RUNNING HEAD: RISKY SEXUAL BEHAVIOR

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Impact of Fathers on Risky Sexual Behavior in Daughters:
A Genetically and Environmentally Controlled Sibling Study

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Abstract

Girls receiving lower quality paternal investment tend to engage in more risky sexual behavior (RSB) than peers. Whereas paternal investment theory posits that this effect is causal, it could arise from environmental or genetic confounds. To distinguish between these competing explanations, the current authors employed a genetically- and environmentally-controlled sibling design (N = 101 sister pairs; ages 18-36), which retrospectively examined the effects of differential sibling-exposure to family disruption/father absence and quality of fathering. Consistent with a causal explanation, differences between older and younger sisters in the effects of quality of fathering on RSB were greatest in biologically disrupted families when there was a large age gap between the sisters (thus maximizing differential exposure to fathers), with greater exposure within families to higher quality fathering serving as a protective factor against RSB. Further, variation around the lower end of fathering quality appeared to have the most influence on RSB. By contrast, differential sibling-exposure to family disruption/father absence (irrespective of quality of fathering) was not associated with RSB. The differential sibling-exposure design affords a new quasi-experimental method for evaluating the causal effects of fathers within families.

Key words: Father involvement, parental investment, risky sexual behavior, behavior genetics, quasi-experimental design, sibling comparisons, paternal investment theory

Risky sexual behavior in adolescence—behavioral patterns that increase risk for sexually transmitted diseases (STDs) and unintended pregnancy—constitute a serious adolescent health problem. Sexual activity has become normative among American teenagers, with more than two-thirds of adolescents engaging in sexual intercourse before age 19 (Terry-Humen, Manlove, & Cottingham, 2006). Despite this prevalence, only a small proportion (10-20%) of sexually active adolescents use condoms consistently (Kotchick, Shaffer, Forehand, & Miller, 2001). These alarmingly high rates of unprotected sexual activity are compounded by behavioral patterns that include multiple short-term sexual partners in adolescence (Overby & Kegeles, 1994; Terry-Humen et al., 2006). Among the ramifications are high rates of adolescent pregnancy and STD infection. Indeed, in the United States, approximately 7-11% of girls between the ages of 15 and 19 become pregnant each year (Cheesbrough et al., 1999; Ventura, Abma, Mosher, & Henshaw, 2008) and 26% of girls between the ages of 14 and 19 are infected with at least one of the most common STDs (Forhan et al., 2008). Given the pervasiveness and consequences of teenage pregnancy and STD infection, it is critical to understand the life experiences and pathways that increase risk for or protect against RSB in adolescents. This understanding would have great relevance to the long-term goal of informing early prevention-intervention strategies for high-risk youth.

Many correlational studies have identified (1) biological family disruption/father absence (i.e., separation/divorce of the birth parents followed by absence of the birth father from the home) and (2) low-quality parent-child relationships (e.g., harsh-conflictual parent-child relationships, low parental warmth or supportiveness, lack of parental monitoring or supervision) as risk factors for RSB in adolescence (Biglan et al., 1990; Coley, Medeiros, & Schindler, 2008; Coley, Votruba-Drzal, & Schindler, 2009; Davis & Friel, 2001; Ellis et al., 2003; Huebner & Howell, 2003; Longmore, Manning, & Giordano, 2001; McBride, Paikoff, & Holmbeck, 2003; Miller, Forehand, & Kotchick, 1999; Ream & Savin-Williams, 2005; Rodgers, 1999; Romer et al., 1999; Rose et al.,

2005; Zimmer-Gembeck & Helfand, 2008). Moreover, the earlier family disruption/father absence occurs, the earlier daughters tend to initiate sexual activity and become pregnant (Ellis et al., 2003). Although this body of research has established replicable empirical phenomena, it has not determined causality because extant correlational designs have not been able to rule out third-variable confounds. Specifically, there are three competing classes of explanation for the observed relations between family environments and RSB:

1.*Family disruption/father absence and/or low-quality parent-child relationships may actually cause higher levels of RSB.* An array of theories in the field of human development—attachment-based theories (e.g., Bowlby, 1988; Cassidy & Shaver, 1999), social learning theories (e.g., Bandura, 1977), evolutionary-developmental theories (e.g., Belsky, Steinberg, & Draper, 1991), life-stress models (e.g., Fergusson & Woodward, 2000; Scaramella, Conger, Simons, & Whitbeck, 1998)—converge on the hypothesis that childhood exposures to family disruption/father absence and dysfunctional parenting, including but not limited to dysfunctional fathering, induce emotionally and behaviorally dysregulated psychological functioning in adolescence, including elevated levels of RSB. Within this context, the focus of the current study on father presence-absence and the quality of father-daughter relationships was guided by an evolutionary-developmental model—paternal investment theory (Draper & Harpending, 1982, 1988; Ellis, 2004; Geary, 2000)—which posits a unique role for fathers in families in regulating daughters' sexual development and behavior (see Discussion, for an extended treatment of paternal investment theory). This emphasis on the unique effects of fathers, independent of the effects of mothers and other sources of stress and support in and around the family, distinguishes paternal investment theory from the other theories cited above. Although paternal investment theory has been programmatically tested in past research in our laboratory and has garnered reasonable support (Ellis & Essex, 2007; Ellis & Garber, 2000; Ellis et al., 1999, 2003; Tither & Ellis, 2008; see also

Coley et al., 2009; Ream & Savin-Williams, 2005; Rodgers, 1999), past studies have not provided adequate tests of the causal influence of family disruption/father absence and father-daughter relationship quality on the timing, incidence, or diversity of sexual behavior in adolescent daughters. Thus, the current study was designed to test the specific hypothesis, drawn from paternal investment theory, that higher quality father-daughter relationships uniquely decrease risk for (are protective against) RSB in daughters.

Although this hypothesis is not inconsistent with any of the major theories cited above, it could still be wrong. As a cautionary note, consider that these same theories have posited that harsh parenting causes aggressive behavior in children; yet, the well-established empirical correlation between corporal punishment and child conduct problems is apparently spurious (i.e., accounted for by children's heritable characteristics; Jaffee et al., 2004; Moffit, 2005).[1]

2. *"Effects" of family disruption/father absence and/or low-quality parent-child relationships on RSB may derive from a family-wide environmental confound*. Family-wide environmental effects are causal factors that differ between families but are shared within families (e.g., socioeconomic status [SES], religion, race, parental psychopathology). A family-wide environmental confound could cause family disruption/father absence and/or low-quality parental investment, on the one hand, and RSB, on the other. For example, poverty is associated not only with elevated rates of family disruption/father absence (e.g. Ellis et al., 2003) and elevated levels of conflict and coercion within families (e.g., Conger, Ge, Elder, Lorenz, & Simons, 1994; Ellis et al., 1999) but also with heightened levels of RSB (e.g., Chandy et al., 1994; Coley et al., 2008; Ellis et al., 2003). If poverty (or some other family-wide environmental factor) is the underlying cause of

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[1] It is important to note that this spurious correlation is between harsh parenting in the normative range (frequency of corporal punishment: grabbing, shaking, spanking) and child aggression. The established correlation between harsh parenting that goes beyond normal limits (physical maltreatment: neglectful or abusive care resulting in injury, sexual abuse, registry with child protection services) and child aggression does not arise from child effects (Moffit, 2005).

the relations between either family disruption/father absence or low-quality parental investment and RSB, then the "effects" of these variables on RSB are in fact spurious (i.e., they arise from a third environmental variable).

3. *"Effects" of family disruption/father absence and/or low-quality parent-child relationships on RSB may derive from a genetic confound*. Behavior geneticists refer to this type of association as a gene-environment correlation. Parents who have aggressive, coercive, conflictual personalities may pass on genes for these personality traits to their children (e.g., Moffitt, 2005; Rhee & Waldman, 2002), as expressed in externalizing behavior problems. Such parents are also at increased risk of becoming single parents or absent parents (e.g., Emery, Waldron, Kitzmann, & Aaron, 1999; Sampson & Laub, 1990). In turn, children who display externalizing behavioral problems early in life are at elevated risk for a variety of negative psychosocial outcomes in adolescence, including RSB (e.g., Bardone, Moffitt, Caspi, Dickson, & Silva, 1996; Ellis et al., 2003; Quinton, Pickles, Maughan, & Rutter, 1993; Timmermans, van Lier, & Koot, 2008; Woodward & Fergusson, 1999). Thus, children who experience family disruption/father absence and/or dysfunctional parenting may display elevated levels of RSB because of higher genetic loading for externalizing behavior problems. Indeed, any genetic factor that is shared by parents and offspring that influences RSB and correlates with family disruption/father absence or low-quality parenting could account for the relations between these variables.

Past Attempts to Address Potential Environmental and Genetic Confounds

Because children cannot be randomly assigned to parenting conditions, past attempts to distinguish between these opposing classes of explanation (causal vs. spurious) have generally relied on establishing temporal precedence through longitudinal analysis and/or incorporating appropriate control variables. Some investigations have demonstrated that family disruption/father absence or low-quality parenting in early to middle childhood (prior to the onset of RSB) predicts

subsequent levels of RSB (e.g., Ellis et al., 2003; McBride et al., 2003). Even in such longitudinal analyses, however, third-variable confounds remain problematic. To address this issue, various studies have tested for associations between family disruption/father absence or low-quality parenting and RSB while statistically controlling for such potential confounds as SES, race/ethnicity, gender, family size, stressful life events, child emotional and behavioral problems, pubertal development, preadolescent hugging and kissing, and early adolescent sexual behavior (Biglan et al., 1990; Chandy et al., 1994; Coley et al., 2008, 2009; Ellis et al., 2003; Friedrich, et al., 2004; Metzler et al., 1994; Mezzich et al., 1997; McBride et al., 2003). Although the effects of family disruption/father absence and of low-quality parenting remained statistically significant in most extant studies after incorporating appropriate controls, the covariate adjustment method necessarily relies on a somewhat arbitrary and incomplete set of control variables that the researcher has measured; it cannot account for unmeasured environmental or genetic factors. This limitation highlights the need for genetically and environmentally controlled research designs that incorporate environmental measures.

One previous research group (D'Onofrio et al., 2006; Mendle et al., 2009) has examined the effects of family disruption/father absence on an indicator of RSB—age at first sexual intercourse— using a research design that controls for environmental and genetic influences that are shared by cousins as a result of having mothers who are sisters. The method compares sisters/mothers (first generation) who have each married and had children (second generation: cousins), but who are discordant for divorce. Although the cousins share either 25% or 12.5% of their genetic makeup through maternal descent (depending on whether the mothers are identical twins or full siblings), they have different environmental exposures to family disruption/father absence. Examining the potential impact of this differential exposure, D'Onofrio et al. (2006) and Mendle et al. (2009) found mixed support for a causal effect of family disruption/father absence on sexual initiation in

cousins. Although the children-of-sisters design constitutes an important methodological advance, the design does not control for either (a) the genetic and environmental influences of the sisters' spouses (Eaves, Silberg, & Maes, 2005) or (b) genetic and environmental risk factors that influence only one of the adult sisters. Family-wide environmental confounds are a major challenge for this design because the children grow up in different homes. Further, the children-of-sisters methodology has only been used to examine the effects of the social address of father presence-absence and has not been applied to the quality of fathering.

The Current Sibling Design

The potential causal influence of exposure to family disruption/father absence and quality of fathering on RSB can be tested through the use of a differential sibling-exposure design, as developed by Tither and Ellis (2008) and extended here. Because this design compares siblings who have the same biological father and mother and grow up in the same home, but are differentially exposed to levels and types of investment from their father (which are measured and analyzed), it does not share the limitations of children-of-sisters approach. Indeed, other than adoption methods, the current differential sibling-exposure design is the only causally-informative method available for examining the impact of natural variation in quality of fathering on child developmental outcomes.

The logic of the differential sibling-exposure design is as follows. Past longitudinal research has demonstrated that girls who spend more of their childhoods in biologically father-absent households are at greater risk for early sexual activity and teenage pregnancy (Ellis et al., 2003). Given this dose-response relationship: Within families in which (a) full biological sisters are discrepant in age, (b) a divorce/separation occurs, resulting in disruption of the biological family unit and departure of the father from the home, and (c) consequently younger sisters in the family spend a larger proportion of their childhood living in a biologically disrupted/father-absent home

than do their older sisters, the younger sisters should engage in more RSB (*Hypothesis 1*). This is because the younger sisters receive a larger dose of family disruption/father-absence and associated factors (i.e., factors that differ between sisters after—and as a result of—family disruption/father absence).

In addition, this should not be explicable as a birth-order or birth-spacing effect because sisters of different ages from two-biological-parent families should not systematically differ in RSB because they do not systematically differ in exposure to their fathers. The differential sibling-exposure design, therefore, necessitates inclusion of a control sample of sisters from two-biological-parent families whose birth spacing and birth order match the study sample of sisters from biologically disrupted/father-absent families. According to the genetic transmission model, and in direct contrast with Hypothesis 1, full biological sisters should not systematically differ in RSB as a function of birth order or birth spacing (even if they have spent different amounts of their childhoods in biologically disrupted families).

The differential sibling-exposure design enables testing of Hypothesis 1 through the following 3-way interaction: The effect of birth order of sisters (younger vs. older) on RSB should be moderated by a combination of family type (biologically disrupted vs. two-biological-parent) and the age gap between sisters (with age gap corresponding to the minimum difference between sisters from divorced/separated families in the amount of time that they resided in two-biological-parent versus biologically disrupted homes). The causal hypothesis is that differences between younger and older sisters in RSB will be greatest in divorced/separated families when there is a large age gap between the sisters (thus maximizing differential exposure to family disruption/father absence), with younger sisters at greater risk.

This social address approach to father presence-absence assumes that being there is what counts. However, because all fathers are not equal, it is unlikely that living in a two-biological-

parent versus biologically disrupted family is always either good or bad for children. Rather, the effects of fathers in families are likely to be moderated by what fathers do. Using the same differential sibling-exposure design that was employed in the current study, Tither and Ellis (2008) found that the effects of differential exposure to fathers on daughters' age at menarche depended on the father's level of social deviance. Specifically, younger sisters from biologically disrupted families who were exposed to serious paternal dysfunction in early childhood, followed by the exit of that father from the home, attained menarche about 11 months earlier than either their older sisters or other younger sisters from biologically disrupted families who were not exposed to such dysfunction. Likewise, Jaffee, Moffitt, Caspi, and Taylor (2003) and Blazei, Iacono, and McGue (2008) found that the potential benefits of living with two biological parents was contingent on the father's behavioral adjustment (see also DeGarmo, 2010). Among children and adolescents whose fathers engaged in low levels of antisocial behavior, co-residence with the father was associated with *lower* rates of conduct problems. Conversely, among children and adolescents whose fathers engaged in high levels of antisocial behavior, co-residence was associated with *higher* rates of conduct problems.

These theory and data suggest that Hypothesis 1, and the 3-way interaction specified to test it, should be conditioned by quality of fathering. Because older sisters in divorced families have more residential exposure to their fathers than do their younger sisters, the impact of quality of fathering and associated factors on RSB should be significantly greater in older than younger sisters (*Hypothesis 2*). In addition, this should not be explicable as a birth order/birth spacing effect because the influence of quality of fathering on RSB should not significantly differ across older versus younger sisters in two-biological-parent families. By contrast, according to the genetic transmission model, the effect of quality of fathering on RSB should not differ between sisters as a function of how long they lived with their father while growing up.

In total, we predicted and tested for the following 4-way interaction: the effect of quality of fathering on RSB should be moderated by a combination of family type (biologically disrupted vs. two-biological-parent), the birth order of sisters (older vs. younger), and the age gap between sisters. The causal hypothesis is that differences between older and younger sisters in the effect of quality of fathering (harsh-coercive fathering, warm-supportive fathering) on RSB should be greatest in biologically disrupted families when there is a large age gap between the sisters (thus maximizing differential exposure to fathers). In this family context, the influence of higher quality fathering should be significantly stronger (operate as a greater protective factor against RSB) in older than younger sisters (Hypothesis 2).

A methodological advance of the current sibling design is that it controls for confounding variables—factors that differ between sisters prior to family disruption/father absence—at three levels. First, family-wide environmental effects are controlled through within-family analyses. Specifically, direct comparisons between biological siblings in the same home obviate confounding effects associated with comparisons between individuals from different homes, such as differences between individuals in race/ethnicity, SES, and religion (Rodgers, Cleveland, van den Oord, & Rowe, 2000; Sulloway, 1996; Tither & Ellis, 2008). Second, genetic effects are controlled through randomization. Because the breaking up and reshuffling of parental genomes through meiosis randomly distributes alleles of the parents' genes across offspring, genetic differences between sisters should be randomly distributed across birth order. Specifically, there is no reason to expect that either younger sisters or older sisters, as a group, have systematically greater genetic liability for anything. (For example, imagine 100 families that have four children and a genetic risk for alcoholism. On average, the 100 firstborns of each family will have the same genetic risk for alcoholism as the 100 secondborns, and so on.) Third, including sister pairs from two-biological-parent families as a comparison group controls for birth order and birth spacing effects. By

controlling for all three levels of confounding variables, the current differential sibling exposure design enabled us to test whether the different (nonshared) experiences of sisters within biologically disrupted families caused differences in RSB. The measured non-shared environmental variables were differential exposure to family disruption/father absence (Hypothesis 1) and quality of fathering (Hypothesis 2).

A further advantage of the differential sibling exposure design is that it enables examination of father-effects independent of mother-effects (which is critical to testing paternal investment theory). Because comparisons are within-family, and both daughters have the same mother and live with her throughout their childhoods, the design stringently controls for maternal characteristics and behavior. Nonetheless, it is possible that the effects of father-daughter relationships could be confounded by mother-daughter relationships. To address this issue, quality of mother-daughter relationships was measured and controlled for in testing Hypothesis 2 (regarding the effects of fathering quality).

Method

The current research necessitated a retrospective design. On the one hand, the methodology requires that pairs of sisters are several years apart in age (so as to ensure large within-pair differences in amount of exposure to their fathers). Following Tither and Ellis (2008), we set the minimum age difference between sisters at 4 years. On the other hand, the design requires outcome information about the same stage of development—adolescence—so as to obtain equivalent and uncensored information about RSB from each sister. To meet both requirements, all participants had to be at least 18 years old. To limit the span of retrospective recall, we set the maximum age of participants at 36 years.

The foremost challenge of this research design is obtaining a sample of full biological sister pairs who are at least 4 years apart in age, experienced the dissolution of their parents' relationship

when the younger sister was under age 14 (before the likely onset of RSB), and then lived primarily

or in joint custody with their mother. Because such sister pairs are rare, they are very difficult to

obtain through standard sampling methods and instead must be located through targeted advertising.

Although advertising results in a self-selected sample, this is not a major issue in the current

research design because all analyses compared sisters in the same family to each other, thus

controlling for the confounding effects of genetics and environmental differences between families.

Further, as summarized below, the demographics of the sample were largely representative of the

U.S., and the pattern of covariation among variables was consistent with past family research.

Participants.

Recruitment. To solicit participation of sisters from both biologically disrupted and two-

biological-parent families, the following advertisement was placed on Craigslist

(www.craigslist.org) in several major U.S. cities over a six month time period:

SISTERS WANTED! (and their parents) to complete a questionnaire. Are you from a family

in which there are two sisters who are at least 4 years apart in age? Are both sisters currently

between the ages of 18 and 36? Did the birth parents separate or divorce while the younger

sister was under 14 years of age? [Sentence deleted when recruiting two-biological-parent

families.]

Compensation is provided for completing a simple questionnaire that takes about 1

hour. We are looking for sisters and their parents to participate in a paid research study on

behalf of the University of Arizona investigating the influence of family relationships on

behavior. Sisters will complete a questionnaire that asks about your teenage years, your life

now, and your family relationships. Parents will complete a questionnaire that asks about

your family relationships and behavior. Your responses to this research are strictly

confidential. This study has been reviewed and approved by the University of Arizona

Human Subjects Protection Program

Respondents to the advertisement were screened by telephone for age, demographics, and family

composition. Of the 274 respondents (families), 130 sister pairs met eligibility criteria, were invited

to participate in the study, and were mailed questionnaires; 144 sister pairs failed to meet eligibility

criteria (e.g., sisters too close in age, sisters had different fathers or mothers, younger sister too old

at time of divorce, participants currently too old or young) and were excluded. Of the 130 eligible

pairs, 101 were successfully recruited into the study and 29 were not (either one or both sisters did

not complete the questionnaire). The final analytic sample included 42 sister pairs from two-

biological-parent families and 59 sister pairs from biologically disrupted families ($N = 202$

individuals). At prescreening, all respondents were asked about their race/ethnicity, number of

siblings, mother's highest education level, father's highest education level, and general SES while

growing up (poor, lower middle class, middle class, upper middle class, wealthy). Independent

samples t-tests and chi-square analyses were conducted, as appropriate, to test for differences

between the 101 participating families and the 173 families who were screened but did not

participate; no statistically significant differences emerged on any of the screening variables. Thus,

the analytic sample appears to be representative of the total population of individuals responding to

the advertisements. Although we did not ask about languages spoken in the home, all participants

were fluent in English.

In the 42 two-biological-parent families, the parents were married or cohabitated throughout

both of the sisters' childhoods (birth to age 18). In the 59 biologically disrupted families, the

parental union terminated through divorce or separation when the younger sister was under 14 years

of age. In the biologically disrupted families, the average age of the younger sisters was 6.42 years

($SD = 4.01$) and the older sisters was 13.10 years ($SD = 4.37$) at the time of divorce/separation. The

average age difference between sisters was 6.58 years (SD = 2.17) in biologically disrupted families and 6.48 years (SD = 1.99) in two-biological-parent families. After the divorce/separation, sisters lived primarily with their mother (84%) or jointly with their mother and father (16%). At the time of data collection, age of the younger sisters ranged from 18-32 years (M = 23.44, SD = 4.10) and age of older sisters ranged from 22-36 years (M = 29.89, SD = 4.05).

We also sought to recruit mothers into the study. Although we obtained the participation of a high percentage of the mothers from the biologically intact families (86%), we were less successful in recruiting mothers from the biologically disrupted families (66%). Because analysis of disrupted families was critical to the current research design, and because one-third of mothers from disrupted families did not participate, mother-data were not used in the multivariate analyses. However, mothers in disrupted families may be in a unique position to report on the amount of time that daughters spent with their biological fathers before and after family disruption (given that some of the younger sisters in our study would have been too young to remember levels of pre-divorce paternal involvement). Thus, as described below, we report descriptive data from mothers regarding amounts of time that fathers spent caring for their daughters before and after the separation/divorce.

Demographics. Because the current sample was obtained through advertising, it is important to show that its demographic characteristics are not unusual. Accordingly, we report demographic comparisons between the current sample and U.S. Census data (United States Census, 2000). The present sample was racially and ethnically diverse (59% Caucasian [non-Hispanic]; 18% Hispanic, 16% African American; 7% other) and parallels the racial-ethnic profile of the U.S. population (Census data: 63% Caucasian [one-race, non-Hispanic]; 13% Hispanic or Latino [of any race], 12% African American [one-race]). To gauge family SES, sisters reported on father's occupational status and mother's education. Using the Hollingshead (1975) system, families were classified into nine groups on the basis of paternal occupation. For the present purposes, the Hollingshead coding was

reduced to a three-level classification as follows: Levels 1/2/3 (21%; laborers, semiskilled, service Industry workers); Levels 4/5/6 (46%; craftsmen, small business owners, clerical, technicians); Levels 7/8/9 (33%; professional, managerial, executive, large business owners). These numbers again closely matched population statistics (Census data for full time workers over age 16: Levels 1/2/3 [22%]; Levels 4/5/6 [46%]; Levels 7/8/9 [32%]). Finally, mothers' highest level of education was coded into 5 levels: No high school diploma or GED (8%); high school diploma or GED (23%); some college (36%); Bachelor's degree (22%); advanced degree (12%). These numbers also approximated population statistics (Census data for individuals 40-45 years old: No high school diploma or GED [15%]; high school diploma or GED [29%]; some college [31%]; Bachelor's degree [17%]; advanced degree [9%]). In sum, the demographics of our sample were largely representative of the U.S. Nonetheless, the relatively low number of mothers with no high school diploma or GED suggests that the current sample may not adequately represent very low socio-economic or impoverished subpopulations.

Measures.

Quality of father-daughter and mother-daughter relationships. Two scales were used to create measures of the quality of father-daughter and mother-daughter relationships. The first scale measured harsh-coercive fathering/mothering and was derived from a subset of items from the parent-child version of the Conflict Tactics Scale (Jouriles, Mehta, McDonald, & Francis, 1997; Straus, 1979), adapted for use in this study. Using a 4-point scale (0 = very unlike to 3 = very like), participants answered the following questions about their father and mother separately: "My father/mother swore (cursed) at me"; "… insulted me or put me down"; "… acted in a way that made me afraid that I might be physically hurt"; and "… pushed, grabbed, or slapped me." Participants were instructed to report the extent to which each item described their father/mother during the first 16 years of their lives. Scores on the four items were averaged together to form a

composite measure of harsh-coercive fathering (biologically disrupted families: $M = .55$, $SD = .78$; two-biological-parent families: $M = .39$, $SD = .61$; $\alpha = .86$). and harsh-coercive mothering (biologically disrupted families: $M = .47$, $SD = .65$; two-biological-parent families: $M = .36$, $SD = .56$; $\alpha = .80$). Although retrospective reports by adults of coercive parental behavior are likely to underestimate the incidence of abuse/neglect, Hardt and Rutter (2004) conclude that these reports are still sufficiently valid to warrant their use. The validity of the current harsh-coercive parenting measures is supported by the moderate to strong correlations between younger and older sisters (harsh-coercive fathering: r [101] $= .67$, $p < .001$; harsh-coercive mothering: r [101] $= .41$, $p < .001$; collapsed across family type).

The second scale, which measured paternal/maternal warmth-supportiveness, was the 12-item Care subscale of the Parental Bonding Inventory (PBI; Parker et al., 1979). Using a 4-point scale (0 = very unlike to 3 = very like), participants answered the questions about their father and mother separately (e.g., "My father/mother spoke to me with a warm and friendly voice." "My father/mother appeared to understand my problems and worries."). Participants were again instructed to report the extent to which each item described their father/mother during the first 16 years of their lives. Scores on the 12 items were averaged to form a composite measure of warm-supportive fathering (biologically disrupted families: $M = 1.55$, $SD = .89$; two-biological-parent families: $M = 2.05$, $SD = .80$; $\alpha = .96$) and warm-supportive mothering (biologically disrupted families: $M = 2.15$, $SD = .70$; two-biological-parent families: $M = 2.39$, $SD = .71$; $\alpha = .94$). The validity of the current warm-supportive parenting measures is supported by the strong correlations between younger and older sisters (warm-supportive fathering: r [101] $= .66$, $p < .001$, warm-supportive mothering: r [101] $= .51$, $p < .001$; collapsed across family type). Further, the Care subscale of the PBI has demonstrated high test-retest reliability over a 10-year period (Wilhelm &

Parker, 1990) and correlates with many forms of adolescent behavioral adjustment and mental health (e.g., Chambers et al., 2001; Gerra et al., 2004; Martin et al., 2004).

The measures of harsh-coercive parenting [reverse-coded] and warm-supportive parenting were then standardized across sister and family type, for fathers and mothers separately, and averaged together to form overall measures of *father-daughter relationship quality* ($\alpha = .72$) and *mother-daughter relationship quality* ($\alpha = .76$). To achieve grand mean centering, the composite measures were then standardized separately for fathers (biologically disrupted families: $M = -.18$, $SD = 1.03$; two-biological-parent families: $M = .25$, $SD = .91$) and mothers (biologically disrupted families: $M = -.12$, $SD = 1.00$; two-biological-parent families: $M = .16$, $SD = .98$) for use in multivariate analysis. Higher scores indicate higher relationship quality.

Paternal care before and after family disruption. An assumption of the current research design is that paternal involvement declines following separation/divorce. To assess the amount of paternal care provided before and after family disruption, older and younger sisters in disrupted families were asked to report the number of hours per week their father spent taking care of them before and after their parents' separation/divorce. Responses were coded on a 5 point scale (1 = None; 2 = 1-2 hours; 3 = 3-5 hours; 4 = 6-19 hours; 5 = 20 hours or more). In addition, mothers from disrupted families (n = 39) also answered this question with respect to each of their daughters. Descriptive information on this variable is presented in Figure 1.

Risky sexual behavior (RSB). Risky sexual behavior was based on number of sexual partners and amount of high-risk sexual activity. Participants reported the frequency of these sexual behaviors when they were high school age (14-17 years old). The first scale assessed number of sexual partners: "How many different partners did you have sex with before age 19?" To reduce the influence of outliers and induce a unimodal distribution, the open-ended reports of number of sexual partners were recoded into the following scale: 0 = None, 1 = one to two, 2 = three to five, 3 = six

or more). In the metric of this recoded scale, the average number of sexual partners for sisters from two-biological-parent and biologically disrupted families was .81 (SD = .92) and 1.14 (SD = 1.02), respectively. The second scale assessed frequency of high-risk sexual activity (behaviors that increased risk for STDs or pregnancy): "Did you ever have unprotected sex (i.e., sex without any method to prevent pregnancy or sexually transmitted disease)?" "Did you ever use alcohol and/or drugs prior to or in conjunction with participation in sexual activity?" "Did you ever engage in or experience sexual intercourse with an injection drug using partner?" "Did you ever engage in or experience sexual intercourse with someone who was physically forceful, hurting, or threatening to you?" "Did you ever engage in or experience concurrent (overlapping) sexual relationships with different partners?" "Did you ever receive money, drugs, or a place to stay in exchange for sexual activity with someone?" (0 = no; 1 = once or twice; 2 = 3 times or more). These six items showed good internal consistency (α = .72). Overall, 48.3% of sisters from biologically disrupted families and 33.3% of sisters from two-biological-parent families reported engaging in at least one of these high risk behaviors between 14 and 17 years of age. The 6 items were standardized and averaged to form a composite measure of high-risk sexual behavior.

To create an overall measure of RSB, number of sexual partners was standardized and then combined with amount of high-risk sexual behavior in a unit-weighted composite (α = .69). Graphical inspection of the composite variable revealed two outliers (high scores), which were truncated. The final RSB measure was standardized to achieve grand mean centering with a mean of 0 and SD of 1. Mean levels of RSB were .13 (SD = 1.02) in biologically disrupted families and -.18 (SD = .95) in two-biological-parent families. Although there is no gold standard for validating self-reports of sexual behavior, available evidence solidly supports the validity of such reports (Fishbein & Pequegnat, 2000; Dunne et al., 1997; Hamilton & Morris, 2010).

Data Analysis

Data from sisters reared within the same household are likely to violate the assumption of independence (i.e., uncorrelated error terms) that is required for standard ANOVA and regression approaches. Non-independence of errors can result in inaccurate significance tests and erroneous conclusions if not modeled accordingly (Kenny, Kashy, & Cook, 2006). To address this we used a commonly recommended technique for cross-sectional distinguishable dyads (described in detail in Kenny, Kashy, & Cook, 2006, Ch. 4; see also Campbell & Kashy, 2002; Kashy & Kenny, 1997; Maguire, 1999). For the fixed predictor portion of the model, this technique is similar to the indistinguishable dyad model in that it makes use of a single dyad-level intercept. For the error covariance portion of the model, however, this technique differs from the indistinguishable dyad approach in that it specifies within-dyad correlated residuals (rather than including a random dyad-level intercept). In addition, because dyad members in the present analysis possess a distinguishable feature (older vs. younger sister), the indistinguishable dyad model was extended to include a dummy coded fixed-effect variable (older vs. younger sister) to model differences between sisters within families. This approach makes use of software designed for multilevel modeling to implement a generalized form of a mixed ANOVA that (1) allows for both categorical and continuous predictors; (2) permits modeling of between-family effects (i.e., two-biological-parent vs. biologically disrupted; age gap between sisters), within-family effects (i.e., older vs. younger sister; quality of the father-daughter relationship experienced by each sister), and their interactions; and (3) adjusts outcome scores for measurement error (Sayer & Klute, 2005).

In this analysis, the REPEATED command in SAS PROC MIXED was used to specify a compound symmetric covariance structure for the residuals from sisters nested within families, and the Satterthwaite degrees of freedom estimation was used to adjust the degrees of freedom based on the amount of interdependence. Initial results showed a significant correlation for RSB between sisters within families (r [101] = .25, $p < .05$), indicating that the error terms for the dependent

variable for sisters within families were indeed non-independent. Having accounted for this interdependence between sisters within families, hypothesis testing can be conducted by specifying appropriate fixed effect models in the same way as is done in standard multiple regression. Parameters that are reported are from models that included all relevant covariates, predictors, and interaction effects.

Results

Preliminary Analyses

Between-family correlations. Because the current sample was recruited through targeted advertising, resulting in possible self-selection biases, it is important to show that the sample is not unusual, either in its demographics (as reviewed in the Method) or in the pattern of relations among variables. Table 1 shows the between-family correlations (with older and younger sister-reports averaged within families) for the study variables. As can be seen in the table, the pattern of between-family correlations largely conforms to expectations. Specifically, family SES was cohesive (i.e., paternal occupation and maternal education were strongly correlated). Lower SES families were more likely to be racial/ethnic minorities and to be characterized by lower quality father-daughter and mother-daughter relationships. Further, there was a positive correlation between quality of father-daughter and mother-daughter relationships. In addition, families in which girls engaged in more RSB were characterized by lower quality father-daughter and mother-daughter relationships. Finally, girls from biologically disrupted homes experienced significantly lower quality mother-daughter and father-daughter relationships and engaged in significantly more RSB. The one seemingly anomalous correlation was that SES was not associated with family structure (biologically disrupted vs. two-biological-parent). The likely explanation is that our sample did not include any single-mother families or short-term, unstable separated/divorced families. Instead, all of our biologically disrupted families were intact for many years prior to the divorce/separation (so

as to produce two daughters who were several years apart in age). Paternal occupational status and maternal education simply did not discriminate between the long-term biological families that eventually did or did not dissolve.

Changes in paternal care following family disruption. An assumption of the current differential sibling exposure design is that amount of contact with the father diminishes following biological family disruption. To test this assumption, we conducted paired samples t-tests comparing levels of paternal care by the father (based on both daughter- and mother-reports) before and after family disruption. As shown in Figure 1, both older daughters ($t[57] = 5.39, p < .001$) and younger daughters ($t[58] = 2.45, p < .05$) reported that their fathers spent significantly more time caring for them prior to the divorce/separation than after it. Likewise, as shown in Figure 1, mothers reported that the fathers spent significantly more time caring for both their older daughters ($t[37] = 5.13, p < .001$) and younger daughters ($t[38] = 2.29, p < .05$) prior to the divorce/separation than after it. In total, consistent with past research showing greatly diminished contact between children and noncustodial fathers following divorce (e.g., Furstenberg & Nord, 1985; Fox, 1995), these results support the assumption of diminished interaction with fathers following biological family disruption. Accordingly, younger sisters, who spent substantially more time in biologically disrupted/father-absent homes, experienced longer periods of diminished paternal care than did their older sisters. This finding serves as an important "manipulation check" for the current natural experiment on differential sibling exposure to fathers.

Between-family vs. within-family variation. The differential sibling-exposure design enables analysis of the causes of within-family variation in child outcomes. The method depends, however, on substantial within-family variation in relevant outcome variables. As a first step in the analyses, therefore, we ran an unconditional means model to assess within- and between-family variance in RSB. The results indicated significant variance in RSB both within ($\sigma^2 = .75, p < .0001$) and

between ($\sigma^2 = .24$, $p < .05$) families. With 75% of the variance in RSB occurring within families, there was ample room for within-family analyses.

Primary Analyses

To test the study hypotheses, two separate models were assessed. Model 1 tested Hypothesis 1 (that greater exposure to family disruption/father absence causes more RSB in daughters); Model 2 tested Hypothesis 2 (that greater exposure to lower quality father-daughter relationships increases RSB). Although Table 2 shows all parameter estimates and standard errors for both models (with birth order coded as 0 = older, 1 = younger; family composition coded as 0 = biologically disrupted, 1 = two-biological-parent; and continuous predictors mean centered), we only interpret those parameters that are directly relevant to testing the hypotheses.

Model 1. As specified by Hypothesis 1, the effect of birth order of sisters (older vs. younger) on RSB should be moderated by a combination of family type (biologically disrupted vs. two-biological-parent) and the age gap between sisters; specifically, differences between younger and older sisters in RSB should be greatest in biologically disrupted families when there is a large age gap between the sisters (thus maximizing differential exposure to family disruption/father absence), with younger sisters in this context engaging in more RSB. Model 1 thus tested for a 3-way interaction between the specified independent variables as follows:

$$RSB_{ij} = B_0 + B_1 famtype_j + B_2 birthorder_{ij} + B_3 agegap_j + B_4 famtype_j * birthorder_{ij}$$
$$+ B_5 famtype_j * agegap_j + B_6 birthorder_{ij} * agegap_j + B_{12} famtype_j * birthorder_{ij} * agegap_j + e_{ij}, (1)$$

where i = sister and j = family with e_{ij} assumed to be distributed normally, with a mean of zero and a compound symmetric covariance structure.

Hypothesis 1 was not supported: differences between sisters in amount of exposure to family disruption/father absence was not associated with differences in RSB, as evidenced by the non-significant 3-way interaction, $F(1, 97) = .02$, *ns*. In total, there was no evidence in our data that greater exposure within families to family disruption/father absence, without taking into account

quality of fathering, increased RSB in daughters. This null finding should be interpreted with caution, however, because our relatively modest sample size did not provide adequate power to guarantee the detection of small effects. As such, our failure to detect an impact of family disruption/father absence may be purely due to limited statistical power.

Model 2. To test Hypothesis 2, we examined whether the 3-way interaction tested in Model 1 was conditioned by quality of father-daughter relationships (FDR). Quality of mother-daughter relationships (MDR) was included as a covariate in Model 2. Simple effects included the three predictors from Model 1 (family type, birth order of sisters, and age gap between sisters) plus quality of father-daughter relationships. The formula for Model 2 was as follows:

$$RSB_{ij} = B_0 + B_1 MDR_{ij} + B_2 famtype_j + B_3 birthorder_{ij} + B_4 agegap_j + B_5 FDR_{ij}$$
$$+ B_6 famtype_j * birthorder_{ij} + B_7 famtype_j * agegap_j + B_8 famtype_j * FDR_{ij} + B_9 birthorder_{ij} * agegap_j$$
$$+ B_{10} birthorder_{ij} * FDR_{ij} + B_{11} agegap_j * FDR_{ij} + B_{12} famtype_j * birthorder_{ij} * agegap_j$$
$$+ B_{13} famtype_j * birthorder_{ij} * FDR_{ij} + B_{14} famtype_j * agegap_j * FDR_{ij} + B_{15} birthorder_{ij} * agegap_j * FDR_{ij}$$
$$+ B_{16} famtype_j * birthorder_{ij} * agegap_j * FDR_{ij} + e_{ij}. \qquad (2)$$

where i = sister, j = family with e_{ij} assumed to be distributed normally with a mean of zero and a compound symmetric covariance structure.

Although Model 2 included all possible interaction terms, the *a priori* causal hypothesis targeted the 4-way interaction (quality of the father-daughter relationship in each sister * family type [two-biological-parent vs. biologically disrupted] * birth order of sisters [older vs. younger] * age gap between sisters), which the theory stipulated should be statistically significant, and two of the simple 3-way interactions that were directly relevant to interpreting the 4-way interaction. The targeted simple 3-way interactions were between quality of father-daughter relationships, birth order of sisters, and age gap between sisters (in biologically disrupted and two-biological-parent families, respectively). We predicted that this simple 3-way interaction would be significant in the biologically disrupted families but not in the two-biological-parent families. Specifically, in biologically disrupted families, differences between older and younger sisters in the effect of quality

of the father-daughter relationship on RSB should be greatest when there is a large age gap between the sisters (thus maximizing differential exposure to fathers).

We tested for the 4-way interaction in the full model; it was statistically significant, $F(1, 115) = 11.53$, $p < .001$. To examine the effect size of the 4-way interaction, a pseudo-R^2 was calculated from the within-person residuals comparing models with and without the 4-way interaction (see Singer & Willett, 2003). Results indicated that adding the 4-way interaction explained an additional 9.4% of the variance in the within-person residuals. Our theoretical model guided our prediction that the 4-way interaction would be a fairly large effect and thus detectable even with relatively low power. The relatively large effect size we observed supports this assumption and makes this finding even more notable.

To interpret the interaction, we examined the two relevant simple 3-way interactions. As predicted, the 4-way interaction arose from the presence of a statistically significant simple 3-way interaction (between quality of father-daughter relationships, birth order of sisters, and age gap between sisters) in the biologically disrupted families (t [118] $= 3.27$, $p < .01$) but not in the two-biological-parent families (t [113] $= 1.78$, ns). The simple 3-way interaction in the biologically disrupted families is illustrated in Figure 2.

The significant simple 3-way interaction in the biologically disrupted families indicates that the effect of quality of father-daughter relationships on RSB depended on the birth order of sisters and the age gap between them. For older sisters that had a large age gap with their younger sisters (and thus resided with their father for a substantially longer period of time), there was a strong linear effect of father-daughter relationship quality on RSB (simple slope: $b = -.64$ [95% CI: -1.01 to -.28], t [183] $= 3.45$, $p < .001$). That is, among these older sisters, a 1 SD increase in father-daughter relationship quality was associated with approximately 2/3 of a SD decrease in levels of RSB (Figure 2). By contrast, for younger sisters that had a large age gap with their older sisters (and

thus resided with their father for much less of their childhood), there was not a statistically significant effect of father-daughter relationship quality on RSB (simple slope: $b = .09$ [95% CI: -.29 to .47], t [184] = 0.46, ns). Importantly, these simple slopes were significantly different from each other (interaction term: $b = .73$ [95% CI: .24 to 1.23], t [123] = 2.93, $p < .01$).

By contrast, for sisters from biologically disrupted families with small age gaps (who thus more closely resembled each other in amounts of time spent living with their father while growing up), the simple slopes of RSB on quality of father-daughter relationships were neither significantly different from zero nor significantly different from each other. Further, among sisters from two-biological-parent families, none of the simple slopes of RSB on father-daughter relationship quality were statistically significant, regardless of whether the sisters were older or younger or had small or large age gaps between them,

In total, as specified by the theory, the effect of quality of father-daughter relationships on RSB was moderated by a combination of family type (biologically disrupted vs. two-biological-parent), the birth order of sisters (older vs. younger), and the age gap between sisters. Specifically, the effect of quality of father-daughter relationships on RSB was greatest in biologically disrupted families when there was a large age gap between the sisters (thus maximizing differential exposure to fathers). This differential exposure elevated risk more steeply in older sisters than younger sisters, as predicted by Hypothesis 2.

To genuinely demonstrate unique fathering effects, it is important to show that comparable results would not be obtained if we switched out father-daughter relationship quality and mother-daughter relationship quality in the analyses. Accordingly, we reran the 4-way interaction analysis exactly as described above, but included father-daughter (rather than mother-daughter) relationship quality as the covariate and included mother-daughter (rather than father-daughter) relationship quality at the level of main effects, 2-way interactions, 3-way interactions, and the 4-way

interaction. Neither the 4-way interaction (F [1, 137] = 1.28, p = .26) nor the 3-way interaction in biological disrupted/father-absent families (t [181] = .80, p = .43) was statistically significant. In total, quality of mother-daughter relationships was not interchangeable with quality of father-daughter relationships, indicating a unique effect of fathering.

To further demonstrate unique fathering effects, it is also important to show that comparable results would not be obtained if we switched out father-daughter relationship quality with a more general parenting measure (the average of father-daughter and mother-daughter relationship quality). Accordingly, we reran the 4-way interaction analysis as described above, but included the averaged father-mother measure (rather than father-daughter relationship quality) at the level of main effects, 2-way interactions, 3-way interactions, and the 4-way interaction. There was no covariate in this analysis. Neither the 4-way interaction (F [1, 133] = 1.20, p = .27) nor the 3-way interaction in biological disrupted/father-absent families (t [166] = 1.23, p = .22) was statistically significant. Thus, a global measure of parent-child relationship quality (averaging mothers and fathers) was not interchangeable with quality of father-daughter relationships, again indicating a unique effect of fathering.

Secondary Analyses

According to the theory, quality of father-daughter relationships should predict RSB in two-biological-parent families because daughters have maximum exposure to their fathers in this context. However, as noted above, none of the simple slopes of RSB on father-daughter relationship quality were statistically significant in two-biological-parent families. Such effects may have been obscured by low statistical power, however, and so additional analyses were conducted to explore these null results for the purpose of directing future research. Given that sisters from biologically disrupted families reported significantly lower quality father-daughter relationships than did girls from two-biological-parent families (Table 1), it may be that variation in quality of fathering only

predicts RSB when that quality is low and does not predict RSB when that quality is high. We examined this possibility in the 101 older sisters in our study (all of whom had high levels of residential exposure to their fathers while growing up). For the 41 older sisters who reported below average father-daughter relationship quality (scores below 0), the correlation between quality of father-daughter relationships and RSB was -.32, $p < .05$. By contrast, for the 60 older sisters who reported above average father-daughter relationship quality (scores above 0), the correlation between father-daughter relationship quality and RSB was -.02, ns. Thus, only variation around the lower end of fathering, as was typical of biologically disrupted families, was associated with RSB.

Discussion

The results of the current study suggest that it is not how long daughters live with their fathers that influences RSB, but rather what the fathers do. Specifically, the present analyses support the hypothesis that higher quality father-daughter relationships are uniquely protective against RSB in daughters, with the caveat that variation around the lower end of father-daughter relationship quality appears to have greatest influence. This finding converges with past research establishing an association between higher quality fathering and less RSB in daughters (Ream & Savin-Williams, 2005; Rodgers, 1999; Coley et al., 2009). Extending past research, however, the current quasi-experimental design—examining differential sibling exposure to paternal behavior within families—enabled a plausible test of causality. In families in which older and younger sisters experienced large differences in length of residence with their father (biologically disrupted families with large age gaps between the sisters that maximized differential exposure to fathers), the protective effect of higher quality father-daughter relationships against RSB substantially and significantly differed across sisters. Specifically, in this family context, where the older sisters had high exposure to their fathers while the younger sisters had low exposure, there was a strong negative effect of father-daughter relationship quality on RSB in older sisters ($b = -.64$) but not

younger sisters ($b = .09$). By contrast, in families in which older and younger sisters both lived with their father throughout childhood (two-biological-parent families) or only experienced moderate differences in length of residence with their father (biologically disrupted families with relatively small age gaps between the sisters), the regression slopes of RSB on quality of father-daughter relationships did not significantly differ between older and younger sisters. Taken together, these within-family results, which cannot plausibly be explained by genetic or family-wide environmental confounds, provide support for a causal interpretation. Further, the data indicate that these results are not an artifact of birth order or birth spacing, which were controlled through comparisons with two-biological-parent families.

Although the differential sibling exposure design randomizes genetic influences across siblings, it is not impervious to gene-environment (G-E) processes. Older sisters, who live with their fathers for substantially longer periods of time than do their younger sisters, not only have more environmental exposure to their fathers but also more opportunity for reactive G-E processes (where the child's heritable characteristics influence parental behaviors such as harsh-coercive or warm-supportive fathering). However, the degree to which fathering effects are either amplified or dampened by reactive G-E processes should be equivalent across older and younger sisters. For example, suppose that an older and younger sister lived with their father for 10 and 4 years respectively (resulting in a 10-to-4 ratio of exposure to harsh-coercive fathering), and that the effects of this differential exposure were doubled by reactive G-E processes (resulting in 20 and 8 units of impact on the RSB in the older and younger sister respectively). The result is that the ratio of differential exposures and its consequences remains the same across sisters.

Among sisters who had extensive exposure to their fathers (as did all sisters in two-biological-parent families and older sisters in biologically disrupted families), the theory predicted that higher quality father-daughter relationships would reduce RSB. Secondary analyses, however,

suggested that this prediction was only supported in sisters who experienced variation around the lower end of father-daughter relationship quality. Among sisters from two-biological-parent families, who generally experienced variation around the higher end of father-daughter relationship quality, none of the simple slopes of RSB on fathering were statistically significant, regardless of whether these slopes were calculated for older or younger sisters with large or small age gaps. These results may be interpretable in a "good enough" parenting framework (Scarr, 1992). Scarr has emphasized that children's developmental trajectories are fairly robust against ordinary variations in parental behavior (e.g., variation around the high end of fathering quality) and may be most susceptible to family experiences outside of the normal range (e.g., variation in neglectful or abusive paternal behavior). Building on previous research on the impact of fathers on daughters' age at menarche (Tither & Ellis, 2008), it appears that exposure to paternal behavior in the deficient or socially deviant range has the most influence on sexual development and behavior.

The current study did not find an effect of amount of exposure within families to family disruption/father absence—irrespective of the quality of fathering—on RSB in daughters. Descriptive longitudinal studies of community samples or cohorts in New Zealand and the United States have established that earlier onset of (i.e., more exposure to) family disruption/father absence is associated with higher rates of early sexual activity and teenage pregnancy in daughters (Ellis et al., 2003). The causal status of these associations has been challenged, however, by recent analyses employing the children-of-sisters design (see Introduction), which suggest that the association between family disruption/father absence and age at first sexual intercourse may be attributable to confounded genetic and environmental risks (Mendle et al., 2009). Consistent with much past research, we found that girls from biologically disrupted/father-absent homes engaged in significantly more RSB than did girls from two-biological-parent homes (Table 1). However, using the environmentally and genetically controlled differential sibling-exposure design, we found no

support for the causal hypothesis that differences between younger and older sisters in RSB would be greatest in biologically disrupted families when there was a large age gap between the sisters (thus maximizing differential exposure to family disruption/father absence).

This null effect should be interpreted with caution, however, for several reasons. First, due to our small sample-size, we had limited power to detect within-family effects. Second, these analyses were limited to the social address of father presence-absence and did not take into account differences between sisters in the amount of interaction they had with their fathers following the divorce/separation. Nonetheless, it is worth noting that older and younger sisters' reports of post-divorce/separation contact with their fathers were substantially correlated (r [58] = .47, $p < .001$) and not significantly different from each other (paired t [57] = 1.44, ns), indicating a similar overall pattern of paternal investment in both sisters following divorce/separation. Third, and most critically, the study design necessitated a restricted range of exposure to family disruption/father absence. Because all of our families had to be together long enough to produce two children who were at least 4 years apart in age, the present research necessarily excluded girls who were either born into single-mother families or who resided in short-term, unstable separated/divorced families—the very family structures that are characterized by the highest rates of RSB in daughters (Ellis et al., 2003). In past studies, early father absence has generally been defined as occurring in the first 5 years of life (e.g., Bereczkei & Csanaky, 1996; Draper & Harpending, 1982; Ellis et al., 2003; Hetherington, 1972). In the current study, the average age of the younger sisters at time of family disruption/father absence was 6.4 years, effectively excluding early father-absent families. In other words, our within-family analyses compared younger sisters who generally experienced family disruption/father absence beginning in middle-childhood with their older sisters who generally experienced it beginning in late childhood to adolescence.

Paternal Investment Theory

As noted in the Introduction, attachment-based theories, social learning theories, evolutionary-developmental theories, and life-stress models are all consistent with the notion that high quality parent-child relationships are protective against RSB in offspring. Operating within the evolutionary-developmental framework, paternal investment theory more specifically posits a unique role for *fathers* in regulating *daughters'* sexual behavior. The theoretical basis for emphasizing father-effects is (a) that the quality and quantity of paternal investment is—and presumably always has been—widely variable across and within human societies; (b) this variation recurrently and uniquely influenced the survival and fitness of children during our evolutionary history (Geary, 2000; cf. Sear & Mace, 2008, for an opposing view); and (c) variability in paternal investment, much more than maternal investment, was diagnostic of the local mating system (degree of monogamy vs. polygyny) and associated levels of male-male competition (Del Giudice, 2009; Kanazawa, 2001). The mating system is important because more polygynous cultures and subcultures are characterized by heightened male intrasexual competition, dominance-striving, and violence, with concomitant diminution of paternal involvement and investment (Draper & Harpending, 1982, 1988). In turn, female reproductive strategies in this context are biased toward earlier sexual debut, reduced reticence in selecting mates, and devaluation of potential long-term relationships with high-investing males, all of which translate into more RSB. In total, paternal investment theory links low male parental investment to more aggressive and hypermasculine behavior in sons and more precocious and risky sexual behavior in daughters (Draper & Harpending, 1982, 1988). The assumption is that natural selection has designed boys' and girls' brains to detect and encode information about their fathers' social behavior and role in the family as the basis for calibrating socio-sexual development in gender-specific ways.

Consistent with paternal investment theory, extant correlational research has documented specific associations between quality of father-child relationships (father knowledge and oversight

of peers and activities, father-daughter closeness, low psychological control by fathers) and indices of RSB in daughters (Ream & Savin-Williams, 2005; Rodgers, 1999; Coley et al., 2009). These studies, however, have not employed causally informative designs and in most cases not employed adequate controls for maternal behavior to demonstrate *unique* fathering effects. The current research thus provides two important legs of support for paternal investment theory. First, it used a quasi-experimental design that goes beyond past correlational studies to establish a plausible causal effect of father-daughter relationship quality on RSB in daughters. Second, it very stringently controlled for maternal effects: Not only did the sisters in the study all have the same mother and live with her throughout their childhoods, but the analyses also controlled for the measured quality of mother-daughter relationships. Further, the effects of mothering versus fathering, and of global parenting quality versus fathering quality, were not interchangeable in the analyses. Thus, the current research established a unique effect of quality of fathering on RSB, above and beyond the effects of mothers and general parenting quality. These data offer support for a key assumption of paternal investment theory: that girls detect and encode information specifically about their fathers' behavior as a basis for calibrating reproductive strategies (Ellis et al., 2003; Ellis, 2004). Nonetheless, future research could provide a stronger test of the theory by investigating the differential effects of quality of father-child relationships on RSB (and aggression and dominance-striving) in daughters versus sons.

A Note on Causal Inference

Causation cannot be definitively established without a randomized experimental design. The current differential sibling-exposure design is quasi-experimental; it can only support soft rather than hard causal inferences (Tither & Ellis, 2008). For example, imagine an idiosyncratic trait, perhaps arising from a set of allelic variations, which causes both RSB and associated behavioral problems that bring about parental separation/divorce. Although such a trait should be randomly

distributed across older and younger sisters, if an older sister has the trait, it reduces the probability that the parents will stay together long enough to produce a younger sister, whereas possession of the trait by the younger sister is independent of the probability of there being an older sister. This type of trait, therefore, could be more common in younger than older sisters in the current study, which would bias parameter estimates. In total, without random assignment, the current research design enables us to make plausible, but not definitive, causal inferences.

Testing for the Influence of Fathers in Families

A potentially powerful method for demonstrating environmental causation while controlling for genetic confounds is the "natural experiment," which involves the introduction or removal of an environmental risk factor by circumstances outside of the control of the affected individuals (Rutter, 2005). The current differential sibling-exposure methodology provides a strong natural (quasi) experimental design: Older and younger sisters were differentially exposed to an environmental factor—family disruption/father absence—due to circumstances beyond their control.[2] This differential exposure created a context in which older and younger sisters had systematically diverging opportunities to be influenced the quality of father-daughter relationships. This differential exposure—the natural experiment—was confirmed by the significant diminution in father-daughter contact following divorce/separation (Figure 1), with relatively high levels of contact with fathers while living together as a family unit and relatively low levels thereafter. Consequently, the older sisters, who lived with their fathers for longer periods of time than did their younger sisters, had systematically greater exposure to their fathers' physical presence and paternal behavior. The effects of this differential exposure were then empirically evaluated.

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[2] Although it is conceivable that genetically influenced personality differences between older and younger sisters differentially affect the probability of family disruption (see above, A Note on Causal Inference), this possibility is remote. The fact that there were no main effects of birth order or age gap between sisters on RSB, and no 2- or 3-way interactions between birth order, age gap, and family type, essentially eliminates this possibility in the current study.

This methodology holds considerable promise for future research on the influence of fathers in families. There has been decades of research on fathers (see reviews in Booth & Crouter, 1998; Lamb, 2004; Tamis-LeMonda & Cabrera, 2002), but almost none of it has been causally informative. The current differential sibling-exposure methodology enabled stronger tests of the impact of fathers on age at menarche (Tither & Ellis, 2008) and RSB than had been achieved in past research, resulting in new knowledge about modifiable determinants of RSB that could be targeted for intervention. Looking forward, this method could be widely applied to other questions regarding the causal role of fathers in child development (e.g., Does greater exposure to harsh-coercive fathering cause more conduct problems in adolescents? Does more exposure to warm-supportive fathering promote better peer relationships in children?)

Limitations and Future Directions

Limitations of the present study should be noted because they provide important directions for future research. First, and foremost, the current research was based on a relatively small sample and could thus have generated unreliable parameter estimates. It will be especially important in future research to study larger numbers of biologically disrupted families in which older and younger sisters are differentially exposed to important dimensions of paternal behavior. Second, the current research employed extensive screening procedures to find qualified families that enabled relevant sibling comparisons. We do not know the extent to which the results from these screened-in families generalize to screened-out families. This is probably not a major issue in the current research, however, because (a) the demographics of our sample closely paralleled U.S population statistics and (b) the relations between variables conformed to expectations. Third, the assessments of fathering and RSB relied on retrospective reports by siblings. This is potentially problematic because such reports may be affected by time and life experience (e.g., perceptions of fathering may differ when daughters are 16 vs. 36 years old). Nonetheless, high levels of agreement between

sisters in ratings of the parenting constructs support the validity of the current assessment method. Unfortunately, there is no way to independently verify reports of RSB. Finally, the current study did not test for mediating mechanisms: factors that differ between sisters after—and as a result of—family disruption/father absence and may explain how variation in father-daughter relationships affects RSB. Following family disruption, this could include such factors as differential exposure to stepfathers and sexual abuse, differential exposure to socioeconomic conditions, different experiences of parental monitoring, and so forth.   Future research is needed to identify these intervening pathways.

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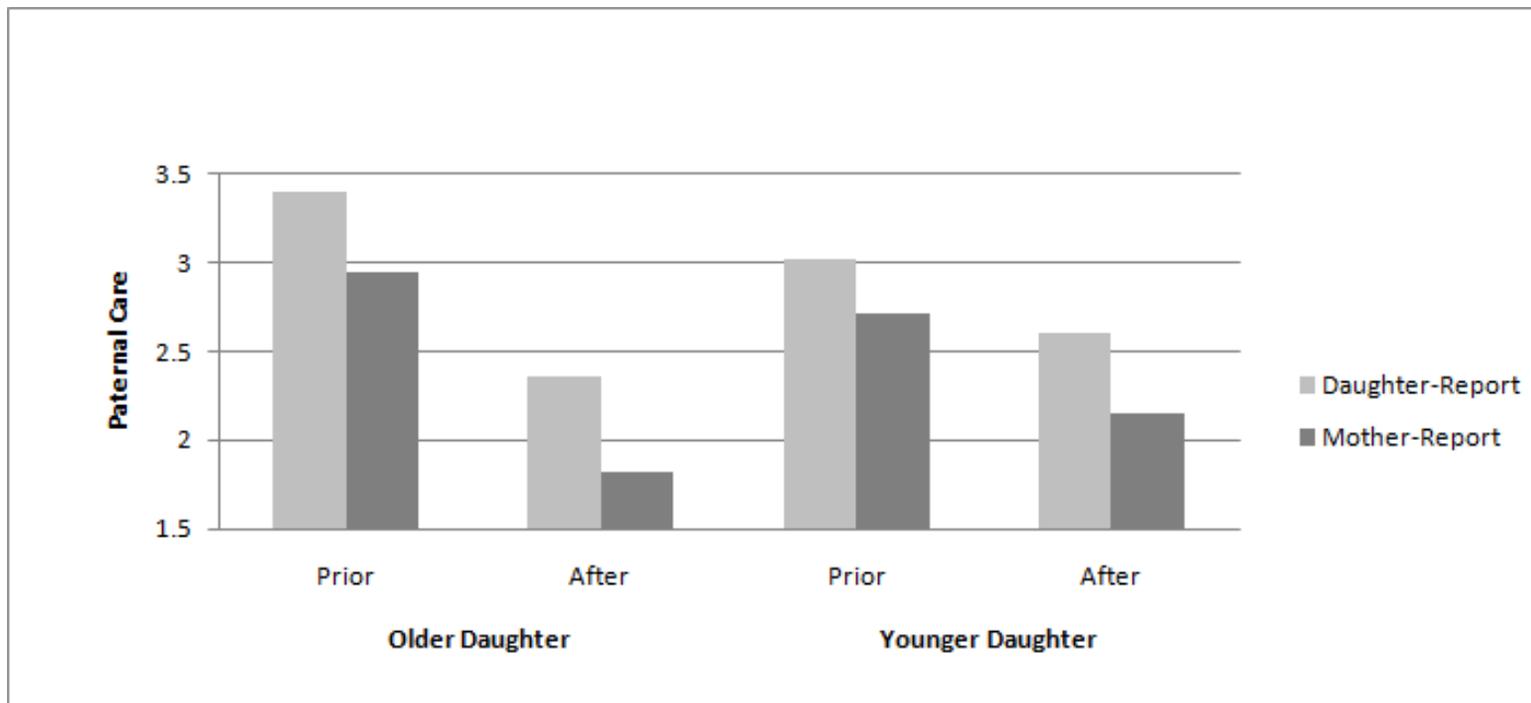the importance of age, gender and ethnic background, *Developmental Revie*w, 28, 153-224.

*Figure 1.*Mother and daughter reports of amount of paternal care prior to and after family disruption.

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The figure includes the following data table at the bottom:

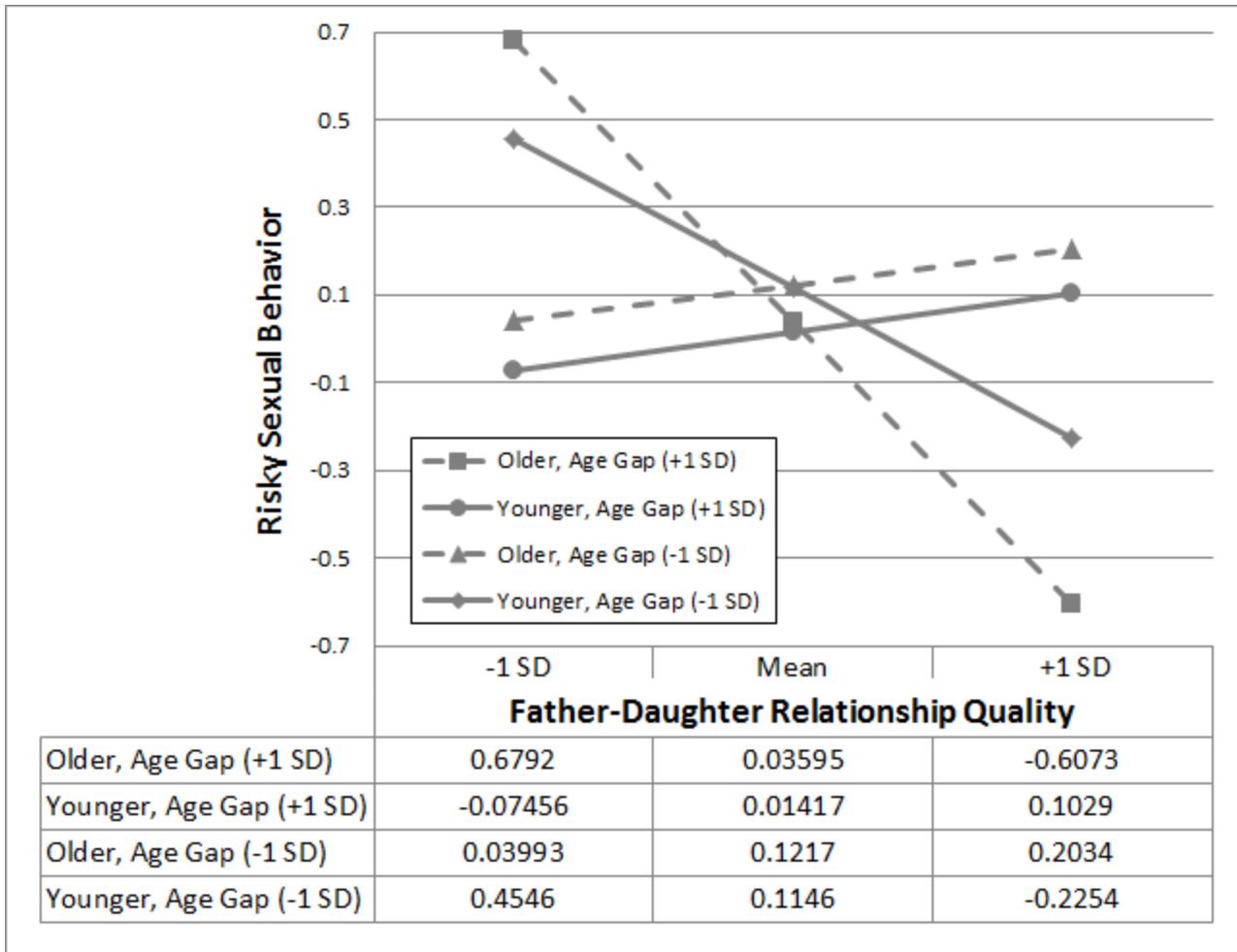|	-1 SD	Mean	+1 SD
Father-Daughter Relationship Quality			
Older, Age Gap (+1 SD)	0.6792	0.03595	-0.6073
Younger, Age Gap (+1 SD)	-0.07456	0.01417	0.1029
Older, Age Gap (-1 SD)	0.03993	0.1217	0.2034
Younger, Age Gap (-1 SD)	0.4546	0.1146	-0.2254

Figure 2. Effects of father-daughter relationship quality on risky sexual behavior in daughters in biologically disrupted families. Risky sexual behavior is standardized. The set of 12 numbers at the bottom of the figure are the predicted values for risky sexual behavior at the mean (0), low (-1 *SD* from the mean), and high (+1 *SD* from the mean) levels of father-daughter relationship quality for older and younger sisters, respectively, with large and small age gaps between them.

Table 1.

Between-Family Correlations

Measure	1	2	3	4	5	6	7	8
1. Maternal Education	--							
2. Father Occupational Status	.57**	--						
3. Race/Ethnicity	-.40**	-.34**	--					
4. Family Type	.06	-.08	.00	--				
5. Father-Daughter Rel. Quality	.23*	.23*	-.09	-.29**	--			
6. Mother-Daughter Rel. Quality	.15	.18	-.09	-.20*	.29**	--		
7. Age Difference between Sisters	.01	.09	.14	.00	.01	-.18	--	
8. Risky Sexual Behavior	-.19	-.17	-.12	.24*	-.27**	-.23*	-.04	--

Note: N = 101. Race/Ethnicity (0 = Caucasian [non-Hispanic]; 1 = Other). Family Type (0 = two-biological-parent; 1 = biologically disrupted). Correlations with race/ethnicity are point-biserial; correlations with family type are biserial. Significance tests are two-tailed. *p < .05; **p < .01

Table 2. *Parameter Estimates and Standard Errors for Models Predicting Risky Sexual Behavior*

Variables	Parameter Estimates (Standard Errors)			
	Model 1		Model 2	
Main Effects				
Birth Order of Sisters (BthOrd)	-.01	(.16)	-.01	(.16)
Family Type (FamTyp)	-.19	(.20)	-.07	(.20)
Age Gap between Sisters (AgeG)	-.02	(.13)	-.04	(.13)
Father-Daughter Relationship (FDR) Quality			-28[*]	(.12)
2-Way Interactions				
BthOrd*AgeG	-.05	(.16)	-.01	(.16)
BthOrd*FamTyp	-.23	(.25)	-.22	(.26)
AgeG*FamTyp	.05	(.20)	.10	(.20)
AgeG*FDR			-.36[**]	(.14)
BthOrd*FDR			.16	(.16)

FamTyp*FDR		.26	(.19)
3-Way Interactions			
BthOrd*AgeG*FamTyp	-.03 (.25)	-.09	(.26)
BthOrd*FamTyp*FDR		-.26	(.27)
BthOrd*AgeG*FDR		.58**	(.18)
AgeG*FamTyp*FDR		.70***	(.20)
4-Way Interaction			
BthOrd*AgeG*FamTyp*FDR		-.99***	(.29)
Covariate			
Mother-Daughter Relationship Quality		-.10	(.08)

Note: N = 101 sister pairs (202 individuals). Birth order of sisters (0 = older; 1 = younger). Family type (0 = biologically disrupted; 1 = two-biological-parent). All other covariates and main effects are continuous and mean-centered.
*$p < .05$ **$p < .01$ ***$p < .001$