.

Paternal social deviance. Given established links between exposure to deviant or antisocial paternal behavior and children's negative outcomes (e.g., Coley, Carrano, & Lewin-Bizan, 2011; Jaffee et al., 2003), we measured daughters' perceptions of their father's social deviance/psychopathology[4] (i.e., mild and severe behavioral and mental health problems; see e.g., DelPriore et al., 2018; DelPriore et al., 2019; Tither & Ellis, 2008). Participants rated

[1]The original differential sibling-exposure design (Ellis et al., 2012; Tither & Ellis, 2008) controlled for birth order and spacing by comparing pairs of sisters from divorced/separated biological families with pairs of sisters from intact biological families (whose amount of differential exposure to the father is zero). Because past research has failed to find differences in the effects of fathers on sisters within intact families (e.g., DelPriore et al., 2017; Ellis et al., 2012; Tither & Ellis, 2008), the current project did not include this comparison group and instead recruited a larger sample of divorced/separated families.

[2]The analytic sample is the same as reported in DelPriore et al., 2019. Further, some of the older sisters (and their reports of paternal social deviance and harsh-coercive fathering) were included in a follow-up experiment reported in DelPriore et al., 2018.

[3]Though we aimed to recruit sisters ages 18–36 and who were separated by at least a four-year age gap, during prescreening the researchers estimated the ages of potential participants using one sister's age and the age gap between sisters as reported by one of the sisters. This approach resulted in two women outside of the desired age range and sister pairs with less than a four-year age gap being invited to participate in the study.

[4]We did not include a measure of maternal social deviance as the current study was designed to focus on the effects of fathers on daughters' outcomes.

the behavior of their birth father up until they were age 18: e.g., "Did your birth father have trouble with drug abuse?" (*no* = 0; *yes* = 1; *yes, a lot* = 2; *don't know* = missing). We averaged participants' responses across the 23 items to produce a composite score (α = .92; M = .42, SD = 0.45; min = 0, max = 1.93). Mean scores were .41 (SD = 0.45) for older sisters and .42 (SD = 0.45) for younger sisters. There was a strong positive correlation (r = .69, $p < .001$) between older and younger sisters' reports of paternal social deviance within families.

Father warmth/involvement. We also assessed the father's warmth and involvement while daughters were growing up (following DelPriore et al., 2019). First, we measured fathers' direct involvement (Dick, 2004) by asking participants to rate how often their father was involved in various activities during their first 16 years of life (e.g., "My father read to me as a child"; 1 = *never*; 5 = *always*). We averaged these eight items to create a composite score (α = .91; M = 2.33, SD = 1.04; min = 1, max = 5), with higher scores reflecting more direct father involvement in daughters' lives. Mean scores were 2.45 (SD = 1.05) for older sisters and 2.20 (SD = 1.01) for younger sisters.

Second, we measured the quality of the relationships between fathers and daughters during childhood and adolescence (Ellis et al., 2012). This measure included items assessing warm-supportive (Parker, Tupling, & Brown, 1979) and harsh-coercive (Jouriles, Mehta, McDonald, & Francis, 1997; Straus, 1979) fathering. Participants were instructed to rate their relationship with their father during the first 16 years of life (e.g., "My father spoke to me with a warm and friendly voice"; 0 = *very unlike*, 3 = *very like*). The ratings were averaged to produce measures of warm-supportive (12 items; M = 2.53, SD = 0.84; α = .94) and harsh-coercive (4 items; M = 1.73, SD = 0.87; α = .86) fathering. The warm-supportive and harsh-coercive (reverse coded) fathering scores were standardized and then averaged to create a composite measure of father–daughter relationship quality (r = .58; M = .00, SD = 0.89; min = −2.21, max = 1.29). Mean scores were −.06 (SD = 0.90) for older and .06 (SD = 0.87) for younger sisters.

The measures of father involvement and father–daughter relationship quality (r = .56, $p < .001$) were standardized and averaged into a final measure of *father warmth/involvement*. This score was grand-mean centered (older sisters: M = .03, SD = 1.02; younger sisters: M = −.03, SD = 0.98), and higher scores correspond to more warm/involved fathering. Older and younger sisters' reports were positively correlated (r = .57, $p < .001$).

Participants also rated their mothers (i.e., maternal involvement and relationship quality) on analogous scales (M = .00, SD = 0.88; min = −.2.81, max = 1.36). The final *mother warmth/involvement* score was standardized. The mean was −.07 (SD = 1.02) for older and .07 (SD = 0.98) for younger sisters. Similar to the measure of father warmth/involvement, sisters' scores were positively correlated (r = .49, $p < .001$) within families.

Frequency of substance use (adolescence). To assess daughters' substance use during adolescence, participants rated the frequency with which they used different types of substances when they were high school age (ages 14–17). First, women reported on their frequency of use of TAC, as research suggests strong clustering of use of these substances (e.g., Passey, Sanson-Fisher, D'Este, & Stirling, 2014). Specifically, we asked women to rate how often they: (1) used tobacco products (such as snuff, chew, or cigarettes); (2) drank alcohol products (such as beer, wine, wine coolers, mixed drinks, and whiskey); and (3) used cannabis products (such as marijuana or hashish). Participants' ratings were made on the following scale: 0 = *Never*; 1 = *Tried once*; 2 = *Occasionally*; 3 = *1–2 times a week*; 4 = *3–5 times a week*; 5 = *Daily*. A composite score was computed by averaging the three items (α = .82; M = 1.73, SD = 1.45; min = 0, max = 5; e.g., Kim-Spoon et al., 2019). Higher scores reflect more frequent *TAC use* during adolescence. The composite measure was standardized, and mean scores were −.02 (SD = 1.01) for older and .02 (SD = 0.99) for younger sisters. These scores were moderately correlated (r = .25, $p < .001$) within families. (This study included items assessing school, family, and legal problems that resulted from daughters' alcohol and marijuana use. Because this measure was not directly relevant to testing the central hypothesis, analysis of these items is reported in Appendix S1.)

Women also reported on their hard drug use (e.g., Timmermans et al., 2008) during the same time period. Specifically, they rated how often they used: (1) crack; (2) cocaine; (3) heroin; (4) speedball; (5) speed; (6) opium; (7) LSD; (8) XTC; (9) mescaline; (10) peyote; and (11) mushrooms. Ratings were made on the same scale as TAC use. A composite score was computed by averaging the 11 items (α = .84; M = .15, SD = 0.34; min = 0, max = 2.55). This score correlated with women's frequency of TAC use (r = .54, $p < .001$) and was

moderately correlated (r = .20, p = .003) among sisters within families. However, there was not sufficient variability on this measure to allow for meaningful analysis. (That is, a majority [68%] of women indicated that they "never" [0] used any of the 11 substances, as contrasted with 17% who reported no TAC use.) Therefore, we focused the analyses on daughters' frequency of TAC use.

Risky sexual behavior (adolescence). We assessed risky sexual behavior following procedures implemented by Ellis et al. (2012). Specifically, we presented two measures indexing daughters' sexual risk taking during adolescence. The first measure asked women to report the number of different partners they had sex with before age 19. To reduce the influence of outliers and skew produced by open-ended questions about numbers of sexual partners (e.g., Penke & Asendorpf, 2008), these responses were recoded: 0 = *none*; 1 = *1 to 2*; 2 = *3 to 6*; 3 = *7 to 15*; and 4 = *16 and above*. On the recoded scale, the average score was 1.52 (SD = 1.10). Mean scores were 1.48 (SD = 1.07) for older sisters and 1.56 (SD = 1.13) for younger sisters.

The second scale assessed the frequency with which the sisters engaged in six high-risk sexual behaviors before age 19: unprotected sex; using alcohol and/or drugs prior to or in conjunction with participation in sexual activity; sexual intercourse with an injection drug using partner; sexual intercourse with someone who was physically forceful, hurting, or threatening; engaging in concurrent sexual relationships with different partners; and receiving money, drugs, or a place to stay in exchange for sexual activity with someone (0 = *none*, 1 = *once or twice*, 2 = *three times or more*). These items were standardized and averaged to form a composite measure of high-risk sexual behavior (α = .64; M = .0003, SD = 0.60, min = −.49, max = 2.88). Mean scores were −.03 (SD = .58) for older and .03 (SD = 0.62) for younger sisters.

To create the aggregate measure of risky sexual behavior[5], the recoded number of sexual partners was standardized and averaged with the amount of high-risk sexual behavior (r = .69). Graphical inspection of this composite score revealed seven outliers, and these high scores were truncated. This final measure was standardized to achieve grand-mean centering. Mean levels of risky sexual behavior were −.04 (SD = 0.97) for older and .04 (SD = 1.03) for younger sisters. Sisters' scores were moderately correlated (r = .20, p = .002).

Data Analysis

Our analytic approach followed recent work implementing the differential sibling-exposure design (e.g., DelPriore et al., 2017, 2019; Ellis et al., 2012). That is, we first conducted a series of multilevel regression models that account for nonindependence in data from sisters by specifying within-dyad correlated residuals (Campbell & Kashy, 2002; Kenny, Kashy, & Cook, 2006). These models included birth order (coded: 0 = *older sister*; 1 = *younger sister*) to compare sisters within families. We used multilevel modeling to test a form of mixed ANOVA that: (1) allows for both categorical and continuous predictors; (2) models within-family effects (i.e., birth order; quality of paternal behavior), between-family effects (i.e., differential exposure to the father between sisters), and their interactions; and (3) adjusts for measurement error (Sayer & Klute, 2005). The *lme* function in the R (version 4.0.0) *nlme* package was used to specify a compound symmetric residual covariance structure for siblings nested within families. We tested our hypotheses using fixed effect models similar to standard multiple regression.

RESULTS

All continuous variables were standardized (M = 0, SD = 1) prior to testing the multilevel regression models. (See Table 1 for bivariate correlations for older and younger sisters and Table 2 for model results.) First, we tested for effects of father presence–absence on daughters' frequency of TAC use during adolescence, independent of paternal behavior quality. Next, we tested if variation in the quality of paternal behavior was a stronger predictor of older (vs. younger) sisters' frequency of TAC use, given their greater exposure to their fathers while growing up. In each case, we used birth order and age gap[6] to index differential exposure

[5]The current analysis focused on a measure of risky sexual behavior used in previous research (e.g., DelPriore et al., 2017; Ellis et al., 2012). Although this study included additional information about women's sexual history (e.g., sexual debut, sexually transmitted infections, pregnancies), we did not analyze these outcomes for the current work.

[6]In all but two families, the amount of differential exposure to fathers was equivalent to the age gap between sisters. In the two cases in which these values were highly discrepant, the amount of differential exposure was recoded. The measures of sisters' age gap and their amount of differential exposure to their father were nearly perfectly correlated (r = .98, p < .001). We analyzed the recoded differential exposure variable within our main models.

TABLE 1
Means, Ranges, and Correlations Between Parental Behavior (Quality and Amount of Exposure), Frequency of Substance Use, and Risky Sexual Behavior for Older and Younger Sisters

Measures		*1*	*2*	*3*	*4*	*5*	*6*	*7*
	M *(range)*	*−.01 (−0.93 to 3.40)*	*.03 (−2.12 to 2.18)*	*−.07 (−3.19 to 1.54)*	*−.02 (−1.19 to 2.26)*	*−.01 (−0.44 to 5.11)*	*−.04 (−1.29 to 2.73)*	*.00 (−1.23 to 4.06)*
1. Paternal social deviance	.01 (−0.93 to 3.04)	–	−.55***	−.12	.33***	.13	.19**	−.03
2. Father warmth/ involvement	-.03 (−2.12 to 2.27)	−.54***	–	.27***	−.28***	−.18**	−.15*	.05
3. Mother warmth/ involvement	.07 (−2.82 to 1.54)	−.03	.01	–	.04	.01	−.02	−.08
4. Frequency of TAC use	.02 (−1.19 to 2.26)	.05	−.17**	−.03	–	.51***	.66***	.003
5. Frequency of hard drug use	.01 (−0.44 to 6.99)	.08	−.09	−.06	.57***	–	.42***	.08
6. Risky sexual behavior	.04 (−1.29 to 2.60)	.06	−.10	−.11	.67***	.44***	–	.01
7. Differential exposure to fathers	.00 (−1.23 to 4.06)	.01	.03	−.13*	−.01	−.04	−.01	–

Note. Scores are standardized ($M = 0$, $SD = 1$). Older sisters (ns = 221–223) are above the diagonal, and younger sisters (ns = 218–223) are below the diagonal. Significance tests are two-tailed.
*$p \leq .05$; **$p \leq .01$; ***$p \leq .001$.

TABLE 2
Parameter Estimates (and Standard Errors) for Models Predicting Daughters' Frequency of Tobacco, Alcohol, and Cannabis (TAC) Use

	Parameter estimates (SE)					
Predictors	*Father absence–presence*		*Paternal social deviance*		*Father warmth/involvement*	
Main Effects						
BthOrd	.04 (.08)	.04 (.08)	.02 (.08)	.02 (.08)	.03 (.08)	.03 (.08)
PaternalQual			.31 (.06)***	.31 (.06)***	−.29 (.06)***	−.29 (.06)***
Exposure		.004 (.07)		.01 (.07)		.02 (.07)
Two-way interactions						
PaternalQual × BthOrd			−.26 (.08)**	−.26 (.08)**	.10 (.08)	.10 (.08)
PaternalQual × Exposure				.01 (.06)		−.02 (.05)
BthOrd × Exposure		−.02 (.08)		−.04 (.08)		−.03 (.08)
Three-way interaction						
PaternalQual × BthOrd × Exposure			−.02 (.08)		−.07 (.08)	

Note. $N = 223$ sister pairs. Birth order of sisters (0 = older, 1 = younger). All other main effects are continuous and mean centered. BthOrd, birth order of sisters; Exposure, differential exposure to fathers between sisters (in years); PaternalQual, quality of paternal behavior (i.e., paternal social deviance or father warmth/involvement).
*$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.

between sisters. Finally, based on the results of these genetically informed models, we tested for the indirect effects of fathers on daughters' risky sexual behavior through daughters' frequency of TAC use (i.e., moderated mediation).

Exposure to Paternal Presence–Absence

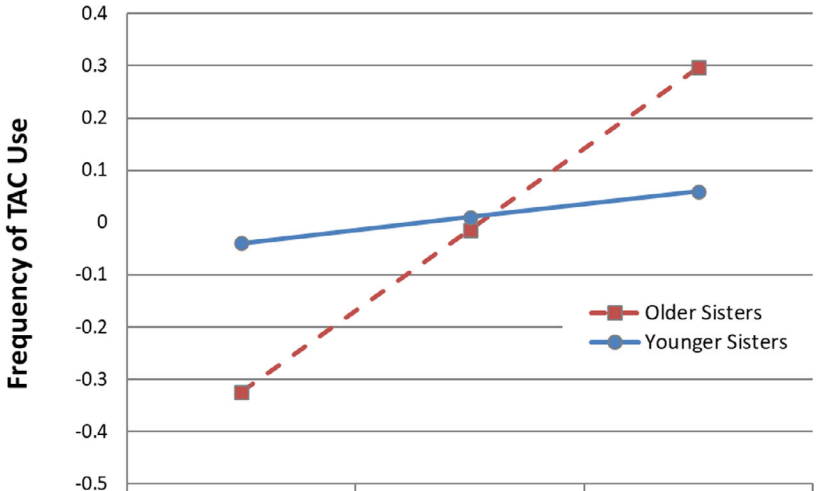
If exposure to biological father absence influences daughters' frequency of substance use, then younger sisters (who experienced more time living in a biologically father absent home) should report more frequent substance use relative to their older sisters. Therefore, we first tested for a main effect of birth order on daughters' frequency of TAC use. There was no significant main effect of birth order ($p = .60$, 95% CI [−0.13, 0.21]). Next, we tested for an interaction between birth order and sisters' amount of differential exposure to father presence–absence (in years) as a finer-grained test of these effects. This two-way interaction was not significant ($p = .84$, 95% CI [−0.18, 0.15]). These results suggest that daughters' frequency of TAC use during adolescence was not meaningfully affected by their fathers' mere presence in, or absence from, the home.

Exposure to Quality of Paternal Behavior

Next, we tested if the effect of father presence–absence on daughters' frequency of substance use is moderated by the *quality* of the father's behavior (e.g., DelPriore et al., 2019; Ellis et al., 2012). Specifically, we first tested two-way interactions between quality of paternal behavior (measured as paternal social deviance and father warmth/involvement) and birth order (older vs. younger) on daughters' TAC use frequency. Then, we tested whether the predicted two-way interaction would be moderated by sisters' amount of differential exposure to the father measured in years (i.e., three-way interactions between the quality of paternal behavior, birth order, and age gap between sisters).

Paternal social deviance. There was a significant two-way interaction[7] between paternal social deviance and birth order, $F(1, 213) = 9.85$, $p = .002$, 95% CI [−0.42, −0.08]. For older sisters, more deviant paternal behavior predicted more frequent TAC use during adolescence, $b = .31$ ($SE = .06$), $t(213) = 4.87$, $p < .001$, 95% CI [0.20, 0.43]. This association was not significant for younger sisters, $b = .05$ ($SE = .06$), $t(213) = .76$, $p = .45$, 95% CI

[7]A traditional Bonferroni correction (alpha divided by the number of tests conducted for a hypothesis) was applied to correct the familywise error rate (e.g., Matsunaga, 2007). We performed two tests of the hypothesis that quality of paternal behavior differentially affects older versus younger sisters' frequency of substance use (including paternal social deviance and father warmth/involvement as predictors). Therefore, to reject the null hypothesis regarding the quality of paternal behavior, the *p*-value for any of these tests should be less than the corrected critical value of .025 (.05/2). Here, the obtained *p*-value (.002) allows us to reject the null hypothesis.



	-1 SD	Mean	+1 SD
	Paternal Social Deviance		
Older Sisters	-0.3248	-0.0138	0.2971
Younger Sisters	-0.0395	0.0101	0.0597

FIGURE 1 The effects of paternal social deviance on daughters' frequency of tobacco, alcohol, and cannabis (TAC) use. The graph displays the predicted values for older and younger sisters' frequency of TAC use at low (1 *SD* below the mean), mean, and high (1 *SD* above the mean) levels of paternal social deviance. [Color figure can be viewed at wileyonlinelibrary.com]

[−0.08, 0.17] (see Figure 1). Further, older sisters who experienced low levels of deviant paternal behavior (1 *SD* below the mean) reported significantly less frequent TAC use relative to their younger sisters, $b = .29$ ($SE = .12$), $t(213) = 2.46$, $p = .01$, 95% CI [0.04, 0.54][8], suggesting a protective effect of such exposures.

Father warmth/involvement. We tested similar models replacing paternal social deviance with the measure of father warmth/involvement. We did not find a two-way interaction between birth order and father warmth/involvement on daughters' frequency of TAC use, $F(1, 218) = 1.37$, $p = .24$, 95% CI [−0.06, 0.26]. There was a main effect of father warmth/involvement across older and younger sisters: daughters with more (vs. less) warm/involved fathers reported less frequent TAC use during adolescence, $b = -.25$ ($SE = .05$), $t(220) = -5.11$, $p < .001$, 95% CI [−0.34, −0.15].

Differential dosage of exposure to quality of paternal behavior (in years). We tested the effects of fathers on daughters' frequency of substance use with a more precise measure of sisters' differential exposure to their father: differences in the number of years that sisters spent with their father in the home (see Footnote 6). The three-way interactions between quality of paternal behavior, birth order, and sisters' amount of differential exposure to the father (in years) were not significant; paternal social deviance: $F(1, 210) = 0.08$, $p = .78$, 95% CI [−0.16, 0.13]; warm/involved fathering: $F(1, 215) = 0.85$, $p = .36$, 95% CI [−0.22, 0.10].

Moderated mediation model. It is theoretically relevant and practically important to understand

[8]The frequency of TAC use variable was positively skewed, a common feature of substance use data (e.g., Atkins, Baldwin, Zheng, Gallop, & Neighbors, 2013). To examine the potential influence of the variable distribution and model specification on the obtained results, we used the *lme4* package in R to fit Poisson and negative binomial regression models to account for the positive skew. The outcome measure was the sum of the three TAC use items. The findings (i.e., the two-way interaction between paternal social deviance and birth order on daughters' TAC use frequency, the significant simple slope for older sisters, and the nonsignificant simple slope for younger sisters) persisted across models, with fit statistics favoring the linear regression model reported herein. Further, the linear and Poisson regression models detected significant differences based on birth order at ±1 *SD*s around the mean on paternal social deviance. However, the negative binomial model only detected this difference at 1 *SD* below the mean (i.e., it did not detect a difference at 1 *SD* above the mean). Therefore, we only interpret the effect of birth order at −1 *SD* on paternal social deviance as significant.

the role that daughters' substance use plays in guiding their sexual risk taking in the context of low paternal investment. Therefore, we tested frequency of substance use as an intervening factor in the relationship between daughters' exposure to low-quality paternal behavior and their risky sexual behavior. Based on the results of the above genetically informed models, we used Hayes' (2017) PROCESS SPSS macro (version 3.1; Model 7) to test the indirect effect of paternal social deviance (*X*; the predictor variable) on daughters' risky sexual behavior (*Y*; the outcome variable) through frequency of TAC use (*M*; the mediator variable). Since the relationship between paternal social deviance and daughters' frequency of TAC use was found to vary across sisters, we included birth order as a moderator (*W*) of this pathway. This allowed us to test whether the proposed mediation effect varied across birth order (i.e., moderated mediation; different indirect effects for older and younger sisters).

This model provided support for moderated mediation, $b = -.19$ ($SE = .07$), 95% CI [−0.31, −0.06] (see Figure 2). Consistent with the genetically informed analyses, the relationship between paternal social deviance and daughters' frequency of TAC use varied across birth order (two-way interaction: $\beta = -.20$ [$b = -.28$, $SE = .09$], $t = -3.02$, $p = .003$, 95% CI [−0.46, −0.10]). In turn, frequency of TAC use predicted risky sexual behavior[9] while controlling for paternal social deviance, $\beta = .66$ ($b = .66$, $SE = .04$), $t = 18.10$, $p < .001$, 95% CI [0.59, 0.74]. Finally, the relationship between paternal social deviance and risky sexual behavior was mediated by frequency of TAC use for older, but not for younger, sisters.[10,11]

Effects of Paternal Social Deviance versus Warmth/Involvement

Despite negative correlations between paternal social deviance and warmth/involvement, it is possible for fathers who engage in socially deviant behaviors to provide high levels of warmth and support to their offspring. Therefore, we tested for unique effects of paternal social deviance while accounting for variation in fathers' warmth/involvement. To this end, we first retested the interaction between paternal social deviance and birth order on daughters' frequency of TAC use while controlling for father warmth/involvement. The main results persisted: 2-way interaction: $F(1, 212) = 9.52$, $p = .002$, 95% CI [−0.41, −0.10]; simple slope for older sisters: $b = .20$ ($SE = .07$), $t(212) = 2.86$, $p = .005$, 95% CI [0.06, 0.35]; nonsignificant simple slope for younger sisters: $b = -.05$ ($SE = .07$), $t(212) = -.72$, $p = .47$, 95% CI [−0.19, 0.08]. We did not find evidence of a two-way interaction between father warmth/involvement and birth order on daughters' frequency of TAC use while controlling for paternal social deviance, however, $F(1, 212) = 2.11$, $p = .15$, 95% CI [−0.06, 0.29]. Finally, we tested the three-way interaction between paternal social deviance, father warmth/involvement, and birth order. This interaction was not significant, $F(1, 209) = 0.26$, $p = .61$, 95% CI [−0.22, 0.13]. These results suggest that the observed within-family effects of daughters' exposures to paternal social deviance operate independent from variation in their fathers' warmth/involvement.

[9]The distribution of the final risky sexual behavior composite score was positively skewed. This skew primarily resulted from the "high-risk sexual behavior" component (rather than the "sexual partner number" component). Therefore, we rescored this component as a count of the different high-risk sexual behaviors in which participants engaged prior to age 19. To this end, we recoded the original six items as binary variables (0 = *never engaged in the behavior prior to age 19*; 1 = *engaged in the behavior prior to age 19*) and summed the responses. We then re-analyzed the pathways in the mediation model that included this outcome (i.e., the *b* and *c* paths) by fitting Poisson and negative binomial regression models. These paths remained significant (*p*s ≤ .01), consistent with the OLS regression paths modeled by PROCESS.

[10]There were two families in which one sister did not respond to the primary outcome measure (frequency of TAC use). We retested our main findings excluding these two families, and the results persisted. These analyses are presented in Appendix S2.

[11]Although alcohol and marijuana are known for lowering inhibitions and impairing judgment (e.g., Bryan, Schmiege, & Magnan, 2012; Coleman & Cater, 2005), the link between tobacco use and disinhibition is typically observed at the individual, rather than the event, level (e.g., Kopstein, Crum, Celentano, & Martin, 2001). Therefore, we retested our main models using a modified dependent measure. This measure focused on daughters' frequency of alcohol and marijuana use during adolescence while excluding their tobacco use. The focal results (i.e., the effects of paternal social deviance on older, but not younger, sisters' frequency of substance use and the corresponding moderated mediation model) replicated using the modified dependent measure. These analyses are presented in Appendix S3.

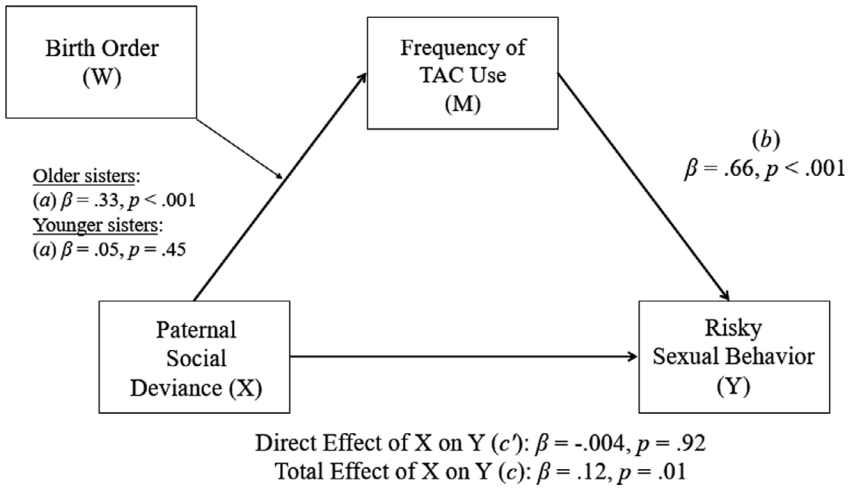


FIGURE 2 Mediation analysis testing the effects of paternal social deviance (*X*) on daughters' risky sexual behavior (*Y*) through their frequency of tobacco, alcohol, and cannabis (TAC) use (*M*). We collected 10,000 bootstrapped samples to generate percentile 95% confidence intervals for each indirect effect. The mediating effect was moderated by birth order (*W*). The indirect effect of *X* on *Y* through *M* was significantly different from zero for older sisters (95% CI [0.14, 0.31]) but not for younger sisters (95% CI [−0.06, 0.13]).

Effects of Quality of Paternal (vs. Maternal) Behavior

Due to established associations between quality of paternal and maternal behavior (e.g., Coley et al., 2009, 2011; Ellis et al., 2012; Simons, Paternite, & Shore, 2001), we also tested the effects of fathers beyond the role of mothers. Specifically, we retested the interaction between paternal social deviance and birth order on daughters' TAC use frequency while controlling for maternal warmth/involvement. The main results were robust to the inclusion of this covariate: 2-way interaction: $F(1, 212) = 10.08$, $p = .002$, 95% CI [−0.44, −0.11]; simple slope for older sisters: $b = .32$ ($SE = .06$), $t(212) = 4.92$, $p < .001$, 95% CI [0.20, 0.44]; nonsignificant simple slope for younger sisters: $b = .05$ ($SE = .06$), $t(212) = .79$, $p = .43$, 95% CI [−0.07, 0.18]. We did not find evidence of a two-way interaction between maternal warmth/involvement and birth order, nor a main effect of maternal warmth/involvement, on daughters' frequency of TAC use while controlling for paternal social deviance, Fs(1, 212) ≤ .15, ps ≥ .69. Finally, we did not find a three-way interaction between maternal warmth/involvement, paternal social deviance, and birth order, $F(1, 209) = 0.05$, $p = .83$, 95% CI [−0.19, 0.14]. Though this work was designed to more rigorously test for the effects of *paternal* behavior on daughters (as noted in Footnote 4), these results suggest a possible unique influence of experiences with fathers (independent of mothers) in driving variation in adolescent daughters' frequency of substance use.

DISCUSSION

Substance use and risky sexual behavior co-occur for many female adolescents (e.g., Ritchwood et al., 2015). In addition to these behaviors influencing each other in a bidirectional fashion, it is likely that some of the same genetic and environmental factors that promote one (risky sexual behavior) also promote the other (substance use). Guided by this logic, and by research and theory linking paternal quality with adolescent daughters' sexual risk taking (e.g., Coley et al., 2009; Ellis et al., 2012), the current study used a within-family design to test for the effects of fathers on daughters' frequency of substance use. We found that daughters exposed to high (vs. low) levels of socially deviant paternal behavior while growing up reported more frequent TAC use during adolescence. This relationship between paternal behavior quality and substance use frequency depended on daughters co-residing with their father throughout much of childhood and into early adolescence (i.e., effects were specific to older sisters in this sample). For these daughters, more frequent TAC use predicted higher engagement in risky sexual behavior during their teenage years. Similar effects were not observed among daughters who spent relatively little time living with their fathers during childhood and

adolescence (i.e., younger sisters in this sample) and thus experienced reduced exposure to their behavior.

Although statistical relationships between family contexts, parental behavior, substance use, and risky sexual behavior regularly appear in the empirical literature, few of these studies have been causally informed (for a review, see, e.g., McLanahan, Tach, & Schneider, 2013; see also DelPriore et al., 2017; Ellis et al., 2012; Ryan, 2015). Therefore, it remains possible that reliably observed associations derive from environmental and genetic risk factors that vary nonrandomly across families. The current study addressed this concern by comparing sisters within biologically divorced/separated families. In such families, the primary systematic difference between sisters is in their exposure to the father (both his presence-absence and behavior). In this context, the current work provided evidence for exposure to the father's socially deviant behavior prompting more frequent substance use among daughters in adolescence. This conclusion was supported by the significant relationship between paternal social deviance and frequency of substance use for older sisters, who received a larger dose of exposure to their fathers' deviant behavior than did their younger sisters. Because our design focused on comparing the outcomes of sisters within families, it is unlikely that these results are attributable to genetic or environmental differences that often exist among daughters between families.

On the other hand, we did not find effects of exposure to biological father absence (vs. presence) on daughters. Given younger sisters' greater average exposure to this context, they should report more frequent substance use compared to their older sisters if father absence had an effect. This possibility was not supported, suggesting that previous links observed between father absence and adolescents' substance use likely are at least partially due to third variable genetic and/or environmental confounds. Instead of supporting causal effects of family structure, the current results align with research suggesting that the quality of the father's behavior while he is in the home, rather than his absence from it, exerts a stronger influence on daughters when genetic and environmental confounds are considered (e.g., DelPriore et al., 2017, 2019; Ellis et al., 2012). This finding also complements research demonstrating strong associations between residential fathers' antisocial behavior and their children's outcomes (e.g., Blazei et al., 2008; Jaffee et al., 2003). The observed effects of paternal social deviance for older sisters extended beyond variation in fathers' warmth and involvement directed toward their daughters. Indeed, the lack of within-family differences in the influence of father warmth/involvement suggests that links between this variable and daughters' substance use frequency may be driven by other between family differences. Although it is unclear why father warmth/involvement did not have the predicted effect on daughters' frequency of substance use, the obtained pattern of results is consistent with research demonstrating effects of fathers' antisocial behavior on children's outcomes beyond their quality of parenting (e.g., warmth, harshness; Coley et al., 2011).

Finally, the hypothesis that fathers exert a unique influence on daughters' sexual and reproductive behavior is a straightforward prediction of our guiding evolutionary-developmental model. The finding that variation in paternal quality predicted older sisters' outcomes independent from (i.e., controlling for) maternal quality is consistent with this view. However, a strict interpretation of PIT does not predict targeted effects of fathers on nonsexual forms of risk taking (e.g., substance use) among daughters. Here, we extended this framework to address the possibility that daughters' substance use in the context of low paternal investment functions to disinhibit psychological barriers that often discourage risky sexual behavior among females given its potential high cost (e.g., unintended pregnancy and STI risk). This perspective fits within a vast developmental literature linking adolescents' risky sexual behavior with substance use at the individual and event levels. Although PIT served as the theoretical starting point for this work, other perspectives similarly posit co-occurrence of risk taking behaviors among youth (e.g., problem behavior theory; Willoughby, Chalmers, & Busseri, 2004).

Implications, Limitations, and Future Directions

Given the health costs associated with risk taking for adolescents, understanding the factors that encourage or deter such behavior is critically important. While the genetic underpinnings of such factors are becoming increasingly recognized and understood (e.g., Zietsch et al., 2010), the current work identifies more frequent use of TAC products as mediating some of the relationship between exposure to socially deviant paternal behavior and risky sexual behavior among adolescent daughters. That is, TAC use frequency is a potential target for prevention and intervention efforts designed to

reduce sexual risk taking among female adolescents exposed to socially deviant paternal behavior. Recent meta-analyses support the effectiveness of a variety of interventions aimed at reducing teens' substance use, including text messaging, motivational interviewing, and internet-based approaches (e.g., Jensen et al., 2011; Mason, Ola, Zaharakis, & Zhang, 2015; Tait, Spijkerman, & Riper, 2013). Further, some interventions that target substance use among adolescents also have been shown to reduce their sexual risk behaviors (Hale, Fitzgerald-Yau, & Viner, 2014). Therefore, it is possible that the most effective interventions for reducing sexual risk taking among female adolescents will simultaneously target their frequency of use of tobacco, alcohol, and marijuana.

Despite the practical significance of this work, there are limitations that future research should address. First, the current analyses focused on frequency of use of TAC products among adolescents, as opposed to their use of harder drugs. Cigarette, alcohol, and marijuana use in early adolescence does predict later hard drug use (Hemovich et al., 2011), and the substances on which we focused are some of the most widely used and abused among American youth (Chassin et al., 2009; Johnston et al., 2019). Further, statistical relationships between substance use and risky sex have been observed across substance type (Ritchwood et al., 2015). Still, it remains possible that adolescents who frequently use hard drugs may be most vulnerable to the negative consequences of risky sexual behavior (e.g., teen pregnancy involvement, STIs).

Second, the differential sibling-exposure design involves recruiting a hard-to-reach sample of participants and requires retrospective assessments to ensure that older and younger sisters can report on their (and their fathers') behavior during the same developmental stages. These constraints produced limitations related to our recruitment procedures and study measures. Regarding the former, women were recruited from across the United States using online methods. Although this approach produced a sample more diverse and representative of the U.S. population than most convenience samples (e.g., Buhrmester, Kwang, & Gosling, 2011), online recruitment and data collection raises concerns related to data quality (e.g., Ogolsky, Niehuis, & Ridley, 2009). We addressed these concerns by speaking with each participant via telephone, embedding attention checks within the survey, and carefully screening study responses to ensure they were valid. Regarding the use of retrospective measures, responses on these measures can be biased and do not consistently correlate with prospective reports (e.g., Hardt & Rutter, 2004; Reuben et al., 2016). We attempted to minimize this concern by comparing sister reports and by assessing objective events in addition to subjective experiences. Although we believe that the strengths of the current methodology balance these concerns, future investigations would benefit from the use of longitudinal designs and in-person interviews.

Finally, the genetically and environmentally informed nature of the differential sibling-exposure design suggests a causal effect of exposure to fathers' deviant behavior on daughters' frequency of TAC use, and more frequent TAC use mediated the relationship between paternal social deviance and risky sexual behavior for older sisters (i.e., moderated mediation). However, because these measures were collected at a single time point and were retrospective (as discussed above), the models reported herein are not temporally informative. Though the results support paternal behavior quality affecting daughters' risky sexual behavior through their frequency of substance use, such effects are unlikely to unfold in a purely linear fashion. As noted in the Introduction, while substance use can increase one's engagement in risky sex via disinhibition (e.g., Coleman & Cater, 2005), engaging in risky sex also can encourage substance use (e.g., Hendershot et al., 2010). Therefore, it remains most likely that across time and at the event-level, substance use and risky sexual behavior will affect each other in a transactional manner (e.g., Cooper, 2006). Similarly, we cannot rule out bidirectional influences related to fathers' and daughters' behavior. Although we interpret our results as suggesting that variation in fathers' social deviance had a stronger effect on older (vs. younger) sisters' frequency of substance use due to their prolonged co-residence, it remains possible that older (vs. younger) daughters also had more opportunity to influence their fathers' behavior (for better or for worse).

Conclusion

Risky behavior constitutes one of the most serious challenges to adolescent health in the U.S. today (Steinberg, 2015). To reduce the considerable costs to youth, their families, and society, it is important to understand the factors that encourage some youth to regularly engage in these behaviors. The current work finds that the quality of resident fathers' behavior, namely their engagement in

socially deviant behaviors, may play an important role in shaping daughters' risk taking during adolescence. This role is expected to be magnified among daughters who experience prolonged exposure to this behavior (here, older daughters from divorced/separated families). By comparing the outcomes of sisters from the same family, our results reveal a potential effect of fathers on daughters' frequency of substance use, independent of environmental and genetic confounds that vary between families (e.g., poverty, genetic risk for impulsive behavior). The observed links between exposure to paternal social deviance, substance use, and risky sexual behavior suggest that prevention and/or intervention efforts targeting teens' frequency of TAC use also may function to improve the sexual health of adolescent females.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Appendix S1. Models Predicting Adolescent Daughters' Problems Related to Alcohol and Marijuana Use.

Appendix S2. Analyses Excluding Families with Missing Frequency of TAC Use Data.

Appendix S3. Models Predicting Adolescent Daughters' Frequency of Alcohol and Cannabis Use.