

The Intergenerational Link to Antisocial Behavior: Effects of Paternal Contact

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We investigated the relationship between paternal antisocial behavior and child conduct problems and we tested whether the degree of contact between father and child moderated the intergenerational link to antisocial behavior. Subjects were 92 children between the ages of 6 and 13 referred to an outpatient mental health clinic. There was a significant association between Antisocial Personality Disorder (APD) in fathers and a diagnosis of conduct problems in their children. However, the relationship between paternal APD and conduct problems in offspring was not dependent on whether or not the father was in the home or on the degree of paternal contact with their child. The theoretical implications of these findings for explaining the intergenerational link to antisocial behavior in terms of observational learning were discussed.

KEY WORDS: paternal antisocial behavior; childhood conduct problems; modeling theory; familial transmission.

The intergenerational link to antisocial behavior is a consistent finding in research and one that has long intrigued social scientists and policy makers alike. Early studies tended to focus on legal definitions of antisocial behavior and these studies consistently found a link between delinquency in children and criminality in their parents (Farrington, 1978; Glueck & Glueck, 1968; McCord 1979; Osborn & West, 1979; Robins, West, & Herjanic, 1975; Wilson, 1975). This research also found that this link is independent of socioeconomic status (Farrington, 1978; Glueck & Glueck,

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1968; McCord, 1979), neighborhood (Glueck & Glueck, 1968), and intelligence (Farrington, 1978; Glueck & Glueck, 1968; Osborn & West, 1979). More recent studies have focused on psychiatric definitions of antisocial disorders. Like studies of criminality, children diagnosed with antisocial disorders (e.g., Conduct Disorder and Oppositional Defiant Disorder) were significantly more likely to have parents, most often fathers, with antisocial disorders (e.g., Antisocial Personality Disorder) than were children without conduct problems (Biederman, Munir, & Knee, 1987; Faraone, Biederman, Keenan, & Tsuang, 1991; Frick et al., 1992; Hamdan-Allen, Stewart, & Beeghly, 1989; Lahey et al., 1988; Stewart, deBlois, & Cummings, 1980).

Whereas research has been clear in documenting the presence of an intergenerational link to antisocial behavior, it has been less helpful in uncovering the mechanisms through which the transmission takes place. There are three common models that have been proposed to explain this link. First, genetic predispositions have been proposed as underlying, at least in part, this intergenerational link to antisocial behavior (Jarey & Stewart, 1985). Second, this link may be mediated through dysfunctional parenting practices, with antisocial parents being less able to provide appropriate child-rearing experiences for their children (Patterson & Capaldi, 1991). Third, it is possible that antisocial parents can model inappropriate behavior which is learned by their children through an observational learning process (Bandura & Walters, 1963).

While modeling of antisocial behavior is a frequently cited mechanism through which antisocial behavior is passed from one generation to the next, it is the least studied of the three causal models. One of the difficulties in studying potential effects of modeling is the various mechanisms through which observational learning can take place. For example, in their seminal work on observational learning, Bandura and Walters (1963) describe how learning can take place without actual exposure to a model, such as through imagined models or simply through hearing descriptions of a model. However, the greater the degree of exposure to a model, the more likely the learning is to occur (Bandura, 1977). For example, Eron (1982) found that the more exposure children had to televised violence, the higher their rated levels of aggression.

Based on this social learning process, one would predict an interaction between the presence of an antisocial model and the degree of contact a child has with the model for predicting the likelihood that a child will show antisocial behavior. Consistent with this prediction, McCord (1991) found that when fathers with criminal records were in the home continuously, defined as no absence of greater than six months prior to the child's 17th birthday, the lifetime prevalence of arrests for male offspring was greater (48%) than in offspring of criminal fathers who were absent for at least

this minimal period (42%). There was an opposite pattern for offspring of non-criminal fathers, in which absent fathers had a higher rate of criminality in sons (33%) than did fathers who were present in the home continuously (27%).

These data reported by McCord (1991) are consistent with predictions made by a modeling theory of transmission. Specifically, greater exposure to an antisocial model should increase the risk for developing antisocial behavior. However, the lenient definition of "absence" in the McCord (1991) study may have underestimated the effects of modeling. Fathers may have been in the home for the majority of a child's life, during which time substantial observational learning could have occurred, but the father was labelled "absent" if he was not in home for any six month time period. Therefore, in the present study we attempt to further test predictions made by the modeling theory to explain the intergenerational cycle of antisocial behavior, using several more stringent definitions of paternal contact. In a sample of elementary school-aged children, we test whether or not the degree of contact with an antisocial father moderates the association between parent and child antisocial behavior.

While we could not rule out all alternative hypotheses, we collected data necessary for ruling out two alternative moderators to the predicted father-child link to antisocial behavior. Since antisocial fathers are more likely to marry women who also show antisocial behavior (Frick, Kuper, Silverthorn, & Cotter, 1995), it is likely that having an antisocial father also increased the risk for having an antisocial mother. Therefore, analyses were run controlling for maternal antisocial behavior to see if the association between father and child antisocial behavior was at least partially independent of maternal behavior. Also, since antisocial fathers were often not in the home and if they were, they often had difficulty maintaining steady employment (see also Widom, 1977), it is possible that the economic stress placed on family by having an antisocial father accounted for the increased risk for antisocial behavior in the offspring. As a result, analyses controlling for family socioeconomic status were also conducted.

METHOD

Subjects

Subjects were 92 children between the ages of 6 and 13 (mean age 8 years, 7 months) drawn from 104 consecutive referrals to an outpatient diagnostic and referral service of a university-based psychological clinic. Referrals were for emotional, behavioral, and learning problems and came

from physicians, teachers, parents, community mental health agencies, and the juvenile court system. Children were excluded if at least one biological parent was unable to accompany the child for the evaluation and provide information on a history of paternal antisocial behavior and the degree of paternal contact with the child ($n = 8$). Also, mentally retarded children ($n = 4$) were excluded from analyses. Demographic characteristics of the sample are summarized in Table 1. As evident from this table, the sample was primarily male (82%), Non-Hispanic Caucasian (78%), and generally skewed toward the lower to lower-middle socioeconomic statuses.

Measures

Child Conduct Problems

The presence of child conduct problems was determined by a diagnosis of Conduct Disorder (CD) or Oppositional Defiant Disorder (ODD) according to strict DSM-III-R criteria (American Psychological Association, 1987). Symptoms of these disorders were assessed by the NIMH Diagnostic Interview Schedule for Children (DISC 2.3; Shaffer, Fisher, Piacentini, Schwab-Stone, & Wicks, 1992) administered to one custodial parent of the child (DISC-P) and the child's teacher (DISC-T). A child version of the

Table 1. Demographic Characteristics of the Sample

Variable	Descriptive Statistic
Gender	82% Male; 18% Female
Race	78% White; 22% African American
Age	M = 8.7 years; SD = 2.0 years Range: 6 to 13 years
Grade	8.7% Kindergarten 15.2% First Grade 25.0% Second Grade 16.3% Third Grade 12.0% Fourth Grade 8.7% Fifth Grade 10.9% Sixth Grade 3.3% Seventh Grade
Wechsler Full Scale IQ (Wechsler, 1974)	M = 94; SD = 13 Range: 71 to 141
Duncan's Socioeconomic Index (Mueller & Parcel, 1981)	M = 36.7; SD = 26.1 Range: 0 to 96

DISC 2.2 (DISC-C) was only administered to children over the age of eight, since the interview has proven to be unreliable for younger children (Edelbrock, Costello, Dulcan, Kalas, & Conover, 1985). A symptom was considered present if reported as present by any informant, as recommended by Piacentini, Cohen, and Cohen (1992), and a diagnosis was considered present if a child met criteria according to this multi-informant report.

DISC interviews were administered by a clinical psychologist or advanced graduate students in clinical psychology who completed a course on the psychological assessment of children and were trained in standardized administration procedures. Forty-one percent of the DISC-P interviews and forty-five percent of the DISC-C interviews were observed through one-way mirrors. Kappa was calculated to determine interviewer-observer agreement for the diagnosis, if the diagnosis was coded at least twice by either interviewer or observer. Kappas for the two conduct problem diagnoses were 1.0 for both CD and ODD for parental report, thus indicating perfect interdiagnostician agreement for the conduct problem diagnoses. Kappa was also 1.0 for a diagnosis of ODD according to child report but there were too few instances of CD reported by the child when an observer was present to calculate kappa.

Due to logistical constraints, it was not possible to observe DISC-T administrations. The DISC-P and the DISC-T used the same questions to assess ODD and CD symptoms and the two interviews were conducted by the same interviewer for each child. Therefore, it is likely that the high level of reliability found for parents should apply to teachers. To support this claim, teachers were asked to complete the Comprehensive Behavior Rating Scale for Children (CBRSC: Neeper, Lahey, & Frick, 1990). Children who met criteria for ODD or CD according to teacher report on the DISC-T ($n = 16$) were compared to all children who did not meet criteria on the DISC-T ($n = 81$) on the Oppositional-Conduct Disorders scale of the CBRSC. The two groups differed significantly, $t(95) = 6.69, p < .0001$, indicating that teachers were consistent in their report across these methods of assessment.

Paternal Antisocial Behavior

The presence of paternal antisocial behavior was determined by a diagnosis of Antisocial Personality Disorder (APD). Diagnoses were made according to DSM-III-R criteria (American Psychological Association, 1987) based on the Diagnostic Interview Schedule (DIS) Version III-A (Robins & Helzer, 1985). Interviewers were graduate students who com-

pleted a class in the assessment of adult psychopathology and who were trained in standardized administration procedures. Interviewers were blind to the child's diagnostic status and reason for referral. To assess reliability in the present study, 32% of the interviews were observed via one-way mirrors. Observers and interviewers independently determined the presence of diagnoses. Kappa for interviewer-observer agreement on a paternal diagnosis of APD was 1.0.

In 25% of the cases, the child's biological father accompanied the child to the evaluation and was interviewed directly with DIS-III-A. Therefore, similar to past research (e.g. Biederman et al., 1987; Frick et al., 1992), assessment of paternal APD was conducted through a family history method with mothers reporting on APD symptoms in the fathers in the majority of cases. The use of this methodology was felt to be justifiable for two reasons. First, due to the nature of antisocial disorders in which there tends to be a bias to minimize symptoms, report of others play an important role in the assessment of APD (Hare, 1985). Second, a significant proportion (46%) of the biological fathers who could not be interviewed directly agreed to complete by mail the 54-item Socialization Scale (Gough, 1969), which is often used as a self-report indicator of APD (Hare, 1985). Within this subsample, fathers who met criteria for APD according to maternal report ($n = 5$) scored significantly lower on the Socialization Scale than fathers who did not meet criteria for APD ($n = 24$), $t(27) = 2.63$, $p < .01$. Therefore, there was empirical support for the validity of this family history method for assessing APD. In fact, this is likely a low estimate of the validity given the fact that fathers with APD were disproportionately less likely to be available and willing to complete the Socialization Scale, leading to a restricted range of scores.

Paternal Contact

Information on the degree of paternal contact with the child was collected via a semi-structured interview conducted with the biological parent(s) who accompanied the child to the evaluation. The sample was divided based on two independent criteria in order to capture different aspects of the degree of contact between father and child. First, the sample was divided into those children who had never had contact with their biological father since their first year of life (No Contact; $n = 19$) and those children who had face-to-face contact with their fathers at some point after the first year of life (Any Contact; $n = 73$). Second, children were divided based on the frequency of contact between father and child. Frequent contact was operationalized as face-to-face contact that occurred at least once

a month in the past year ($n = 60$). Infrequent Contact was operationalized as children with fathers a) who were out of the home for three or more years prior to the evaluation (including those who had never been in the home) and b) who had face-to-face contact with their child less than once per month ($n = 29$). This second definition led to the elimination of three children who did not fit into either the Frequent or Infrequent Contact groups (i.e., biological father had left the home within the past three years but had not maintained face-to-face contact in the past year).

Procedure

Each child was accompanied to the evaluation by at least one biological parent. The child and biological parent were asked to provide assent/consent for the information collected during the evaluation to be used in research. However, they were explicitly informed that participation in the research was voluntary and that their decision to participate would not affect the clinical services they received. Each measure was administered as part of a comprehensive psychological evaluation and was administered in a standard sequence. The evaluation began with administration of the semi-structured interview to the accompanying parent which obtained demographic information and information on paternal contact. This interview was followed by administration of the DISC-P. While the parent was being interviewed, the child was administered the Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler, 1974) to screen for intellectual impairment. Following a break for lunch, the parent was administered the DIS-III-A and the child was administered the DISC-C, if the child was older than eight years of age. Within one week following the evaluation, the child's teacher was administered the DISC-T by telephone.

RESULTS

In the entire sample, 38% ($n = 35$) of the children received a conduct problem diagnosis. Twenty-three percent ($n = 21$) of the children were diagnosed as having ODD and 15% (14) were diagnosed as having CD. Using chi-square analysis, a paternal APD diagnosis was found to be significantly associated, $\chi^2(1, N = 92) = 11.07, p < .001$, with children's conduct problem diagnoses. There were 20 (22%) biological fathers who received an APD diagnosis. Fourteen (70%) of the fathers receiving an APD diagnosis had a child with a conduct problem diagnosis.

To test whether the relationship between paternal APD and child conduct problems was moderated by degree of paternal contact, 2×2 logit

model analyses were conducted with paternal APD (present vs. absent) and the degree of contact (any vs. none/frequent vs. infrequent) as the two factors and child conduct problem diagnosis as the dichotomous response variable. Results of these analyses are presented in Table 2. Whereas the main effects for paternal APD were significant, the main effects for the contact variables were not significant, nor were the interaction effects significant.

Analyses were also run to rule out several alternative explanations for the association between paternal APD and child conduct problems. Logistic regression analyses were run predicting child diagnosis with paternal APD and paternal contact, but controlling for socioeconomic status and maternal antisocial behavior. Duncan's Socioeconomic Index (Mueller & Parcel, 1981) was used to measure the family's socioeconomic status. The composite of the K-corrected T-scores from the F, Psychopathic Deviate, and Mania scales of the Minnesota Multiphasic Personality Inventory-2nd Edition (Hathaway & McKinley, 1989) was used as the index of maternal antisocial behavior (Frick, Lahey, Hartdagen, & Hynd, 1989). In all analyses, paternal APD continued to significantly predict child diagnosis after controlling for socioeconomic status and maternal antisocial personality.

A subset of the sample ($n = 12$) consisting of a) children with a conduct problem diagnosis, b) who had a father with an APD diagnosis and c) who had a father leave the home at some point during the child's life was selected for more in depth analysis. This subset was studied to determine the relationship between presence of an antisocial model and the onset of first conduct problem symptom, retrospectively reported by the child's parent on the DISC-P. This information is important due to the fact that many of the children with conduct problems had displayed behavioral

Table 2. Pattern of Associations Between Paternal APD, Degree of Paternal Contact, and Child Conduct Problem Diagnosis

Degree of Contact	APD Diagnosis	No APD Diagnosis
Any Contact	71% (n = 17)	29% (n = 56)
No Contact	67% (n = 3)	31% (n = 16)
Significant Effect:	APD- X^2 (1, N = 92) = 4.96, $p < .05$	
Frequent Contact	50% (n = 10)	30% (n = 50)
Infrequent Contact	86% (n = 7)	27% (n = 22)
Significant Effect:	APD- X^2 (1, N = 92) = 6.93, $p < .01$	

Note: The percentages in this table represent the proportion of children in each cell who had a conduct problem diagnosis. Effects are the significant effects of 2×2 logit model analyses with paternal APD and degree of contact as the two predictors and conduct problem diagnosis in the child as the dichotomous response variable.

difficulties for significant periods of time prior to being referred for services. Within this subsample, 58% of the children were observed to have had their conduct problems onset over one year after the biological father's departure and 42% evidenced onset of conduct problem symptoms either when the father was still in the home or within one year after his departure.

DISCUSSION

This study was conducted to further examine the processes involved in the intergenerational link to antisocial behaviors. The findings support the current literature showing a significant relationship between paternal APD and child conduct problems (Biederman et al., 1987; Faraone et al., 1991; Frick et al., 1992; Hamdan-Allen et al., 1989; Lahey et al., 1988; Stewart et al., 1980). However, the degree of exposure to an antisocial model did not moderate the relationship between paternal APD and child conduct problems, as would be predicted by modeling theory. That is, offspring of antisocial fathers were as likely to have significant conduct problems, whether or not the father had ever been in the home and whether or not the father had maintained frequent contact with their offspring.

These results are in contrast to the results of McCord (1991) who reported that criminal fathers who were present in the home were more likely to have antisocial sons than were criminal fathers who had been out of the home for any six month period prior to the child's 17th birthday. There are several possible explanations for these discrepant results. First, the risk for arrest in sons of criminal fathers who were continuously present (48%) was only slightly higher than the risk for arrest in offspring of criminal fathers not continuously present in the home (42%). The risk rates were not subjected to statistical analysis. Therefore, it is quite possible that this difference was a chance variation.

Second, McCord (1991) studied lifetime histories of arrests in fathers and sons as the measure of antisocial behavior, which intermixes people with childhood-onset antisocial behavior with those showing an adolescent or adult-onset of criminal behavior. Research has shown very different developmental trajectories and different correlates to these two patterns of antisocial behavior (Lahey, Loeber, Quay, Frick, & Grimm, 1992). Because of the young age of our sample, our study focused primarily on early-onset antisocial behavior and may not generalize to other patterns of antisocial behavior.

Like many family history studies, we did not have access to all fathers to assess antisocial behavior directly through self-report (Biederman et al., 1987; Frick et al., 1992). Unfortunately, limiting analyses only to those who

could be assessed directly would eliminate the more highly antisocial fathers who are more typically out of the home. Therefore, results must be interpreted in light of the fact that 75% of the fathers were diagnosed with APD by the report of the child's mother. However, we were able to provide data supporting the validity of maternal report by obtaining a self-report questionnaire measuring antisocial tendencies which was completed by a significant proportion of fathers who did not report on themselves for a diagnosis.

In addition to this methodological consideration, interpretation of these results must be placed in the appropriate theoretical context. Our analyses did not reveal the predicted interaction between the presence of an antisocial model and the degree of contact for predicting the risk for antisocial behavior in offspring that would be predicted by observational learning theory. However, the failure to find such an interaction does not rule out the possibility that more subtle forms of observational learning were taking place, such as through a child hearing stories about his father even if there was no direct contact between father and son.

It is also possible that having an antisocial father increases the likelihood of a child having other antisocial models, such as an antisocial mother or antisocial aunts and uncles (e.g., Frick et al., 1995). We repeated our analyses controlling for maternal antisocial behavior which allowed us to rule out the possibility that maternal antisocial behavior was solely accounting for the antisocial link between father and child. However, we did not measure antisocial behavior in other first degree relatives that could have served as antisocial models. Therefore, our data cannot rule out many alternative routes through which observational learning could influence the intergenerational link to antisocial behavior. However, we also feel strongly that one should be wary of creating a theory that can never be disproved. It should be recognized that to date, there is little empirical support, the present study included, which would support observational learning mechanisms to account for the intergenerational link to antisocial behavior.

On the other hand, we have a long way to go to develop better alternative theories. In the introduction, we mentioned that genetic explanations (Jarey & Stewart, 1985) and models that suggest that the poor socialization practices used by antisocial parents lead to conduct problems in children (Patterson & Capaldi, 1990) are two examples of plausible explanations that have been developed in the past. It is also quite likely that models that posit any single cause will have problems accounting for significant variance in an outcome as complex as childhood antisocial behavior (Frick & Jackson, 1993). Therefore, the one clear outcome of this study is to emphasize the continuing need to test well-formulated theory-driven models

to explain the intergenerational link to antisocial behavior, since an adequate explanation has eluded the field thus far.

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