Life With (or Without) Father: The Benefits of Living With Two Biological Parents Depend on the Father’s Antisocial Behavior

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The salutary effects of being raised by two married, biological parents depend on the quality of care parents can provide. Using data from an epidemiological sample of 1,116 5-year-old twin pairs and their parents, this study found that the less time fathers lived with their children, the more conduct problems their children had, but only if the fathers engaged in low levels of antisocial behavior. In contrast, when fathers engaged in high levels of antisocial behavior, the more time they lived with their children, the more conduct problems their children had. Behavioral genetic analyses showed that children who resided with antisocial fathers received a “double whammy” of genetic and environmental risk for conduct problems. Marriage may not be the answer to the problems faced by some children living in single-parent families unless their fathers can become reliable sources of emotional and economic support.

A substantial body of research has shown that, on average, children who are raised from birth in two-parent families have better cognitive and behavioral outcomes compared with children who have ever lived in single-parent families, more than 80% of which are headed by single mothers (Carlson & Corcoran, 2001; Fields & Casper, 2001; Hetherington & Clingempeel, 1992; McLanahan & Sandefur, 1994; Fryor & Rodgers, 2001). Thus, in linking poor child outcomes to family structure, some researchers and policymakers have pointed to the causal role of absent and uninvolved fathers in the development of children’s behavioral and academic problems (Blankenhorn, 1995; Popenoe, 1996). In an overview of the role of fathers in children’s development, Lamb (1997) summarized several reasons father absence is associated with poor outcomes for children, citing the emotional distress of single mothers who may receive little social support, economic stressors, perceptions of abandonment by children, and predivorce and postdivorce marital conflict.

These findings have led some researchers and policymakers to conclude that if children fare better when they are raised in two-parent families, parents should be offered incentives to get married and remain married (Horn, 2001; Popenoe, 1996). This article reviews the evidence that children raised in single-parent families experience poorer outcomes compared with children raised by two biological parents, to evaluate whether the salutary effects of being raised by two biological parents apply to all families and to consider the implications of policy designed explicitly to promote marriage.

Family Structure and Children’s Development

National survey studies of family structure and children’s outcomes consistently find that children raised in two-parent families do better than children raised in single-parent families on measures of educational achievement and adjustment. These differences arise because children in single-parent versus two-biological-parent families grow up in vastly different socioeconomic contexts and because single mothers have lower educational attainment, less social support, and poorer psychological well-being (Carlson & Corcoran, 2001; Dunn, Deater-Deckard, Pickering, & O’Connor, 1998; McLanahan & Sandefur, 1994; O’Connor, Dunn, Jenkins, Pickering, & Rabash, 2001). With respect to socioeconomic context, researchers have found that as much as 50% of the association between family structure and
adolescent and young adult outcomes, including school dropout, teen childbearing, and unemployment, can be accounted for by the fact that single-parent families have lower incomes than two-parent families (McLanahan & Sandefur, 1994). Income differentials do not tell the whole story, however, as evidenced by the fact that children in stepfamilies with incomes equivalent to those of two-biological-parent families are also at risk for a range of adverse outcomes (McLanahan & Sandefur, 1994). Researchers have found that aspects of the parent–child relationship further mediate the association between family structure and children’s outcomes. Compared with children in two-biological-parent families, those in single-parent families have more conflictual relationships with their parents (Dunn et al., 1998; O’Connor et al., 2001); receive less emotional support, cognitive stimulation, and supervision, and have less involved parents (Carlson & Corcoran, 2001; McLanahan & Sandefur, 1994).

Findings from studies of nonresident fathers and their children are consistent with these results. In a meta-analysis of 63 studies dealing with nonresident fathers and children’s well-being, Amato and Gilbreth (1999) found that children had fewer behavioral problems and more academic success when fathers paid child support, when children felt emotionally close to their fathers, and when fathers engaged in authoritative parenting practices. However, children’s well-being was not associated with how often the father and child saw one another, suggesting that the quality of the father–child relationship is a better predictor of children’s outcomes than the amount of father–child contact.

In sum, a growing consensus reveals that living in a single-parent family is, on average, a robust risk factor for children’s development, although it is also true that most children ever raised in single-parent families do not suffer long-term adversity (Hetherington & Kelly, 2002). The effects of family structure on children’s outcomes are mediated via the family environment and children’s relationships with their parents.

Is Marriage the Answer?

These findings linking family structure and children’s poor outcomes have led some researchers and policymakers to conclude that children would benefit if their parents were offered incentives to get married and remain married. Marriage, and not simply cohabitation, is a key element of this proposal. Pointing to evidence from evolutionary psychology that men and women use fundamentally different reproductive strategies, these researchers argue that the institution of marriage exerts a form of social control over men who would otherwise be unmotivated to invest emotional and economic resources in their partners and children (Popenoe, 1996). This perspective was summed up by David Popenoe, director of the Rutgers Marriage Project who, in his testimony before the U.S. House of Representatives Subcommittee on Human Resources, reported:

Left culturally unregulated, men’s sexual behavior can be promiscuous, their paternity casual, their commitment to families weak. Marriage is society’s way of engaging the basic problem of fatherhood—how to hold the father to the stronger mother-child bond. (May 22, 2001)

This message is getting through to legislators. Speaking at the Fourth National Summit on Fatherhood, U.S. President George W. Bush stated, “If we are serious about renewing fatherhood, we must be serious about renewing marriage” (June 7, 2001). Notably, a great deal of promarriage policy is being enacted via welfare reform where legislators are (a) removing welfare regulations that potentially discourage marriage, (b) establishing programs to promote healthy marriages, and (c) providing additional incentives (e.g., cash rewards) for couples to get married (Brito, 2002). For example, the state of West Virginia adds a flat $100 payment to a family’s monthly benefits if the parents are married, residing in the same household, and are both named on the assistance check. This policy has the strong support of the Department of Health and Human Services (DHHS), which oversees Temporary Aid for Needy Families, and state governments are now competing for $10 million in DHHS funds that will be divided among the 10 states that show the greatest percentage point increase in the number of children who reside in married-couple families (Federal Register, 2000).

Policy of this sort is premised on the assumption that the effects of a father’s presence are uniform across families. Yet, relatively little is known about unmarried and nonresident fathers. Existing data relate largely to fathers’ demographic characteristics. For example, the Fragile Families Study (Garfinkel, McLanahan, Tienda, & Brooks-Gunn, 2001) follows a birth cohort of approximately 4,700 U.S. children, three fourths of whom were born to unmarried parents. Approximately 75% of the fathers were interviewed shortly after their child’s birth. Preliminary results from seven cities showed that

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unwed fathers were more likely to have been younger than 20 years of age at interview, to have less education (38% had less than a high school education), and to have lower incomes than married fathers (Wilson & Brooks-Gunn, 2001). Using data from the National Longitudinal Survey of Youth, Nock (1998) found that men who had children before marriage had lower educational attainment, lower earnings, more unemployment, and were more likely to live in poverty than men who did not father children before marriage. Although this demographic profile suggests that unwed and nonresident fathers may have difficulty contributing financially to their families, an equally important issue concerns the quality of parenting that these men could provide were they to be persuaded to reside with their children.

Unfortunately, virtually no studies of nonresident and unwed fathers have gathered data on men’s personalities or behavior, but what little evidence there is suggests that some unmarried and nonresident fathers may have difficulty providing positive rearing experiences for their children. For example, compared with married fathers in the Fragile Families Study, unmarried fathers were more likely to have used illicit drugs at least several times a month (12% vs. 3%), to have engaged in partner violence (4% vs. 2%), and to have reported moderate to high levels of depression (22% vs. 10%; Wilson & Brooks-Gunn, 2001). In a recent study, Jaffee, Caspi, Moffitt, Taylor, and Dickson (2001) compared resident and nonresident young fathers (in both married and cohabiting contexts) in their mid-20s on a range of psychosocial outcomes. Controlling for marital status, they found that, compared with resident fathers, nonresident fathers had lower socioeconomic status and more unemployment, and were characterized by a low threshold for the experience of negative emotions such as fear, anxiety, and anger. Nonresident fathers also experienced more symptoms of anxiety and drug and alcohol problems and they engaged in more crime, violence, and abusive behavior toward women.

The obvious question is whether unwed fathers’ employment, relationship prospects, and psychosocial adjustment would improve if they were married to the mothers of their children. The evidence on this point is inconclusive, with most studies finding that selection effects (e.g., the father’s background and his own characteristics) account for only a portion of the link between partnership status and men’s outcomes (Horwitz & White, 1998; Maughan & Taylor, 2001; Nock, 1998). Although some researchers have found that the development of quality marital bonds facilitates men’s desistance from crime (Laub, Nagin, & Sampson, 1998), high levels of assortative mating for antisocial behavior decrease the likelihood that men will form such high-quality bonds in the first place (Krueger, Moffitt, Caspi, Bleske, & Silva, 1998; Moffitt, Caspi, Harrington, & Milne, 2002). What is clear is that simply advocating that these men marry the mothers of their children without also addressing their multiple needs might do their children and partners more harm than good.

Intergenerational Transmission of Risk for Antisocial Behavior

An understanding of absent and unmarried fathers’ personality and behavior is critical if researchers and policymakers hope to understand how a father’s presence affects his children’s outcomes. The quality of a father’s involvement matters more than his mere presence, and the studies of unmarried and nonresident fathers described earlier suggest that some of these men are characterized by behaviors that may compromise their ability to be reliable sources of financial and emotional support. Specifically, some nonresident fathers engage in a wide range of antisocial behaviors, including illegal activities, irritable and aggressive behavior, and fiscal and emotional impulsivity and irresponsibility, all of which are significant risk factors for the development of children’s conduct problems (Frick et al., 1992; Huesmann, Eron, Lefkowitz, & Walder, 1984; Loeber & Stouthamer-Loeber, 1986). Children’s conduct problems are the strongest predictor of a range of adverse outcomes in adolescence and adulthood that take their toll on both the individual and on society, including school dropout, teen childbearing, crime, and unemployment (Moffitt et al., 2002). Fathers’ antisocial behavior may increase risk for children’s conduct problems via a range of family problems, including family poverty (Moffitt et al., 2002), child and spousal abuse (Farrington, 1994; Margolin & Gordis, 2000), harsh and coercive discipline (Fagot, Pears, Capaldi, Crosby, & Leve, 1998; Patterson, Reid, & Dishion, 1992), and comorbid drug and alcohol problems (Robins, 1998). Thus, the advantages of growing up in a two-parent family may be negated when one or both parents are characterized by a history of antisocial behavior.

The offspring of antisocial fathers may also develop behavioral problems because they are at genetic, as well as environmental, risk. Children’s conduct problems are moderately heritable, meaning
that 40% to 80% of the variance in children’s antisocial behavior can be accounted for by genetic factors (Rhee & Waldman, 2002; Rutter, Silberg, O’Connor, & Simonoff, 1999). Thus, children whose resident fathers engage in high levels of antisocial behavior may be in double jeopardy for developing conduct problems. Not only are they at genetic risk, but they are also more likely to be raised in socioeconomically disadvantaged environments in which they are exposed to domestic and neighborhood violence, harsh and inconsistent discipline, and a father’s comorbid drug and alcohol problems.

The Environmental Risk (E-Risk) Longitudinal Study, which provided the data for our analyses, is ideal for answering questions about the effects of a father’s residence in the home on his children’s antisocial behavior. First, because of the study’s high-risk sampling design, there is a wide range of antisocial behavior observed among fathers in the sample, providing sufficient power to test whether the effect of a father’s presence on his children’s problem behaviors is moderated by his antisocial behavior (McClelland & Judd, 1993). Second, because the sample comprises monozygotic and dizygotic twins and their families, genetically sensitive analyses can be conducted to determine whether a father’s presence and his antisocial behavior have effects on children’s antisocial behavior independent of genetic risks (Kendler, 1993).

In sum, this study had three goals. The first was to describe fathers in an epidemiological sample who engaged in high and low levels of antisocial behavior. The second was to determine whether the effects of father presence were uniform across families. Our hypothesis was that fathers’ antisocial behavior would moderate the effect of father presence, such that when a father engaged in low levels of antisocial behavior, the less time he resided with his children, the more behavior problems his children would have. In contrast, when a father engaged in high levels of antisocial behavior, the more time he resided with his children, the more behavior problems his children would have. The third goal of the study was to determine whether the offspring of men who had a history of antisocial behavior were at both genetic and environmental risk for the development of behavior problems. To this end, our sample comprised twins and their parents so as to explore how much of the effect of a father’s antisocial behavior on his children’s behavior problems was genetically mediated and how much was mediated via the children’s rearing environment. Our hypotheses were that antisocial behavior would be moderately heritable, but that fathers’ antisocial behavior would also be environmentally associated with children’s behavior problems beyond this genetic risk. Following from these dual-risk processes, we predicted that fathers’ antisocial behavior would be associated with the worst child behavior problems when highly antisocial fathers resided with their children.

Method

Participants

Participants were members of the Environmental Risk (E-Risk) Longitudinal Twin Study, which investigates how genetic and environmental factors shape children’s development. The E-Risk sampling frame was two consecutive birth cohorts (1994 and 1995) in the Twins’ Early Development Study (TEDS), a birth register of twins born in England and Wales (Dale et al., 1998; Trouton, Spinath, & Plomin, 2002). The full register is administered by the government’s Office of National Statistics, which invited parents of all twins born in 1994 and 1995 to enroll in TEDS. Of the 15,906 twin pairs born in these 2 years, 71% joined the TEDS register. Our sampling frame excluded opposite-sex twin pairs and began with the 73% of TEDS register families who had same-sex twins.

The E-Risk Study sought a sample size of 1,100 families to allow for attrition in future years of the longitudinal study while retaining statistical power. An initial list of 1,210 families was drawn from the TEDS register to target for home visits, representing a 10% oversample to allow for nonparticipation. The probability sample was drawn using a high-risk stratification sampling frame. High-risk families were those in which the mother had her first birth when she was 20 years of age or younger. We used this sampling (a) to replace high-risk families who were selectively lost to the TEDS register via nonresponse and (b) to ensure sufficient base rates of problem behaviors given the low base rates expected for 5-year-old children. Early first child-bearing was used as the risk-stratification variable because information on mother’s age at first birth was present for virtually all families in the register, it is relatively free of measurement error, and it is a known risk factor for children’s problem behaviors (Maynard, 1997; Moffitt & the E-Risk Study Team, 2002). The high-risk sampling resulted in a final sample in which two thirds of study mothers accurately represented all mothers in the general population (15–48 years) in England and Wales in 1994 and 1995 (estimates derived from Birth Statis-
tics, 1996). The other one third of study mothers (younger only) constituted a 160% oversample of mothers who were at high risk based on their young age at first birth (15–20 years). To obtain unbiased population estimates from the sample, the data were weighted for all analyses. The sample weight was based on the inverse of the selection probability with an additional adjustment to make the weighted proportion of young mothers exactly equivalent to the overall proportion in the population (28%; Birth Statistics, 1996).

Of the 1,210 families targeted, 7 were discovered to be ineligible for inclusion in our study because the twins had moved overseas, did not speak English, were being reared by neither biological parent, or were opposite sex. Of the 1,203 eligible families, 1,116 (93%) participated in home-visit assessments when the twins were age 5 years in 1999 and 2000, 4% of families refused, and 3% were lost to tracing. Zygosity was determined using a standard zygosity questionnaire that has been shown to have a high level of accuracy (Price et al., 2000). For 15% of the sample, zygosity was determined using DNA ascertainment. The sample includes 56% monozygotic and 44% dizygotic twin pairs. Sex was evenly distributed across zygosity (49% male).

Data were collected within 120 days of the twins’ fifth birthday. Research workers visited each home in teams of two for 2.5 to 3 hr. While one interviewed the mother, the other tested the twins in sequence in a different part of the house. All research workers had university degrees in behavioral science and experience in psychology, anthropology, or nursing. Each research worker completed a formal 15-day training program on either the mother interview protocol or the child assessment protocol to attain certification to a rigorous reliability standard. With parent’s permission, questionnaires were posted to the children’s teachers, and teachers returned questionnaires for 94% of cohort children. Families were compensated for their participation.

Measures

Father’s and mother’s history of antisocial behavior was reported by the mothers, who were interviewed using the Young Adult Behavior Checklist (Achenbach, 1997), modified to obtain lifetime data and supplemented with questions from the Diagnostic Interview Schedule (Robins, Cottler, Bucholz, & Compton, 1995) that assessed the (lifetime) presence of Diagnostic and Statistical Manual (IV) (DSM-IV; American Psychiatric Association, 1994) symptoms of Antisocial Personality Disorder (ASPD). The internal consistency reliabilities of the maternal and paternal antisocial behavior scales were .90 and .95, respectively. Scores ranged from 0 to 88 \((M = 14.82, \ SD = 16.30)\) on the paternal antisocial behavior scale, and from 0 to 60 \((M = 11.29, \ SD = 9.72)\) on the maternal antisocial behavior scale. A methodological study of mother–father agreement about men’s antisocial behavior in this sample showed that the women provided reliable information about their children’s father’s behavior. The correlation between men’s and women’s reports about men’s antisocial behavior was .74 (95% confidence interval = .53–.95; Caspi et al., 2001).

Father presence refers to the percentage of the twins’ first 5 years that their biological father lived with them. On the Life History Calendars (LHC; Caspi et al., 1996), mothers reported the number of months from the children’s birth until their 5th year that the biological father resided with the children. Father presence was calculated by dividing the number of months the biological father resided with the children by the total number of months in the LHC (equivalent to the children’s age at interview). Of the fathers, 4.5% had never resided with the children and 74% had always resided with the children. On average, fathers resided with the children for 87% of their lives \((SD = 28\% )\). Four biological fathers were nonresident because they died after the children’s birth.

Father caretaking was measured by asking the mother how often in the year before the age-5 interview the children’s biological father spent time taking care of the children. Responses were scored on a 6-point scale ranging from 0 (never) to 5 (daily), \(M = 4.19, SD = 1.38\).

Father’s marital status at the birth of twins was measured by asking mothers whether they were married to or cohabiting with the biological father of the children or another partner at the time of the children’s birth. Mothers who did not have a partner at the time of the children’s birth were coded as single (6.8%). Of the mothers, 73% said they were married to the biological father at the time of the children’s birth and 19.9% said they were cohabiting with him. Fewer than 1% of the mothers said they were married to or cohabiting with a partner other than the twins’ biological father at the time of the children’s birth.

Children’s antisocial behavior was assessed with the Achenbach family of instruments (Achenbach, 1991a, 1991b). The antisocial behavior syndrome reported in this article is the sum of items from the Delinquent Behavior (“lying or cheating,” “swearing or bad language”) and Aggressive Behavior (e.g.,
physically attacks people, temper tantrums or hot temper) scales of the Child Behavior Checklist (CBCL) and the Teacher Report Form (items scored from 0 to 2). These scales were supplemented with DSM–IV items assessing conduct and oppositional defiant disorder (e.g., spiteful, tries to get revenge, uses force to take something from another child). Mother and teacher reports of children’s antisocial behavior were summed to create a composite antisocial behavior score. Scores ranged from 0 to 130 (M = 21.17, SD = 16.27). Mother and teacher reports of antisocial behavior correlated .29, p < .001. Correlations of this magnitude are typical in studies of children’s behavioral and emotional problems (Achenbach, McConaughy, & Howell, 1987). The internal consistency reliability of the combined score was .94.

To assess child behavior problems in the clinical range, we derived diagnoses of conduct disorder (CD) on the basis of mothers’ and teachers’ reports of children’s behavior problems, using the Achenbach family of instruments and additional DSM–IV items assessing CD. Fourteen of 15 DSM–IV symptoms of CD were assessed (forced sexual activity was age inappropriate), covering aggressive and nonaggressive conduct problems, deceitfulness or theft, and rule violations. A child was considered to have a given symptom if either the mother or the teacher scored the symptom as being “very true or often true” (score = 2) of the child in the past 12 months. We counted a symptom as present if there was evidence of it from either source, following evidence that this approach enhances diagnostic validity (Bird, Gould, & Staghezza, 1992; Piacentini, Cohen, & Cohen, 1992). Symptom counts ranged from 0 to 11 (M = .47, SD = 1.20). Consistent with DSM–IV criteria, children with three or more symptoms were assigned a diagnosis of CD (unweighted, the prevalence of CD in the sample was 8.5%; weighted to represent the population, it was 6.6%). Within this group, a smaller number of children with five or more symptoms met criteria for severe CD (un-weighted prevalence, it was 3.4%; weighted to represent the population, it was 2.5%).

Results

In the first section, we describe fathers who are high or low in antisocial behavior. In the second section, we describe whether the effect of father presence on children’s antisocial behavior is moderated by fathers’ antisocial behavior. In the third section, we ask whether the offspring of antisocial fathers are at both genetic and environmental risk for the development of behavior problems. Correlations among the continuous measures used in the analyses are reported in Table 1.

What Characterizes High- and Low-Antisocial Fathers?

For illustrative purposes, we defined fathers with high levels of antisocial behavior as those who scored at or beyond the 85th percentile of the unweighted antisocial behavior distribution (n = 171). Fathers with low levels of antisocial behavior were defined as those who scored at the 15th percentile and lower of the unweighted distribution (n = 167). Weighting the distribution of fathers’ antisocial behavior before comparing the top and bottom 15th percentiles would have resulted in there being unequal numbers of fathers in the two groups. High- and low-antisocial fathers and the mothers of their children differed on a number of behaviors that might affect the development of children’s antisocial behavior (Table 2). First, most high-antisocial fathers’ behavior fell in the clinical range whereas low-antisocial fathers’ behavior did not. According to DSM–IV criteria, a diagnosis of ASPD is made if an individual has a history of childhood CD and meets three or more of the Criterion A symptoms (Table 3). High-antisocial fathers were significantly more likely to meet this criterion than low-antisocial fathers, \( \chi^2(1) = 318.58, p \leq .001. \)

Table 1

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<td>3. Father’s presence</td>
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<td>4. Father’s caretaking</td>
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<td>-.22***</td>
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<td>5. Child’s antisocial behavior</td>
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***p ≤ .001.
Table 2 also shows that the high- and low-antisocial fathers differed significantly in the amount of time they resided with their children, $t(336) = 17.41, p \leq .001$. The high-antisocial fathers were less likely to spend time taking care of their children, $w^2(5) = 168.66, p \leq .001$, but not simply because they were more likely to be nonresident; even among the fathers who had resided with their children for more than 90% of the children’s lives, high-antisocial fathers were less likely to take care of the children on a daily or weekly basis (77%) than low-antisocial fathers (97%).

High-antisocial fathers were less likely to have been married to the children’s mother when the children were born, $w^2(1) = 99.08, p \leq .001$. Finally, the mothers of children born to high-antisocial men reported higher levels of their own antisocial behavior as compared with the mothers of children born to low-antisocial men, $t(336) = -5.75, p \leq .001$. All analyses were performed again on the weighted sample and the results were unchanged.

**Does Fathers’ Antisocial Behavior Moderate the Association Between Father Presence and Children’s Behavior Problems?**

A hierarchical linear regression analysis was conducted to determine the effects of fathers’ antisocial behavior and fathers’ presence on child antisocial behavior (see Table 4; Baron & Kenny, 1986) and fathers’ presence ($r = .30, p \leq .001$) were significantly correlated with child behavior problems (Table 1). The predictor variables were centered (i.e., expressed as deviations from the mean; Aiken & West, 1991). Because all twins were included in the analyses, all of the ordinary least squares regression analyses were conducted using the sandwich variance estimator to correct for the nonindependence of data from children in the same family (StataCorp, 1999). Analyses were performed again, selecting 1 child from each twin pair at random. Results were unchanged. At the first step, we asked whether fathers’ antisocial behavior and father presence independently predicted child
behavior problems. The model was estimated as,

\[
\text{CHILDA\text{SB}} = \beta_1 + \beta_2(\text{DADASB}) + \beta_3(\text{DADHOME}) + e,
\]

where \text{CHILDA\text{SB}} refers to child antisocial behavior problems; \text{DADASB} and \text{DADHOME} refer to fathers’ antisocial behavior and father presence, respectively; and \text{e} refers to error. Fathers’ antisocial behavior significantly predicted elevated levels of child antisocial behavior problems, \(b = .32, p \leq .001\), but father presence did not when fathers’ antisocial behavior was controlled, \(b = 1.80, p = .33\) (Table 4, Model 1). At the second step, we asked whether the effect of father presence was moderated by fathers’ antisocial behavior. Thus, the interaction between fathers’ antisocial behavior and father presence was entered and the model was estimated as,

\[
\text{CHILDA\text{SB}} = \beta_1 + \beta_2(\text{DADASB}) + \beta_3(\text{DADHOME}) + \beta_4(\text{DADASB} \times \text{DADHOME}) + e.
\]

The interaction was statistically significant, \(b = .28, p \leq .001\) (Table 4, Model 2). Figure 1 plots the interaction and shows the simple slopes for the effect of father presence on child antisocial behavior at three values of the fathers’ antisocial behavior distribution: the 15th, 50th, and 85th percentiles. The figure shows that at low and median levels of fathers’ antisocial behavior, father presence was negatively associated with children’s antisocial behavior, such that the longer a father resided with his child, the less antisocial behavior the child had. However, at high levels of fathers’ antisocial

Note: Model 1 contains main effects only and Model 2 adds the interaction term between fathers’ antisocial behavior and father presence. ASB stands for antisocial behavior.

*aLogistic regression coefficients.

\*p \leq .05.

\***p \leq .001.
behavior, father presence was positively associated with child antisocial behavior, such that the longer a father resided with his child, the more antisocial behavior the child had. Post hoc analyses tested whether these three simple slopes were significantly different from zero (Aiken & West, 1991). The effect of father presence on child antisocial behavior was statistically significant and negative when evaluated at the 15th percentile of the fathers’ antisocial behavior distribution, $t(1103) = -2.34, p \leq .05$, and statistically significant and positive when evaluated at the 85th percentile of the fathers’ antisocial behavior distribution, $t(1103) = 2.11, p \leq .05$. The effect of father presence was not significant at the 50th percentile, $t(1103) = -1.58, ns$. Statistical sensitivity analyses were conducted to test whether the effects of father presence were significantly different from zero at more and less extreme cutoffs of the father antisocial behavior distribution. The pattern of significant simple effects described earlier was evident at more stringent cutoffs of the father antisocial behavior distribution (e.g., the top and bottom 5th and 10th percentiles). However, at less extreme cutoffs, the effect of father presence on twin antisocial behavior was only significant at low levels of fathers’ antisocial behavior (e.g., the 35th percentile and below). In sum, at high levels of fathers’ antisocial behavior (i.e., at or above the 85th percentile), father presence was associated with elevated levels of children’s behavior problems whereas at low levels of fathers’ antisocial behavior (i.e., at or below the 35th percentile), father presence was associated with reduced levels of children’s behavior problems.

We conducted four additional analyses to test the robustness of the interaction between fathers’ antisocial behavior and father presence. First, we tested whether fathers’ antisocial behavior moderated the effect of father presence, controlling for the presence of nonbiological father figures in the home. Second, we tested whether fathers’ antisocial behavior moderated the effect of father presence, controlling for maternal antisocial behavior. Third, we tested whether the interaction between fathers’ antisocial behavior and father presence predicted child behavior problems in the clinical range. Fourth, we tested whether fathers’ antisocial behavior moderated a more fine-grained measure of his involvement, such as his caretaking behavior.

Controlling for the presence of nonbiological father figures. Children whose biological fathers are not resident in the home sometimes reside with another male, such as a stepfather or a mother’s cohabiting partner. Thus, father absence may be associated with variations in a child’s behavior problems because the child is exposed to variations in caretaking provided by the mother’s partner. To assess this, we reestimated the models, controlling for the number of months since the children’s birth that a partner other than the children’s biological father resided with the family. The results were unchanged (Table 4).

Controlling for maternal antisocial behavior. Population studies of psychiatric disorder have found evidence of substantial assortative mating (i.e., spousal similarity) for antisocial behavior (Galbaud du Fort, Bland, Newman, & Boothroyd, 1998). The correlation between fathers’ and mothers’ antisocial behavior in our sample was .53, consistent with estimates from other studies (Krueger et al., 1998). Thus, children whose biological father engages in high levels of antisocial behavior are likely to reside with a mother who engages in high levels of antisocial behavior herself. Children who reside with two such parents may be at the greatest risk of developing behavior problems. To assess whether mothers’ antisocial behavior accounted for the interaction between father presence and fathers’ antisocial behavior, we reestimated the models, controlling for mothers’ antisocial behavior. Although controlling for mother’s antisocial behavior reduced the effect of father’s antisocial behavior by approximately 50% (i.e., from $b = .32$ to $b = .14$), the effect of fathers’ antisocial behavior on children’s behavior problems and the interaction between his behavior and his presence remained statistically significant (Table 4).

Assessing child behavior problems in the clinical range. We assessed whether the interaction between
father presence and fathers’ antisocial behavior predicted severe child behavior problems (i.e., a diagnosis of CD), as opposed to behavior problems in the normal range. In a hierarchical logistic regression analysis, we first regressed CD on fathers’ antisocial behavior and father presence (Model 1). We then regressed CD on the main effects plus the interaction between fathers’ antisocial behavior and father presence (Model 2). The results were consistent with those predicting the range of child behavior problems (Table 4). Holding fathers’ antisocial behavior and father presence at their means, the predicted probability of a child having a diagnosis of CD was 6%. Figure 2 shows the predicted probabilities of a child being diagnosed with CD as a function of the fathers’ antisocial behavior and his presence in the home. Of the fathers, 4.5% had never lived with their children and 74% had always lived with their children. When fathers’ antisocial behavior was high (i.e., at the 85th percentile), the predicted probability of a child having a diagnosis of CD was 16% (95% confidence interval = 12%–22%) if the father had always lived with the family and 9.6% (95% confidence interval = 6%–15%) if the father had never lived with the family. In contrast, when a father’s antisocial behavior was low (i.e., at the 15th percentile), the probability of a child having a diagnosis of CD was 3.1% (95% confidence interval = 2%–4%) if the father had always lived with the family and 4.7% (95% confidence interval = 2%–10%) if the father had never lived with the family.

Assessing fathers’ caretaking behavior versus father presence. Our measure of father presence provided only a rough index of fathers’ involvement in their children’s lives. Even when fathers do not reside with their children, they may be involved in their children’s care. Thus, we performed the analyses again, replacing the father presence variable with the variable measuring father caretaking (the caretaking variable was centered). The results replicated those for father presence (Table 4). Figure 3 shows that when fathers’ antisocial behavior was high (at the 90th percentile of the fathers’ antisocial behavior distribution), children whose fathers cared for them on a daily basis had the worst behavior problems, $t(1103) = 2.69$, $p \leq .05$. When fathers’ antisocial behavior was low (at the 15th percentile of the fathers’ antisocial behavior distribution), children whose fathers never took care of them had the worst behavior problems, $t(1103) = -2.94$, $p \leq .05$. 

![Figure 2](image2.png)

Figure 2. Predicted probability of child’s conduct disorder diagnosis as a function of fathers’ antisocial behavior and residence with child.

![Figure 3](image3.png)

Figure 3. Child’s antisocial behavior as a function of father’s antisocial behavior and time father spends taking care of child.
Are the Children of Antisocial Fathers Getting a Double Whammy of Genetic and Environmental Risk?

DeFries–Fulker (DF; 1985) regression analyses were conducted to determine the extent to which individual differences in children’s behavior problems could be accounted for by genetic influences, parental antisocial behavior, and father presence. DF analysis uses kinship-pair data (e.g., data from twins or adoptive siblings) to separate heredity and shared environmental influences in a regression framework (DeFries & Fulker, 1985). For examples of this approach in the developmental psychology and social demography literatures, see Rodgers, Kohler, Kyvik, and Christensen (2001) and Rodgers, Rowe, and Li (1994). The DF approach was chosen over a maximum likelihood model-fitting approach because the former is conceptually simpler to understand, the parameter estimates of genetic and environmental effects derived from DF regression models are equivalent to those from structural equation models of twin data (Cherny, DeFries, & Fulker, 1992), and the DF models more easily incorporate measured variables. The sandwich variance estimator was used to correct for the nonindependence of twin observations, as recommended by Kohler and Rodgers (2001). The equation for the DF regression model is as follows:

$$\text{ASB}_twin = \beta_1 + \beta_2 (R) + \beta_3 (\text{ASB}_twin) + \beta_4 (R' \text{ASB}_twin) + e.$$ 

In this equation, \(\text{ASB}_twin\) represents Twin 1’s score on the antisocial behavior composite; \(\beta_1\) represents the constant term; \(R\) represents the coefficient of genetic relatedness (1.0 for monozygotic twins, 0.5 for dizygotic twins); \(\text{ASB}_twin\) represents Twin 2’s score on the antisocial behavior composite; \(\beta_4\) represents the population heritability estimate \(h^2\) because, when it is statistically significant, it demonstrates that Twin 1 and Twin 2’s resemblance for antisocial behavior is conditioned on their degree of genetic relatedness; and \(\beta_3\) estimates shared environmental variation because it represents the twin’s resemblance for antisocial behavior independent of genetic resemblance (Rodgers & McGue, 1994).

DF regression analyses are underpowered to detect effects of the latent shared environment in the classical twin design unless sample sizes include more than 1,000 twin pairs (Martin, Eaves, Kearsey, & Davies, 1978). Statistical power to detect potential effects of the shared environment is increased, however, when the putative environmental factor is measured and its effects are estimated (Kendler, 1993). Thus, at the second step, the formula was expanded to estimate the effects of parental antisocial behavior and father presence on children’s antisocial behavior.

$$\text{ASB}_twin = \beta_1 + \beta_2 (R) + \beta_3 (\text{ASB}_twin) + \beta_4 (R' \text{ASB}_twin) + \beta_5 (DADASB) + \beta_6 (MUMASB) + \beta_7 (DADHOME) + e.$$ 

Here, \(\beta_5\) and \(\beta_6\), respectively, represent the effect of fathers’ and mothers’ antisocial behavior on individual differences in children’s behavior problems, and \(\beta_7\) estimates the effect of fathers’ presence in the home.

At the final step, the interaction between fathers’ antisocial behavior and father presence was entered.

$$\text{ASB}_twin = \beta_1 + \beta_2 (R) + \beta_3 (\text{ASB}_twin) + \beta_4 (R' \text{ASB}_twin) + \beta_5 (DADASB) + \beta_6 (MUMASB) + \beta_7 (DADHOME) + \beta_8 (DADASB' DADHOME) + e.$$ 

In this formula, \(\beta_8\) estimates the effect of the interaction between fathers’ antisocial behavior and father presence, controlling for all main effects, including the effect of mothers’ antisocial behavior.

Table 5 presents the results of the DF regression analysis. Five findings are highlighted. First, focusing on Model 1, childhood antisocial behavior is highly heritable with an initial estimate of .92 (95% confidence interval = .70–1.14). However, this estimate is inflated because, in the DF regression framework, estimates of how much genetic and shared environmental influences (i.e., \(\beta_3\) and \(\beta_4\), respectively) account for variation in children’s antisocial behavior are not bounded at 0 and 1. When these estimates are bounded (e.g., using maximum likelihood estimation; Neale & Cardon, 1992), the heritability estimate drops to .73 (95% confidence interval = .68–.78), meaning that 73% of the variance in behavior problems in this sample of 5-year-old children is accounted for by genetic influences. The magnitude of this heritability estimate is consistent with estimates from other studies of antisocial behavior in toddlers and preschool-aged children (Arseneault et al., in press). With the exception of Schmitz et al. (1999), who found a heritability of .30 for 4-year-old children’s CBCL externalizing problems, other researchers have found heritabilities for CBCL externalizing ranging from 50% to 75% (van den Oord, Verhulst, & Boomsma, 1996; van der Valk, Verhulst, Neale, & Boomsma 1998). Second, shared environmental influences did not account significantly for variation in childhood antisocial behavior. Thus, this effect was dropped from the analysis (Model 2), with a
subsequent drop in the heritability estimate to its bounded level. Third, focusing on Model 3, both fathers’ and mothers' antisocial behavior were positively associated with children’s behavior problems. Thus, even controlling for genetic transmission, parental antisocial behavior had effects that manifested themselves in the child’s environment. Fourth, the effect of father presence was not statistically significant once we controlled for the influence of genes and parental antisocial behavior on children’s behavior problems. Fifth, focusing on Model 4 we found that the interaction between fathers’ antisocial behavior and father presence was statistically significant, even after controlling for genetic influences on children’s behavior problems.

To evaluate whether dropping the shared environmental term from the models substantively influenced the results, the models were performed again, retaining the shared environmental term. The results were unchanged with respect to the magnitude and significance of the effects of parental antisocial behavior, father’s presence, and the interaction between fathers’ antisocial behavior and father’s presence (analyses available on request). Figure 4 graphs the interaction and shows that when fathers’ antisocial behavior is low, children whose fathers have never resided with the family have the most behavior problems. In contrast, when fathers’ antisocial behavior is high, children whose fathers have always resided with them have the most behavior problems. Thus, children born to highly antisocial men experience a double whammy of risk when their father resides with the family. First, they are at genetic risk because childhood antisocial behavior is highly heritable. Second, they are at environmental risk because fathers’ antisocial behavior accounts for variation in children’s behavior problems independent of genetic influences. This suggests that fathers who are characterized by high levels of antisocial behavior not only pass on “risky” genes to their children, as evidenced by the high heritability coefficient reported earlier, but also provide rearing experiences that contribute to the development of their children’s antisocial behavior.

**Discussion**

These data yielded two findings with implications for research and policy. First, in families in which fathers engage in very high levels of antisocial behavior, children have the worst behavior problems when the father resides in the home. Under these circumstances, children’s behavior problems reach clinically significant levels and their behavior is significantly worse than among their peers whose fathers also engage in high levels of antisocial behavior but do not reside with their children. Second, when highly antisocial fathers reside with the family, children experience a double whammy of risk for antisocial behavior. They are at genetic risk because antisocial behavior is highly heritable. In addition, the same parents who transmit genes also provide the child’s rearing environment. We found that a father’s antisocial behavior accounted for his children’s behavior problems independent of any genetic risk he may have imparted, particularly when he resided with the family and spent time taking care of the children. Given the importance of fathers’ antisocial behavior as an environmental risk factor, further research is needed to determine

### Table 5

<table>
<thead>
<tr>
<th></th>
<th>Model 1 $b$ (95% confidence interval)</th>
<th>Model 2 $b$ (95% confidence interval)</th>
<th>Model 3 $b$ (95% confidence interval)</th>
<th>Model 4 $b$ (95% confidence interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$R_{ASB_{twin}}$ (estimate of shared environment)</td>
<td>0.17 (-0.37; 0.04)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>$R^{2}_{ASB}$ ($h^2$)</td>
<td>0.92** (.70; 1.14)</td>
<td>0.73*** (.68; .78)</td>
<td>0.64*** (.57; .70)</td>
<td>0.63*** (.57; .70)</td>
</tr>
<tr>
<td>Father’s ASB</td>
<td>0.07** (.04; .11)</td>
<td>0.09*** (.05; .13)</td>
<td>0.26*** (.19; .32)</td>
<td>0.25*** (.18; .31)</td>
</tr>
<tr>
<td>Mother’s ASB</td>
<td>0.26*** (.19; .32)</td>
<td>0.29*** (.18; .31)</td>
<td>0.08 (-1.70; 1.87)</td>
<td>-0.76 (-2.58; 1.06)</td>
</tr>
<tr>
<td>Father presence</td>
<td>0.08 (-1.70; 1.87)</td>
<td>-0.76 (-2.58; 1.06)</td>
<td>0.09** (.02; .16)</td>
<td></td>
</tr>
</tbody>
</table>

Note. ASB stands for antisocial behavior. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$. 

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whether children model a father’s antisocial behavior or whether the effects of his behavior are mediated via other aspects of the family environment, such as his relationship with the children’s mother, his parenting behavior, his abuse of his children, or his job instability. Once further research has identified likely mediating mechanisms, experimental programs might be designed to test whether these mechanisms represent component causes of children’s conduct problems (Rutter, Pickles, Murray, & Eaves, 2001).

Implications for Theory

Our finding that a father’s presence is beneficial to children only when he engages in low levels of antisocial behavior is consistent with a large body of theoretical and empirical work suggesting that the quality of the parenting children experience more strongly influences their adjustment than the type of family in which they are raised (Bornstein, 1995). Previous research has shown that the effects of family structure tend to be mediated through other aspects of the family environment and the parent–child relationship, including the parents’ behavior. Our results show that characteristics of the parent not only mediate the effects of family structure on children’s adjustment, but also moderate those effects.

Implications for Policy

As noted in the introduction, promarriage policy is increasingly enacted via U.S. welfare reform, which is currently up for renewal in 2002. Policy that is designed to promote marriage among low-income couples must consider the very real fears that cause some low-income single mothers to consider nonmarriage a better alternative to marriage. Low-income single mothers are concerned with both monetary and nonmonetary factors in their decision to marry, including a spouse’s greater-than-minimum-wage income, job stability, and source of income, as well as their own control over household decisions, mistrust of men, and fear of domestic violence (Edin, 2000). Policy that promotes marriage without simultaneously addressing these myriad concerns is unlikely to persuade some single mothers that marriage is in their own or their children’s best interest. Moreover, marriage is unlikely to provide a turning point out of antisocial behavior for men if women cannot be convinced that such men make good marriage material. Findings from our study demonstrate that some mothers may be justified in their concerns about the negative influence some fathers may exert over their children’s adjustment. The timing of interventions designed to improve employment or marital prospects among nonresident fathers may be crucial in demonstrating their effectiveness. For example, fathers may be most motivated to take up multiple aspects of an intervention (and thus increase the chances that the intervention will prove effective) shortly after the birth of a child, when many couples in “fragile” families express hope that their relationship can succeed (Carlson & McLanahan, 2002).

Our findings are relevant not only for policy concerning fathers, but also for policy and prevention concerning at-risk children. We found that the offspring of antisocial fathers were already at genetic risk for the development of behavior problems. These children should be targeted as early as possible for interventions that involve the entire family to reduce the possibility that environmental risk associated with parents’ antisocial behavior will exacerbate children’s genetic vulnerabilities.

Finally, the kind of extreme antisocial behavior (i.e., in the top 15th percentile and higher of the distribution) that was linked to children’s conduct problems in our sample does not characterize most fathers in the population. Should we then question policy that promotes children being raised by two married, biological parents on the basis of an extreme subset of fathers who engage in criminal behavior, lie to their partners, get in fights, are irresponsible and impulsive, and do not feel sorry
about any of it? Yes, for three reasons. First, at least in early childhood, when fathers engaged in high levels of antisocial behavior, their presence was linked to children’s conduct problems in the clinical range. That is, among children whose fathers engaged in high levels of antisocial behavior and had always resided with the family, 16% were diagnosed with CD at age 5 years. In contrast, among children whose fathers engaged in low levels of antisocial behavior and had never lived with the family, fewer than 5% were diagnosed with CD at age 5 years. Thus, to the extent that policymakers are concerned with protecting children, the greatest attention in early childhood should be paid to those whose fathers engage in high levels of antisocial behavior and who reside with the family, especially considering that clinically significant early-childhood conduct problems are a strong predictor of antisocial behavior that persists across the life course (Moffitt et al., 2002).

Second, despite the fact that fathers who engage in high levels of antisocial behavior make up a small proportion of fathers overall, they are responsible for a disproportionate number of births. For example, Moffitt and colleagues (2002) found that although men who engaged in high levels of antisocial behavior constituted only 10% of a birth cohort, they accounted for 27% of the babies fathered by the time the men were age 26 years.

Third, our findings do not suggest that most children’s antisocial behavior would be reduced if they were raised by two biological parents. That is, a father’s presence had no effect whatsoever on children’s behavior problems when a father’s antisocial behavior was in the average range (between the 35th and 85th percentiles). This suggests that for a great many children, having a father resident in the home makes little difference in terms of their behavioral adjustment. We would hasten to add that it may indeed be the case that a father’s presence in the home reduces the likelihood of other poor outcomes that we did not assess in our sample (e.g., school failure, low self-esteem). However, in terms of young children’s antisocial behavior, father presence appeared to buffer children in only about a third of the families in our sample, and moreover, these are not the families at whom promarriage policy is targeted; 96% of fathers in this group had been married to or residing with the mother of their children during the children’s first 5 years. The important point is that the effect of a father’s presence on children’s adjustment depends a great deal on other characteristics of the father.

Implications for Methodology

Historically, data on fathers have not often been collected because (a) mothers are considered the primary caretakers of their children and their parenting is considered the more important factor in children’s adjustment and (b) with respect to absent fathers, it is often difficult to locate them, particularly when samples are selected on the basis of children. Our findings suggest that data on fathers, as well as mothers, are crucial for understanding the effects of family structure on children’s outcomes. Our findings also show that a sufficient range of fathers’ behavior must be observed to capture the complex interactions between a father’s behavior and family structure. Two features of our study facilitated our ability to do this. First, the high-risk nature of our sampling design meant that the full range of fathers’ antisocial behavior was observed and that sufficient base rates of antisocial behavior (among fathers and children) were observed such that we had adequate statistical power to detect moderator effects (McClelland & Judd, 1993). Second, by querying mothers about fathers’ behavior, we mitigated bias associated with nonresponse (Berk, 1983). The fathers who engage in the most antisocial behavior are generally the most difficult to interview. Thus, in studies that rely solely on data from fathers who are willing to be interviewed, the men who engage in the most antisocial behavior will be missing and the range of observed antisocial behavior will be restricted, resulting in lower power to detect moderator effects and biased statistical estimates. Although the best option is to collect data about fathers from fathers themselves, when this is not feasible, mothers can provide reliable reports about fathers’ antisocial behavior (Caspi et al., 2001) and their experience of men’s violence (Moffitt et al., 1997).

Limitations

This study is characterized by several limitations. First, children’s behavior problems were measured at a single point in time. It is possible that the effects of a father’s presence and his antisocial behavior on children’s behavior problems may change over time. Indeed, it is unlikely that many of the fathers who engaged in high levels of antisocial behavior and who were resident for the twins’ first 5 years would remain present throughout the twins’ childhood and adolescence. Moreover, the stability of a father’s presence or absence may matter more than the
overall proportion of the child’s life that the father is resident.

Second, our sample comprised 5-year-old children and their parents living in the United Kingdom, yet our findings were discussed primarily in terms of their relevance to U.S. family policy. Current base rates of CD in children under age 11 years are similar in the United States (2.4%; Lahey et al., 2000) and the United Kingdom (4.4%; Melzer, Gatward, Goodman, & Ford, 2000), although the prevalence of single-parent families is currently higher in the United States (31%; Fields & Casper, 2001) versus the United Kingdom (21%; Social Trends, 2001). Moreover, the effects of family structure on children’s adjustment are the same across U.S. and U.K. samples (McLanahan & Sandefur, 1994; Pryor & Rodgers, 2001). Nevertheless, future research will be needed to determine whether our findings will generalize to other cohorts in other nations, particularly the United States.

Third, the children in our sample were twins. Although twins and singletons do not differ in mean levels of behavior problems (Gjone & Novik, 1995; Kendler, Martin, Heath, & Eaves, 1995; Levy, Hay, McLaughlin, Wood, & Waldman, 1996; Moilanen et al., 1999; Simonoff et al., 1997; van den Oord, Koot, Boomsma, Verhulst, & Orleveke, 1995) parents of twins may experience more economic and social stressors than parents of singletons (Spillman, 1984). Such stressors may cause fathers of twins to engage in more antisocial behavior relative to fathers of singletons or cause the effects of a father’s presence (or absence) on children’s antisocial behavior to be magnified. Nevertheless, it must be noted that all the fathers in our sample were fathers of twins, so this factor was held constant in comparisons between high- and low-antisocial fathers. It may be that mean levels of social stressors differ in families of twins versus singletons, but that the processes by which fathers’ antisocial behavior and father presence interact to influence children’s behavior problems may be the same. Replications in singleton samples will address this issue.

Fourth, data on fathers’ antisocial behavior, caretaking, and presence in the home were collected from mothers and not from fathers themselves. However, validation work has shown that father’s and mother’s reports of father’s antisocial behavior are highly correlated (Caspi et al., 2001).

Conclusion

This study of father’s antisocial behavior provides new evidence that children do not always benefit from growing up in two-parent families. A narrow focus on family structure without a parallel focus on who is raising the children may do some children more harm than good.

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