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The contribution of parents and siblings to antisocial and depressive behavior in adolescents: A double jeopardy coercion model

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Abstract
A dual coercion model of family processes associated with the development of antisocial and depressive behavior during adolescence was assessed, using an at-risk sample of families and children. Consistent with the model, involvement in family coercion during childhood and adolescence increased both boys' and girls' risk for antisocial behavior in adolescence and girls' risk for depressive behavior. Coercive family processes served as a link between older and younger siblings' antisocial behavior. Childhood exposure to maternal depression predicted boys' and girls' depressive behavior 10 years later, but this association was not mediated by coercion. The data suggest that family risk factors and processes for antisocial development are similar for boys and girls but pathways to depression may be gender specific.

Antisocial behavior and depression are common and often co-occurring problems during adolescence (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993) and adulthood (Merikangas et al., 1998). Such co-occurring problems consume a large proportion of clinical services, are treatment resistant, are associated with increased severity and persistence of each disorder, and result in significant collateral impairments (Biederman, Farone, Mick, & Lelon, 1995). Research on the origins, timing, and sequencing of co-occurring depression and antisocial behavior prior to adolescence is rare, but it provides a promising venue for a better understanding of the developmental trajectories and risk factors associated with each disorder as well as their simultaneous expression (Snyder & Stoolmiller, 2001). Whether co-occurring problems are causally related to one another, reflect increasing severity of the index problem, or result from overlapping versus unique risk factors and developmental processes has not been clearly established in the current research literature (Caron & Rutter, 1991; Garber & Hollon, 1991).

Depression and antisocial behavior share similar family environmental antecedents, such as frequent exposure to aversive social exchange (Davis, Sheeber, & Hops, 2002; Patterson, Reid, & Dishion, 1992) and maternal depression (Goodman & Gotlib, 1999). However, antisocial behavior and depression differ in developmental onset, temporal course (Loeber & Keenan, 1994), and gender-related prevalence (Hartung & Widiger, 1998). Re-

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search on each problem has generally proceeded independently, precluding a better understanding of comorbidity (Zahn–Waxler, 1993). Using a longitudinal design and multiagent and multimethod assessment, this research examined how social environments provided by parents and siblings contribute to the occurrence of and gender differences in antisocial and depressive behavior in adolescence.

Mechanisms in the Family Transmission of Psychopathology

Research indicates considerable aggregation among family members for both depressive and antisocial behavior, but pathology displayed by family members is not necessarily of the same type. Maternal depression is associated with increased risk for both child depression and antisocial behavior (Davis et al., in press; Goodman & Gotlib, 1999). The antisocial behavior of one sibling is associated with the display of similar behavior by other siblings (e.g., Pike, McGuire, Hetherington, Reiss, & Plomin, 1996) and with a range of other problems, including school failure, early sexual activity, and drug use (Bank, Snyder, & Burraston, 2001). Sibling relationships impact adolescent internalizing and externalizing problems, even in the context of parent–child relationships (Conger, Conger, & Scaramella, 1997). Antisocial and depressive behavior tend to occur at a family-systemic level, but the processes mediating family aggregation are not clearly understood. A model specifying these processes must parsimoniously and simultaneously account for co-occurrence of and sex differences in depression and antisocial behavior and for the modest specificity with which antisocial and depressive behavior are observed in members of the same family.

Shared genes account for similarities in family members' display of antisocial behavior and depression (e.g., Reiss, Niederhiser, Hetherington, & Plomin, in press; Rowe, 1994), but the social environment as main effect or in interaction with genetic influences is also likely to play a substantial role in family transmission of antisocial behavior (Rowe, Linver, & Rodgers, 1996) and depression (Strober, 1995). Coercion theory provides a promising social process model that specifies the mechanisms by which familial risk for antisocial behavior and depression are transmitted (Davis et al., 2002; Hops, 1996; Patterson, 1982). According to coercion theory, aggressive and depressive behavior are two distinct response classes that children may display in response to highly aversive home environments. Helplessness, self-derogation, withdrawal, and sadness may deflect aversive social events as effectively as opposition, anger, and attack. The display of aggression and depression relative to each other (and to more skilled behavior) depends, in part, on past direct and vicarious learning about their utility in deflecting aversive social events or in obtaining attention (Hops, Biglan, Sherman, Arthur, Friedman, & Osteen, 1987; Snyder & Patterson, 1995). Frequent and persisting aggression and depression reflect topographically distinct but not necessarily incompatible responses that are potentially functional in dealing with environments characterized by frequent aversive and infrequent positive events.

Three conditions need to be satisfied to support the hypothesis that children's chronic exposure to aversive social environments mediates the link of maternal depression and sibling antisocial behavior to children's increased risk for later depression and antisocial behavior. First, maternal depression and sibling antisocial behavior should be associated with increased risk for child antisocial and depressive behavior. Second, maternal depression and sibling antisocial behavior should be associated with children's exposure to frequent aversive family interchange. Third, high levels of aversive family interchange should be associated with increased risk for child antisocial behavior and depressive behavior. The previously described data about family aggregation for depression and antisocial behavior provide support for the first condition. We now turn to evidence relevant to the second and third conditions.

Research has repeatedly demonstrated that depressed relative to nondepressed mothers are more irritable, punitive, and inconsistent, thus increasing children's exposure to and involvement in aversive family exchanges

(Goodman & Gotlib, 1999). Sibling interaction often entails even higher levels of aversive exchange than is observed in parent–child and peer interaction (Shortt, Capaldi, Dishion, Bank, & Owen, 2000). Sibling aggression occurs in very serious forms, such as punching, hitting with objects, and weapon use (Roscoe, Goodwin, & Kennedy, 1987). Aversive sibling exchange is especially likely to be a powerful source of coercion if one of the siblings is an antisocial male (Bank, Snyder & Burraston, 2001). Conger et al. (1997) and Pike et al. (1996) have reported that aversive sibling interaction is associated with increased risk for antisocial and depressive behavior in adolescence, over and above aversive parent–child interchange. Thus, there is empirical support for the second condition.

Research consistently shows that child and adolescent aggression and depression are concurrently and prospectively associated with family environments in which children are exposed to frequent coercive social exchange. This association has been observed in both clinical (for depressive behavior: Fendrich, Warner, & Weissman, 1990; for antisocial behavior: Patterson, 1982) and community (for depressive behavior: Cole & McPherson, 1993; Hops, Lewinsohn, Andrews, & Roberts, 1990; for antisocial behavior: Forgatch, 1991; Patterson et al., 1992) samples. Observational studies indicate that both aggression (Snyder, Edwards, McGraw, Kilgore, & Holton, 1994; Snyder & Patterson, 1995) and depression (Dadds, Sanders, Morrison, & Rebgetz, 1992; Hops et al., 1987) are functional in deflecting the aversive behavior and obtaining the attention of other family members. Empirical support exists for the third condition.

In summary, all three conditions requisite to the validation of coercion as a core social process in the intrafamilial transmission of antisocial and depressive behaviors are supported empirically, but this picture is cobbled together from research looking at each of the conditions and outcomes one at a time. Clearer support for the theory would derive from research that examines all three conditions and both outcomes simultaneously.

Coercion is likely only one of several, converging family processes that increase a child's risk for persistent antisocial behavior and depression. One additional family process entails children's modeling of maternal depressive behavior. Depressed mothers frequently display dysphoric affect, engage in self-derogation, complain, seek relief from normative roles, and solicit care taking. Children may incorporate these depressive behaviors into their own repertoire, facilitating their display of depressive behavior when confronted with aversive social events (Garber & Robinson, 1997; Radke–Yarrow, Belmont, Nottelman, & Bottomly, 1990). Girls, relative to boys, may be more likely to imitate maternal depressive behavior (Hops et al., 1990). Thus, exposure to maternal depression as an independent direct effect or as mediated by coercive family process may enhance risk for child depression, especially in girls.

Gender Differences in Antisocial Behavior and Depression

Exposure to coercive family environments does not explain gender differences in depression and antisocial behavior (Keenan & Shaw, 1997; Lewinsohn et al., 1993). The origins of such differences in antisocial and depressive behavior may entail two steps in development. In the first step, initial, small gender differences in responses to social challenge are shaped during children's normative socialization experiences in family, school, and peer settings. Parents, teachers, and particularly peers provide social responses that, on the average, promote bossy, self-enhancing, noncompliant, competitive, and demanding responses to aversive stimuli by boys, and compliant, tractable, deferential, verbal–persuasive responses by girls (Fagot, Hagan, Leinbach, & Kronsberg, 1985; Kavanaugh & Hops, 1994; Maccoby, 1998).

In a second developmental step, normative differences in how boys and girls respond to challenge are amplified by chronic exposure to aversive family environments. Maternal depression and sibling antisocial behavior increase the frequency of aversive events experienced by a child. Based on previous socialization experiences, boys relative to girls may be more likely to respond to chronically aver-

sive family exchanges with aggression, and less likely to do so with depression. Girls are more likely than boys to become enmeshed in maternal depression and to imitate maternal depressed mood (Cummings & Davies, 1994), exacerbating girls' risk for depression. Thus, developmental pathways to antisocial and depressive behavior, both in single and co-occurring expression, may show some gender specificity. Boys' initial and primary responses to maternal depression, antisocial siblings, and an aversive family environment are more likely to be aggressive whereas that of females is more likely to be depressive. As exposure to an aversive family environment becomes chronic and negative sequalae accrue to the display of the aggressive or depressive behavior, the initially less dominant, alternate behavior may be increasingly displayed, resulting in co-occurrence of both behaviors. Male depression, for example, appears to result from peer rejection and school failure accompanying antisocial behavior rather than directly from family environmental effects (Capaldi, 1992; Capaldi & Stoolmiller, 1999).

Hypotheses

The following hypotheses were tested in relation to risk conditions during early childhood.

1. Children's exposure to depressed mothers and antisocial older brothers would be associated with increased risk for both depression and antisocial behavior in adolescence.
2. The frequency of aversive social exchange experienced by children would be higher in families with depressed mothers and antisocial older brothers.
3. The association of children's exposure to depressed mothers and antisocial older brothers with increased risk for antisocial behavior in adolescence would be mediated by aversive family exchange for both boys and girls.
4. The association of children's exposure to depressed mothers and antisocial older brothers with increased risk for depression in adolescence would be mediated by aversive family exchange for girls but not for boys. That is, child gender would moderate the relationship of family aversive exchange to depressive outcomes.

The following hypotheses were tested in relation to risk conditions during adolescence.

5. Children's exposure to antisocial older brothers would be associated with increased risk for both antisocial behavior and depression in adolescence.
6. Children would be involved in more aversive sibling interaction when exposed to antisocial older brothers.
7. The association of children's exposure to antisocial older brothers with increased risk for adolescent antisocial behavior would be mediated by aversive sibling interaction for both boys and girls.
8. The association of children's exposure to antisocial older brothers with increasing risk for adolescent depression would be mediated by aversive sibling interaction for girls but not for boys. That is, child gender would moderate the relationship of sibling aversive interaction to depressive outcomes.

Method

Participants

Tests of the proposed models were based on data from the Oregon Youth Study (OYS), which was designed to longitudinally ascertain the family origins and developmental course of antisocial behavior in a community sample of 206 at-risk boys (Capaldi & Patterson, 1987). The target children and families were selected from neighborhoods with the highest delinquency rates in a moderate-sized city in the northwest United States. The OYS began when the target boys were age 10 and has continued through age 20, the time span from which data were derived for the current research. The OYS was extended to include 75 younger brothers and 86 younger sisters of

the OYS boys, using data first collected when the OYS boys were ages 10–13 (Waves 1–3, at the initiation of the longitudinal study) until the OYS boys were age 20 (Data Collection Wave 11). The average age of the younger brothers at the initiation of the OYS study (Wave 1) was 5.3 years (*SD* = 2.6, range = 0–10), and the average age of the younger sisters was 5.8 years (*SD* = 2.5, range = 0–10). At the onset of the OYS, 42% the families were headed by two biological parents, 23% by a biological parent and stepparent (almost all stepfathers), and 35% by a single biological parent (almost 90% mothers). Families were predominantly lower income and working class. Ten percent of the target OYS boys self-identified minority status. Median family income was $14,848 (in 1983–1984). One-fifth of the families had no employed parent and one-third received public assistance.

A subset of 34 younger brothers and 39 younger sisters of the OYS target boys was selected for the current research based on three criteria: the availability of observational data concerning parent–child and sibling interaction at the initiation of the OYS (Waves 1 and 3); a minimum younger sibling age of 2 years at the initiation of the OYS (Wave 1); and the availability of sibling interaction data during the younger siblings' adolescence (Wave 11). In families for which data for two younger siblings were available, the sibling closest in age to the OYS boy was selected for the current analyses. The average age of selected focal younger brothers was 6.2 years (*SD* = 2.0) and of focal younger sisters, 6.5 years (*SD* = 1.8) at the initiation of the OYS (Wave 1). The mean age of the younger siblings was 16.4 years (range = 12–19) when their developmental outcomes on antisocial and depressive behaviors were assessed at Wave 11, which occurred 10 years after the initiation of the OYS. The sample of families with younger siblings selected for the current research did not differ significantly from the total OYS sample or from OYS families with younger siblings of target OYS boys on income, number, and biological/step status of parents in the home, total number of children in the family, maternal depression, OYS boys' arrests, or biological relatedness of the younger sibling and OYS boy.

Measures

Six constructs were derived from the OYS/sibling data. Each construct was defined by two or more indicators derived from different informants (parent, OYS target boy, younger sibling, observers) or by different methods (interviews, checklists, observer ratings, behavior rates from observation) such that adjacent constructs in the models to be tested did not share totally overlapping informants or methods. A single score was calculated for each construct by standardizing each indicator and then averaging across indicators.[1] The indicators for each construct are described in the following sections, briefly for constructs already specified in published research and more thoroughly for new constructs developed for this research.

Maternal depression. Maternal depression was measured during the 1st and 3rd years of the OYS, when focal younger siblings were an average of 6 and 8 years of age.[2] Three self-report indicators defined the maternal depression construct in each year: the CES-D Scale (Radloff, 1977), the Depression Affect Adjective Checklist (Lubin, 1963), and self-reported depression averaged across six daily telephone interviews. The three indicators all loaded reliably (>.50) on one factor, and the associated confirmatory factor analysis (CFA) indicated a good fit (NFI = .95). Additional

1. Although a structure equation modeling (SEM) approach would have better taken advantage of the multi-indicator constructs available in this data set to estimate (and minimize) measurement error, as well as the structural relations among the constructs, the number of subjects (74) was grossly insufficient relative to the number of parameters (~35 in the simplest model) that would have to be estimated using an SEM analytical approach. Typically, there should be a minimum of 5 subjects for every parameter to be estimated.
2. Constructs for paternal depression were not included in the models because of the large number of single-parent families and stepfather families, which were relatively unstable over time.

psychometric properties for the construct are given in Capaldi and Patterson (1987).

Older brother (*OYS boy*) *antisocial behavior.* Older brothers' (target boys in the OYS) antisocial behavior was measured during the 1st and 3rd years of the OYS when younger siblings were an average of 6 and 8 years old, and again in the 5th and 7th years of the OYS, when younger siblings were an average of 10 and 12 years old. Four or five indicators were used to assess older brothers' antisocial behavior at each developmental point: the delinquent behavior scale from the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983) and from the Teacher Report Form (Achenbach & Edelbrock, 1986), the OYS boy's self-report of antisocial activities during a structured interview, peer nominations (Wave 1 only), and daily phone interviews with the OYS boys. Each indicator loaded reliably on an antisocial construct (>.53) at each point, and the associated CFAs indicated a good fit (NFI > .94). Additional psychometric properties for this construct can be found in Capaldi and Patterson (1987).

Coercive family interaction. The construct for coercive family interaction was derived from measures obtained during the first and third years of the OYS study, when younger siblings were an average of 6 and 8 years of age. The construct consisted of five observational indicators and two reports by family members; it reflected aversive interaction between younger siblings and their older (OYS) brothers and between younger siblings and their parents.[3] Four indicators were derived from observation of family interaction in the home, coded using the Family Process Code (FPC; see Capaldi & Patterson, 1987; and Dishion et al., 1983 for details on observation procedures and reliability). The first two observational indicators were the rates per minute at which mothers and OYS boys directed aversive behavior toward younger siblings. The second two observational indicators entailed rates of conflict bouts between younger siblings and their mothers and between younger siblings and other children in the family. The onset of a conflict bout was defined as the reciprocation of aversive behavior, and its offset, as the absence of aversive behavior by parties to the conflict for a period of 18 s or more. Conflict bouts, in contrast to rate per minute of aversive behavior, reflect contagion and escalation in aggressive interchange in the family (Snyder et al., 1994). The fifth indicator was derived from ratings made by FPC coders immediately following each observation session concerning the degree of sibling conflict. The last two indicators were derived from ratings made by the parents and the OYS boy concerning the quality of sibling relationships within the family. Each indicator loaded reliably on the coercive family interaction construct (>.35), and a CFA indicated a good fit (NFI = .90).

Coercive sibling interaction. The sibling coercive interaction construct was defined by three indicators assessing aversive social exchange between the target OYS boys and their younger brothers or sisters, obtained when younger siblings were an average age of 16. The first indicator was the rate per minute of aversive behavior that older (OYS) boys directed at their younger sibling, using the Family and Peer Process system (Stubbs, Crosby, Forgatch, & Capaldi, 1998) to code interaction during a 30-min sibling interaction task (SIT). In the SIT, the OYS boy and his younger sibling were asked to engage in problem solving concerning issues identified by each sibling and to talk about current and previous family relationships. The second indicator was a single item, completed by an assessor who met with the siblings prior to the SIT, about the degree to which the OYS boy

3. One or more indicators of the family coercion construct were occasionally missing. When this was the case, the construct score was comprised of the mean of the available indicators. Some of the measures were derived in a manner that reflects interfamilial relationships toward younger (sometimes younger and older) siblings of the OYS boys more generally, thus representing only an approximation of the family environment of the specific younger siblings who were targets in this study. Additional information concerning the reliability of the observations plus the items comprising the observer impressions and family members' reports of sibling relationships can be obtained from Lew Bank, Oregon Social Learning Center.

was "unpleasant, cold, or mean" toward his younger sibling. The third indicator was derived from ratings on five Likert items (e.g., criticized, bullied) about the quality of the relationship between the siblings, which was completed by staff who coded the SIT interaction. Internal reliability of this rating scale was $\alpha = .82$. The three indicators loaded on the sibling coercive interaction construct (>.42) and a CFA indicated a good fit (NFI = .93).

Younger sibling antisocial behavior. Younger siblings' antisocial behavior was defined by five indicators. Each was assessed when the younger siblings were a mean age of 16. The first indicator was the delinquent behavior scale from the CBCL (Achenbach & Edlebrock, 1983). The second indicator was based on a parental report of the younger siblings' delinquent activity during the previous year, using the Elliott delinquency scale (Elliott, Ageton, Huizinga, Knowles, & Canter, 1983), including both major and minor offenses. The third indicator was younger siblings' self-reports of delinquent acts on the Elliott scale. The fourth indicator consisted of younger siblings' self-reported gang involvement (four items; e.g., participated in gang activities, spent time with gang members). Internal reliabilities for each of the four scales was >.70. The fifth indicator was defined by a series of daily telephone interviews in which younger siblings reported their own performance of nine covert and overt antisocial behaviors (e.g., stealing, aggression, illegal activity). Internal reliability of this scale was .68. Each indicators loaded >.60 on the antisocial construct for younger sisters and younger brothers, and the CFA indicated a good fit (NFI > .97).

Younger sibling depression. The construct for younger sibling depression at a mean age of 16 years was defined by three indicators. The first indicator was the younger siblings' self-reports of depressive symptoms on the CES-D if age 16 or over (Radloff, 1977) or on the Child Depression Inventory if below age 16 (CDI; Kovacs, 1983). The second indicator was derived from four Likert scale items completed by an assessor after a structured interview with the younger sibling (e.g., sad, unhappy with life). Internal reliability for the interviewer rating scale was $\alpha = .75$. The last indicator was derived from 12 items concerning depressive affect and behavior from a parent CBCL. Internal consistency for the CBCL scale was $\alpha = .81$. The common factor loadings for the three indicators for depressive behavior were >.35 for both younger brothers and younger sisters, and the CFAs indicated a good fit (NFI > .91).

Procedures

Informed consent was obtained from participating parents and children at the onset and in the various extensions of the OYS/siblings study. Data relevant to the current research were collected at Waves 1 and 3 of the OYS (mean age of younger siblings = 6 and 8 years, mean age of older brothers = 10 and 12 years), Waves 5 and 7 of the OYS (mean age of younger siblings = 10 and 12 years, mean age of older brothers = 14 and 16 years), and at OYS Wave 11 (mean age of younger siblings = 16, mean age of older brothers = 20). Interviewers and observers were thoroughly trained prior to collecting data, and ongoing recalibration meetings were held to promote measurement fidelity and reliability.

Results

Descriptive Data

The sample represents a full range of maternal depression, older brother and younger sibling antisocial behavior, younger sibling depression, and coercive family and sibling processes. Descriptive statistics for selected measures for which normative data are available are shown in Table 1. The cumulative rate of ever being arrested and of multiple arrests for the older, OYS brothers was higher than that of community samples of male adolescents in the years in which the data were collected (H. N. Snyder, 1988). Mean maternal self-reported depression on the CES-D, when the mean ages of the younger siblings were 6 and 8 years, was 12.9. This is considerably higher than normative samples, with 15% of mothers

Table 1. *Mean gender-specific levels of older brother antisocial behavior, maternal depression, family and sibling coercion, and younger sibling depressive and antisocial behavior at adolescence*

	Younger Siblings	
Variate	Male	Female
Older brother ever arrested by age 17	45%	44%
Older brothers arrested 2+ times by age 17	30%	18%
Mean maternal CES-D depression score (at *M* sib. age 6–8)	12.9 (7.2)	13.0 (8.1)
Mothers CES-D score >20 (moderate)	12.1%	17.9%
Mean RPM sibling coercive (at *M* sib. age 6–8)	.30 (.26)	.28 (.27)
Mean RPM mom–child conflict bouts (at *M* sib. age 6–8)	.12 (.07)	.10 (.05)
Mean RPM sibling coercive (at *M* sib. age 16)	.314 (.554)	.269 (.302)
Mean CES-D of younger siblings aged >15 (at *M* sib. age 16)	16.9 (9.9)	19.7 (10.7)
Younger sibling CES-D score >20 (moderate)	11%	17%
Mean CDI of younger siblings aged <16	7.9 (6.0)	9.2 (7.1)
Younger siblings CDI score >20 (moderate)	10%	14%
Younger sibling ever arrested (self-report)	30%	13%*
Younger sibling index crime (parent Elliott)	27%	10%*

*$p < .05$.

reporting at least moderate levels of depressive symptoms. As preadolescent children (Waves 1 and 3, mean ages 6 and 8) in the home setting, and during extended conversations as adolescents (Wave 11, mean age 16), target younger siblings and their OYS older brothers were observed to engage in frequent coercive social exchange, involving about one aversive behavior every 3 to 4 minutes. Mothers were also frequently involved in conflict sequences with their children, about once every 10 min (Waves 1 and 3).

The targeted younger brothers and sisters evidenced considerable antisocial and depressive behavior during adolescence. Thirty percent of boys and 13% of girls self-reported one or more arrests, a percentage about twice as high as the national average (Snyder & Sikmund, 1999). Parental reports of the commission of index crimes by the focal younger siblings were approximately 1.5 times higher than child self-reports of comparable crimes by a community sample of adolescents (Elliott, Huizinga, & Menard, 1988). Younger siblings' mean self-reported depression scores on the CES-D or on the CDI were 18.7 and 8.8, respectively. Over 10 percent of younger siblings under age 16 at Wave 11 reported at least moderate depression (CDI > 20). Thirteen percent of the younger siblings who were 16 years or older at Wave 11 reported at least moderate depression (CES-D > 20). These levels of depressive symptoms are higher than those found in epidemiological samples. The higher occurrence of more extreme forms of antisocial and depressive behaviors in family members and of coercive family processes reflects the high-risk community sampling strategy used in the OYS. Two gender differences were found. Younger male relative to younger female siblings more often self-reported arrests, $\chi^2 = 4.59$, $p < .05$, and parents were more likely to report that their younger sons had been involved in an index crime than their younger daughters, χ^2 (1) = 4.36, $p < .05$. Gender differences on the other variables presented in Table 1 were not statistically reliable. Two gender differences were found in construct scores (not shown in tabular form). At Waves 5 and 7, the older brothers of male siblings were more antisocial than those of the younger sisters ($t = 2.67$, $p < .05$). At Wave 11, younger brothers evidenced higher levels of antisocial behavior in adolescence than did younger sisters ($t = 2.03$, $p < .05$).

Table 2 shows bivariate correlations among the various construct scores for family and sibling environments and sibling develop-

Table 2. *Correlations among maternal depression, older brother antisocial behavior, family and sibling social processes, and younger siblings' depressive and antisocial behavior in adolescence*

	1	2	3	4	5	6	7
1. Maternal depression (w1–3)							
2. Older brother antisocial (w1–3)	.37**						
3. Older brother antisocial (w5–7)	.13	.58**					
4. Early family coercion (w1–3)	.14	.51**	.56**				
5. Later sibling coercion (w11)	.12	.30**	.26*	.29*			
6. Sibling gender	−.03	−.12	−.21†	−.07	−.12		
7. Younger sibling depressive behavior (w11)	.30**	.11	.08	.20†	.21†	−.04	
8. Younger sibling antisocial behavior (w11)	.11	.27*	.59**	.39**	.45**	−.24*	.43**

Note: w, the wave or timing of collection of the construct; w1–3, mean younger sibling ages 6 and 8; w5–7, mean younger sibling ages 10 and 12; w11, mean younger sibling age 16.
†$p < .10$. *$p < .05$. **$p < .01$.

mental outcomes. The significant correlation of younger sibling gender with adolescent antisocial behavior reflects more antisocial behavior for males. Younger siblings' antisocial behavior during adolescence was reliably associated with a variety of earlier (Waves 1 and 3) and later (Waves 5–7 and Wave 11) family constructs. Younger siblings' depression in adolescence was marginally associated with family constructs. Maternal depression was not correlated with early family coercion (Waves 1–3) or later sibling coercion (Wave 11), obviating the role of coercion in mediating relationships between maternal depression and adolescent outcomes. These relationships, in part, may reflect measurement and sampling strategies in OYS that focused primarily on family variables associated with increased risk for antisocial rather than depressive behavior. The antisocial and depressive behaviors of younger siblings in adolescence (Wave 11) were reliably intercorrelated (.43), and this association was observed for both younger male ($r = .37$, $p < .05$) and female siblings ($r = .51$, $p < .001$). Co-occurrence was also calculated at a categorical level using clinical cutoffs of 20 for the CES-D and CDI and a T score > 67 (95th percentile) on the delinquency scale of the CBCL. Two girls (5%) and 3 boys (9%) displayed clinical-level co-occurring depression and conduct problems. Four girls (10%) and one boy (2%) displayed clinical-level depression only, and 2 girls (5%) and 5 boys (15%) evidenced clinical-level antisocial behavior only.

Tests of theoretical models

Tests were made of the dual coercion model using linear regression. These tests were made separately for antisocial and depressive adolescent outcomes and for early family (Waves 1 and 3) and later sibling (Waves 5 and 7 and Wave 11) influences. In each case, the following model testing sequence was used, congruent with the guidelines provided by Holmbeck (1997). First, the direct association of maternal depression (Waves 1 and 3), sibling gender, and older brother antisocial behavior (Waves 1 and 3 in the early socialization model and Waves 5 and 7 in the late socialization model) with younger sibling antisocial and depressive behavior (Wave 11) was tested. Maternal depression was retained in the models to assess its direct effects on adolescent outcomes, even though its mediated effects via family coercion was not supported. Second, coercive family process (Waves 1 and 3) and coercive sibling process (Wave 11) were added to the direct association models to test whether these processes served as mediators of any observed associations of maternal

depression and older brother antisocial behavior with younger sibling outcomes. Third, interaction terms of younger sibling gender by family coercive process and younger sibling gender by sibling coercion were stepped into the corresponding models (just described) to assess hypothesized gender-moderated developmental effects (particularly for depression) of family and sibling coercive processes. When a significant moderator term was observed, linear plots of the relationship between exposure to coercive processes and adolescent outcomes were specified by younger sibling gender. Prior to tests of the models, construct scores were transformed using log or square root functions to better approximate normal distribution.

Early socialization: Association of maternal depression, older brother antisocial behavior, and family coercion with younger sibling antisocial and depressive behavior

The contribution of early family socialization to younger sibling's antisocial behavior in adolescence is shown as Step 1 in the top of Table 3. Older brothers' antisocial behavior, when younger siblings were an average of 6–8 years old, was reliably associated with younger siblings' antisocial behavior at mean age 16. Younger siblings' early exposure to maternal depression was not predictive of antisocial behavior in adolescence. The marginal association of sibling gender with antisocial behavior is consistent with the descriptive data, indicating that boys relative to girls displayed higher levels of antisocial behavior in adolescence. The mediational role of younger siblings' involvement in family coercion with mothers and older brothers at mean ages 6 to 8 was tested by entering the family coercion term in a block (labeled Step 2 in the upper portion of Table 3) along with maternal depression, older brother antisocial behavior, and younger sibling gender. Family coercion was a reliable prospective predictor of younger siblings' antisocial behavior in adolescence, and it reduced the direct effects of older brothers' antisocial behavior to nonsignificance, supporting its status as a mediator. The mediational model was reliable, and accounted for 21% of the variance in younger siblings' antisocial behavior 8 to 10 years later. The moderator term, younger sibling gender by family coercion, which was stepped into the model to examine gender effects of coercion on younger siblings' antisocial outcomes at age 16, was not significant (see Step 3, top of Table 3). Younger brothers' and sisters' antisocial behavior in adolescence was similarly influenced by early exposure to coercive family environments.

Tests of early socialization models for younger siblings' adolescent depression are shown in the bottom portion of Table 3. Exposure to maternal depression at mean child ages 6 and 8 was reliably associated with adolescent depression, but neither younger sibling gender nor older brothers' antisocial behavior were reliable predictors (see Step 1). Family coercion did not mediate the relation of maternal depression to child depression. Early family coercion was reliably associated with adolescent depression ($p = .039$) when added to the "direct effects" model (see Step 2, bottom half of Table 3) and did not reduce the prediction value of maternal depression. The addition of family coercion improved the overall fit of the model, from $p = .103$ to $p = .039$, and accounted for an additional 5% of the variance in younger siblings' adolescent depression. The younger sibling gender by family coercion moderator term was significant (see Step 3, bottom of Table 3) when added to the mediator model, reliably incremented overall prediction, and added 6% to the total R^2. This suggests that the impact of family coercion on younger siblings' adolescent depression was conditional on younger sibling gender. As shown in Figure 1a, younger sisters' but not brothers' depressive symptoms in adolescence increased as they were exposed to increasing family coercion at mean ages 6 and 8. Given that maternal depression was not reliably associated with early family coercion and that older brother antisocial behavior was reliably associated with coercion but not with younger siblings' adolescent depressive behavior (see Table 2), this moderator suggests that older brothers impact younger sisters' depression only insofar as they are highly coercive toward their sisters,

Table 3. *Tests of early coercion models of adolescent antisocial and depressive behavior of boys and girls*

	β	*t*	*p*	ΔR^2
Antisocial Behavior				
Step 1: Direct effects model				
Maternal depression at child ages 6–8	.02	.14	.891	
Older brother antisocial at child ages 6–8	.23	2.03	.041	
Gender of target child	−.22	−1.90	.062	
Entered as a block; F (3, 69) = 2.75, p = .050				.12
Step 2: Family coercion as a mediator				
Maternal depression at child ages 6–8	.04	.32	.750	
Older brother antisocial at child ages 6–8	.04	.26	.800	
Gender of target child	−.20	−1.78	.079	
Family coercion at child ages 6–8	.35	2.69	.009	
Entered as a block; F (4, 68) = 7.21, p = .009				.21
Step 3: Child gender as a moderator of family coercion				
Gender of Target Child × Family Coercion	.11	.21	.833	
F (1, 67) change = .045, p = .83				(.00)
F (5, 67) total model = 2.27, p = .008				.21
Depressive Behavior				
Step 1: Direct effects model				
Maternal depression at child ages 6–8	.28	2.23	.029	
Older brother antisocial at child ages 6–8	.04	.31	.761	
Gender of target child	.02	.16	.870	
Entered as a block; F (3, 69) = 2.14, p = .103				.09
Step 2: Family coercion as a mediator				
Maternal depression at child ages 6–8	.30	2.42	.018	
Older brother antisocial at child ages 6–8	−.11	−.74	.458	
Gender of target child	.02	.20	.843	
Family coercion at child ages 6–8	.28	2.11	.039	
Entered as a block; F (4, 68) = 4.45, p = .039				.14
Step 3: Child gender as a moderator of family coercion				
Gender of Target Child × Family Coercion	.26	2.07	.042	
F (1, 67) change = 4.30, p = .042				(.06)
F (5, 67) total model = 2.61, p = .043				.20

which is an indirect effect (Holmbeck, 1997). The family processes by which early maternal depression increases children's risk for adolescent depression were not successfully specified by the coercion model.

Late socialization: Association of older brothers' antisocial behavior and sibling coercive process with younger siblings' antisocial and depressive behavior

Tests of models of younger siblings' exposure at mean ages 10 and 12 to older brothers' antisocial behavior and to sibling coercion at age 16 on younger siblings' antisocial behavior at mean age 16 are shown in the top of Table 4. Older brothers' antisocial behavior at child ages 10 and 12 was a robust predictor of their younger siblings' antisocial behavior at age 16, accounting for considerable criterion variance (see Step 1). Child gender was not a reliable predictor. Sibling coercion at age 16 was also a reliable (p = .001) predictor when entered simultaneously with older brothers' antisocial behavior (see Step 2, top of Table 4), but the older brothers' antisocial term remained significant. This suggests that older brothers' antisocial behavior had an independent, or additive, rather than a mediated, influence. This additive model was reliable and increased the amount of outcome variance predicted by 9%. The addition of a younger

sibling gender by coercion moderator did not reliably increase prediction or model fit (Step 3, top of Table 4); the association of sibling coercion in adolescence on younger siblings' antisocial behavior was gender neutral.

The association of younger siblings' exposure to older brothers' antisocial behavior at ages 10 to 12 and to maternal depression[4] at ages 6 to 8 with younger siblings' depression at mean age 16 is shown as Step 1 in bottom portion of Table 4. Neither older siblings' antisocial behavior nor younger sibling gender served as reliable predictors of younger siblings' adolescent depression. Early exposure to maternal depression, as shown in the previous early socialization analyses, was a reliable predictor of child depression some 10 years later, at age 16. As shown in Table 2, sibling coercion was marginally correlated with younger siblings' depressive behavior at age 16, and with older brothers' antisocial behavior at younger siblings' ages 10 to 12 (both ps $< .10$); older brother antisocial behavior was unrelated to younger siblings' age 16 depression, and early exposure to maternal depression was not reliably correlated with sibling coercion at age 16. As such, the prerequisites for testing a mediational model were not met, so such a model is not supported. However, concurrent involvement in sibling coercion, when added to the direct effects model (see Step 2, bottom of Table 4), was a marginally reliable predictor of child depression and increased the amount of criterion variance accounted for by 4%. Thus, sibling coercion during adolescence may have a modest additive impact on adolescent depression in the presence of early exposure to maternal depression. However, this model was not reliable ($p = .112$). The role of concurrent sibling coercion in risk for younger siblings' depression at age 16 appears to be moderated by child gender (see Step 3, bottom of Table 4). The addition of a younger sibling gender by sibling coercion moderator term increased the fit of the model ($p = .049$) and incremented prediction of variance in adolescent depression from 13% to 19%. Younger sisters' but not younger brothers' risk for depressive behavior in adolescence increased with increases in concurrent coercive interaction with an older brother was more extensive, as shown in Figure 1(b).

Co-occurrence models

Up to this point in the analyses, the role of the family ecology on adolescent outcomes was considered separately for depression and antisocial behavior. It is possible that the family's role in the development of one problem (e.g., depression) may be mediated by a second problem (e.g., antisocial behavior), reflecting the developmental sequencing and causal relationship between the two. This hypothesis is reasonable, given the reliable correlation between age 16 depression and antisocial behavior for current sample of boys and girls. Repeated measurement of antisocial and depressive behavior from childhood through adolescence is needed to provide a strong test of their sequential and causal relationships during development and the degree to which they share common risk factors. Such repeated measures are not available for younger siblings in the OYS. However, this hypothesis was tested in a weak way by reassessing each of the models shown in Tables 3 and 4 after adding either age 16 adolescent depression (for models predicting antisocial behavior) or antisocial behavior (for models predicting depressive behavior) in the first step of the regression models. This addresses the question of whether maternal depression, older sibling antisocial behavior, and early family and later sibling coercion still predict a given age 16 outcome when first accounting for its association with the other age 16 outcome.

Age 16 depression was reliably associated with age 16 antisocial behavior in both the early and late socialization models; it incre-

4. Early exposure to maternal depression was integrated into this late socialization model post hoc for several reasons. First, the potential role of such depression in facilitating later sibling coercion was of interest. Second, we wanted to ascertain the power of maternal depression by assessing its continuing predictive role in the presence of later socialization influences. The significance of the regression weights in the late socialization model for child depression was largely unchanged when maternal depression was excluded from the analyses.

Table 4. *Tests of a late coercion model of adolescent antisocial and depressive behavior of boys and girls*

	β	*t*	*p*	ΔR^2
Antisocial Behavior				
Step 1: Direct effects model				
Older brother antisocial at child ages 10–12	.56	5.66	.000	
Gender of target child	−.12	−1.24	.219	
Entered as a block; F (2, 70) = 19.16, p = .000				.36
Step 2: Sibling coercion as a mediator				
Older brother antisocial at child ages 10–12	.48	5.08	.000	
Gender of target child	−.10	−1.11	.271	
Sibling coercion at child age 16	.31	3.32	.001	
Entered as a block; F (3, 69) = 11.03, p = .001				.45
Step 3: Child gender as a moderator of sibling coercion				
Gender of Target Child × Sibling Coercion	.17	.92	.357	
F (1, 68) change = .86, p = .357				(.01)
F (4 ,68) total model = 16.30, p = .000				.46
Depressive Behavior				
Step 1: Direct effects model				
Older brother antisocial at child ages 10–12	.06	.50	.621	
Gender of target child	−.01	−.02	.977	
Maternal depression at child ages 6–8	.23	1.99	.050	
Entered as a block; F (3, 69) = 2.11, p = .106				.09
Step 2: Sibling coercion as a mediator				
Older brother antisocial at child ages 10–12	.02	.17	.858	
Gender of target child	−.01	−.08	.940	
Maternal depression at child ages 6–8	.21	1.85	.068	
Sibling coercion at child age 16	.20	1.80	.077	
Entered as a block; F (4, 68) = 2.53, p = .112				.13
Step 3: Gender of child as a moderator of sibling coercion				
Gender of Target Child × Sibling Coercion	.24	2.21	.030	
F (1, 67) change = 3.97, p = .049				(.06)
F (4, 67) total model = 2.97, p = .044				.19

mented the predicted variance in antisocial behavior by 8–9%. Similarly, age 16 antisocial behavior was reliably associated with age 16 depression in both the early and late socialization models; it incremented the predicted variance in depression by 7–9%. This is expected given the correlations between these outcomes shown in Table 2. The addition of concurrent age 16 outcomes to these models did not reduce the reliability of any of the family-level predictors (including mediator and moderator effects) in any of the four models. The only change occurred in the late socialization model for depressive behavior (bottom of Table 4) in which, with the addition of age 16 antisocial behavior as a predictor in Step 1, older brothers' antisocial behavior became a reliable, inverse predictor (b = −.27, p = .046) of younger sibling depression four years later at age 16. Exposure to an antisocial older brother increased risk for younger siblings' antisocial behavior (top half of Table 4) and decreased their risk for depression 4 years later. This is akin to a suppressor effect and should be interpreted cautiously.

Discussion

The results provide partial support for the hypothesized models. Antisocial and depressive behavior in midadolescence were associated with chronic involvement in coercive family interaction, as measured both during earlier childhood and concurrently in midadolescence.

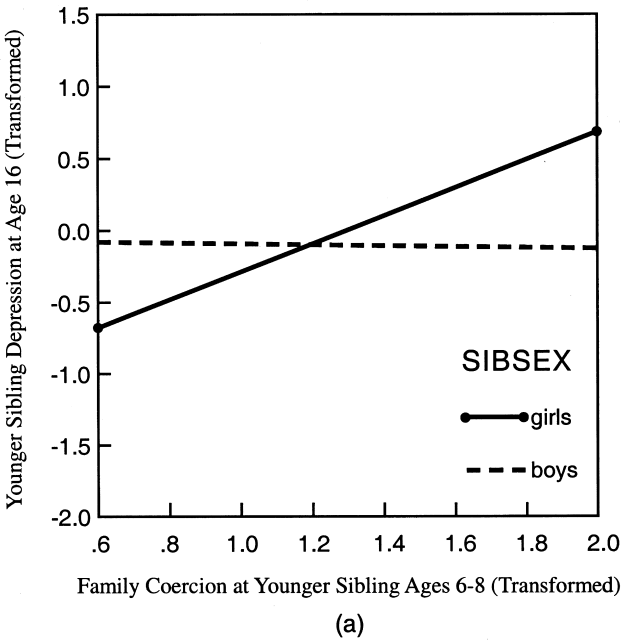

Figure 1. The gender-moderated relationships of involvement in (a) early family coercion and (b) later sibling coercion to adolescent depression.

The association of coercive family interaction with adolescent depression was conditional on child gender, whereas its association with antisocial behavior was not. Involvement in coercive family interaction increased both boys' and girls' risk for antisocial behavior, but it increased risk for depression only for girls. In this sense, coercive family interaction appears to place girls in double jeopardy and may serve as a common pathway for comorbid

conduct problems and depression. Epidemiological data suggest that there is also substantial comorbidity for conduct problems and depression in boys during late adolescence and young adulthood, but the data from this research suggest that the pathway to comorbid expression may be gender specific.

These findings replicate previous research indicating that familial coercion generally (Patterson et al., 1992), and sibling coercion more specifically (Bank, Patterson, & Reid, 1996), may serve as processes that increase risk for child antisocial behavior. The results extend previous research in several ways. Family coercion was associated with increased risk for antisocial behavior regardless of child gender, thus extending to girls the relevance of models previously derived primarily from boys. It complements older (Patterson, 1984) and more recent research (Bank et al., 1996) suggesting that siblings play an important role in the family coercion process and in the development of antisocial behavior of younger siblings (Rowe et al., 1996). Siblings' contribution to family coercion and antisocial behavior appears to span long periods of development, beginning in the preschool years and continuing through adolescence.

As previously documented (Pike et al., 1996; Rowe & Gulley, 1992), substantial cross-sibling similarity in antisocial behavior was observed in the current research, even across measurements taken a decade apart. Involvement in coercive family interaction may be one process by which cross-sibling similarity in antisocial behavior is engendered. The association of childhood exposure to highly antisocial older brothers with younger siblings' antisocial behavior in adolescence appeared to be mediated by intrafamilial coercion. Exposure to an antisocial older brother in late childhood and involvement in coercive sibling interaction in adolescence increased younger siblings' risk for adolescent antisocial behavior in an additive fashion. These models for antisocial behavior applied to both boys and girls.

Although a directional (from older to younger sibling) model of influence was tested in this research, coercive family interaction is more likely a systemic social process. Children, their siblings, and their parents all contribute to and are affected by their involvement in the process. The chronicity of exposure is also likely to be the product of transactional developmental processes. Family members who are irritable and aggressive as individuals are likely to engage one another in coercive ways. Persistent involvement in coercive family interchange, in turn, is likely to sustain and amplify irritability and aggressiveness in all family members. As such, models supported in the current research probably represent only one aspect of a more complex route by which antisocial behavior is engendered and sustained within families.

Risk for adolescent depression appears to be associated with exposure to high levels of familial and sibling coercion both earlier and later in socialization. Based on the gender-differentiated moderator effects, the risk for depression associated with increasing involvement in coercive family and sibling interaction appears to be greater for girls than boys. Developmentally chronic involvement in coercion may place girls at increasing risk for both antisocial and depressive behavior. This provides modest support for the coercion formulation offered by Hops and his colleagues (Davis et al., 2002; Hops, 1996) in which depressive, as well as antisocial, behaviors occur as common responses to chronically and highly aversive family environments. The gender-differentiated risk associated with exposure to family coercion is consistent with commonly observed gender differences in the prevalence of depression in adolescence (Lewinsohn et al., 1993). Chronic involvement in coercive family environments may exacerbate already existing, normative gender differences in response to social challenge that are shaped in family, peer, and school social settings (Maccoby, 1998).

Data from this study suggest that residing in a household with an antisocial older brother does not directly increment girls' (or boys') risk for depression. However, the increasing involvement in coercive family interchange that accompanies the presence of a highly antisocial older brother may serve as an indirect social process that is associated with increases in adolescent depressive behavior by girls.

This gender-specific, indirect effect is open to a number of interpretations. One possibility is that the initiation and the resolution of conflicts involving older brothers and younger sisters are asymmetrical. Younger sisters relative to younger brothers might have less physical prowess and skill in fighting with their older brothers. Girls' responses to conflicts with antisocial older brothers may more often entail capitulation and withdrawal than those of younger brothers. Some observational data support this asymmetry (Patterson, 1986; Pepler, Abramovitch, & Corter, 1981) and some do not (Patterson, 1984). The asymmetry interpretation along with the double jeopardy associated with girls' exposure to familial coercion is consistent with suggestions that the developmental history of delinquent girls often involves victimization (Giordano & Cernkovich, 1997).

Some aspects of the hypothesized model were clearly not supported. Boys' and girls' early exposure to maternal depression was unrelated to their later antisocial behavior, either directly or as mediated through the coercive family processes as assessed in this research. The impact of maternal depression on child antisocial behavior may be time limited, consistent with the significant correlation between maternal depression and the concurrent antisocial behavior of the OYS boys at ages 10–12, but a nonsignificant correlation of maternal depression with OYS boys' antisocial behavior 4 years later (see Table 2), as well as with that of the targeted younger siblings 8–10 years later. The specificity of the impact of maternal depression on child outcomes is not well understood and may depend on both the level of maternal depression (Downey & Coyne, 1990) and its timing and chronicity in relation to children's development (Goodman & Gotlib, 1999). Maternal depression may impact the development of child antisocial behavior via processes other than family coercion.

Children's exposure to maternal depression during the early elementary school years was reliably related to their depressive symptoms in adolescence 8–10 years later, both for boys and girls. This association occurred even in the context of the contribution of family and sibling coercive exchange to child depressive behavior in adolescence. The risk conveyed by maternal depression was not mediated by early family coercion, an outcome that fails to support the proposed model. Several alternatives might be proposed. The direct effect of maternal depression may reflect the role of genes or similarities in maternal and child temperament. The effect may be mediated by processes not captured in this research. One such process may entail exposure to maternal depressive affect, behavior, and talk that promotes a latent predisposition for the display of similar affect, behavior, and talk by children. This predisposition may then be activated when children are exposed to aversive familial (Hammen, 1996) or extrafamilial events such as peer rejection and school failure (Capaldi & Stoolmiller, 1999).

The reliability of coercive family process in predicting adolescent depression and antisocial behavior after controlling for their concurrent co-occurrence provides some increased confidence about the role of aversive family environments in increasing risk for both outcomes. Although it is likely that depression and antisocial behavior in adolescence reciprocally influence and exacerbate one another, they appear to arise from common environmental experiences, in this case, chronic exposure to a coercive family environment, consistent with the notion of multifinality.

The association of family coercion with increased risk for the development of antisocial behavior, depressive behavior, and their co-occurrence is subject to a number of interpretations. A functionalist–reinforcement perspective would infer that opposition/aggression and dysphoria/withdrawal are alternative behavior sets that can be used to cope with highly aversive and non-supportive social environments. The degree to which each set is used depends on its value in palliating aversive social exchange and in attaining attention in the short term (Cummings & Davies, 1994; Davis et al., 2002; Hops, 1996). Cognitive models would infer that chronic exposure to aversive family environments results in negative cognitions and emotions (Alessandri & Lewis, 1996; Alloy & Abramson, 1988; Garber & Flynn, 1998) that may involve both hostile and depressive attributional styles and

associated anger and sadness (Quiggle, Garber, Panak, & Dodge, 1992). A third model might infer that growing up in an aversive family environment and with a depressed mother interferes with parent–child attachment and facilitates the development of representational models of social relationships in which others are perceived as untrustworthy and unpredictable (Cicchetti & Toth, 1998). Such working models are associated with difficulties in emotion regulation and with maladaptive behavioral strategies that have been associated with increased risk for both the early appearance of disruptive behavior (Shaw & Vondra, 1995) and adolescent depression (Hammen, Burge, Daley, Davila, Paley, & Rudolph, 1995). Finally, the covariation of depressive and antisocial behavior among family members may reflect shared genes. Reciprocal involvement in coercive family interaction may be one of several processes by which genotype is expressed phenotypically (Maccoby, 2000).

In some respects, the current analyses are conservative. There was very little method overlap between family predictors and child outcomes, thus minimizing shared method variance as a competing explanation for the results. Most of the empirical relationships being examined were prospective in nature. The analyses were somewhat underpowered; a Satoris–Saris univariate power analysis indicated that, given the sample size in the current analyses, the power to detect a regression weight of .30 is approximately .60.

On the other hand, the current data are inadequate in several respects. Depression and antisocial behavior were measured at only one developmental point. Thus, the degree to which the family environment would be similarly associated with these outcomes after controlling for earlier manifestations of antisocial and depressive behavior could not be determined. Given the considerable continuity in antisocial behavior (Patterson, 1993), the observed association between younger siblings' antisocial behavior in adolescence and their earlier exposure to coercive environments may be fully mediated by the antisocial behavior of younger siblings at an earlier age. However, such autoregressive models are themselves limited; they primarily represent rank-order continuity in individual differences. Such models fail to provide adequate opportunity to estimate the contribution of processes associated with other important aspects of developmental trajectories, including age changes in the magnitude of individual differences (increased or decreased variance) and between-individual differences in the direction and amount of change over time (slopes or growth curves; Stoolmiller & Bank, 1995). The lack of repeated measures of antisocial and depressive behavior clearly limits the interpretation of the data in this study, but autoregressive models represent only one and a somewhat limited perspective on specifying sources of variation and change in development. The limited geographic location, the specific family demographic attributes, and the neighborhood selection criteria characterizing the OYS sample may limit generalization of the findings. The manner in which maternal depression, for example, is related to family interaction and child outcomes may be diminished or moderated by the high levels of antisocial behavior apparent in the OYS sample. Alternately, the nature and expression of maternal depression may be different than would be found in more representative, epidemiological samples. The relations observed among variables vary as a function of the sample under consideration (Cummings, Davies, & Campbell, 2000).

Several steps are needed to more clearly ascertain the processes by which chronic exposure to coercive family environments increment risk for the independent and overlapping occurrence of depressive and antisocial behavior. First, the display of depressive behaviors in family interaction, both by children and by parents, needs to be explicitly coded, in addition to coding verbal aggressiveness and irritability (Davis et al., 2002). Second, affect and cognitions, as well as overt behavior, need to be assessed to ascertain how risk may be engendered by multiple processes. Third, antisocial and depressive behavior need to be assessed repeatedly from early childhood through adolescence. The contribution of various risk factors and processes to growth on one developmental trajectory may be mediated or moderated by growth on another trajectory. Collection of data on both trajectories

is needed to model their co-occurrence, sequential timing, and developmental dynamics. Fourth, the contribution of fathers and older sisters, as well as that of mothers and older brothers, would provide a more complete, family-systemic picture of coercion and other social processes associated with developmental shifts in children's liability for antisocial behavior, depression, and their co-occurrence. Ultimately, family-based risk factors and processes must be integrated with risk emanating from extrafamilial environments and genetic influences.

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