

Developmental Psychology (2003)

doi: 10.1037/0012-1649.39.2.309

Integrating Biological and Social Processes in Relation to Early-Onset Persistent Aggression in Boys and Girls

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This study was supported by funds from the National Health and Medical Research Council in Australia, the Mater Misericordiae Mother's Hospital in Queensland, Australia, and National Institutes of Health Grant R01 MH52239. We are grateful to the Brisbane project staff, Robyne LeBrocq, Cheri Dalton, Barbara Mann, Eileen Tone, Sandra Fergusson, Molly Robbins, and Lisa Manning. We also thank Margaret Andersen and Michael O'Callaghan for their contributions to the longitudinal study of the Mater University Study of Pregnancy birth cohort and Sharon Foster and Eric Vanman for their helpful comments on a draft of this article.

Abstract

This study examined the relationship between biological and social risk factors and aggressive behavior patterns in an Australian high-risk sample of 370 adolescents. Perinatal, temperamental, familial, sociodemographic, and behavioral data were collected during interviews completed during pregnancy, immediately postpartum, and when the children were 6 months old and 5, 14, and 15 years old. Youths were given tests of verbal and neuropsychological functioning at the age 5 and age 15 follow-ups. Youths were divided into early-onset persistent aggression, adolescent-onset aggression, and nonaggressive behavior groups. Results revealed that the interaction of biological and social risk factors was significantly related to early-onset persistent aggression. Gender and developmental phase of measurement moderated the relationship between biosocial risks and the outcomes of early-onset persistent aggression and adolescent-onset aggression.

The developmental course of aggression, and its evolution into adult criminal offending, has long been a source of concern and contention for researchers and public policymakers alike. In this area of inquiry, two distinct developmental pathways for youthful offending have been observed: the early-onset persistent type and the late-onset adolescent-limited type ([Moffitt, 1993](#); [Patterson, DeBaryshe, & Ramsey, 1989](#)). In the former case, behavior problems begin in the early years of development; signs of antisocial acting out are sometimes evident as early as 3 years of age ([Moffitt, Caspi, Dickson, Silva, & Stanton, 1996](#)). Early behavior problems give way to serious juvenile delinquency in the adolescent years, which evolves into a stable pattern of adult offending and repeated criminal convictions. Late starters, on the other hand, do not begin offending until middle to late adolescence. The prognosis for this group is

somewhat more promising than for the early-onset group—late starters typically desist from antisocial behavior by their early twenties ([Elliott, Huizinga, & Ageton, 1985](#); [Farrington, 1986](#)). This article focuses on the early-onset persistent group in an investigation of two developmental theories of antisocial behavior and the etiological predictions that follow from a combination of these theories.

Theories that explain antisocial behavior from a developmental psychopathology perspective offer unique advantages over general theories of crime. For instance, as [Rutter \(1997\)](#) has outlined, developmental theories focus on continuities and discontinuities across the life span. Furthermore, the developmental perspective emphasizes the ongoing processes that serve to initiate, maintain, and diminish individual levels of antisocial behavior over time. Awareness of these transactional processes is perhaps more useful than a discourse on general “causes” and correlates of juvenile delinquency, especially for the purposes of informing the future direction of interventions and public policy.

[Patterson's \(1982\)](#) coercion model is one theory that approaches the early-onset persistent offending pattern from a developmental psychopathology perspective. Patterson contended that poor parental monitoring of child activities, disruptive family transitions (e.g., divorce), and inconsistent parental discipline are the major etiological contributors to the early-onset trajectory. He theorized that the key predictor of early-onset persistent offending is a family environment in which the child learns to use coercive behaviors (e.g., temper tantrums, whining) to escape aversive parental discipline. Via this dynamic, the child learns the adaptive value of coercive behavior through escape conditioning; likewise, the parent is taught, through negative reinforcement, to placate the coercive child by acquiescing. Coercion becomes the child's primary interpersonal strategy and thus generalizes to environments outside the home. As parental monitoring and discipline wane, delinquent peers are likely to step in to serve as the proximal cause that facilitates the transition from simple coercive behavior to actual delinquency. Lacking learned prosocial alternatives to coercion, the early-onset delinquent evolves into the persistent adult offender.

[Patterson's \(1982\)](#) coercion model has received impressive support from empirical work involving childhood antisocial behavior and aggression. Studies using correlational ([Block, Block, & Keyes, 1988](#); [Kellam, Brown, Rubin, & Ensminger, 1983](#); [Patterson, 1982](#)) and structural equation modeling techniques ([Biglan et al., 1996](#); [Forgatch, 1991](#); [Metzler & Dishion, 1992](#)) have suggested that family process variables play a prominent role in the development of persistent antisocial behavior. Furthermore, the relationship between social context variables (e.g., neighborhood, poverty) and childhood antisocial behavior is often mediated by parental discipline practices ([Patterson, 1983](#); [Patterson, Reid, & Dishion, 1992](#)) even in high-risk environments ([Sampson & Laub, 1994](#)). In addition, experimental research has demonstrated that family management training for parents produces significant reductions in child antisocial behaviors ([Chamberlain, Ray, & Moore, 1996](#); [Dishion, Patterson, & Kavanagh, 1992](#)).

Another developmental theory that has recently been influential in the study of persistent antisocial behavior is [Moffitt's \(1993\)](#) theory of life-course-persistent offending. Moffitt's theory bears many points of similarity to [Patterson's \(1982\)](#) coercion model, and it also gives prominence to parenting practices as one of the important causal mechanisms in the production of early-onset aggression and delinquency. Moffitt's theory, however, adds an explicit and specific statement about the role of biological factors in this early-onset trajectory. Moffitt theorized that biological deficits and disrupted social environments work

together in a transactional process to produce early-onset persistent offending. According to the theory, prenatal and perinatal disruptions in neural development lead to neuropsychological deficits—namely, developmental impairments in executive and verbal functioning. The result is an infant or toddler with an irritable temperament, poor behavioral regulation, and deficient verbal and reasoning abilities. All three deficits individually, [Moffitt \(1993\)](#) observed, are established predictors of later antisocial behavior. Furthermore, infants at risk for these deficits are also likely to be raised in homes that are poorly equipped, both financially and psychologically, to deal adaptively with a troublesome child. As [Moffitt \(1993\)](#) claimed, “Vulnerable infants are disproportionately found in environments that will not be ameliorative because many of the sources of neural maldevelopment co-occur with family disadvantage or deviance” (p. 681). Thus, the stage is set for a series of maladaptive parent-child interactions in which troublesome child behaviors evoke negative responses from parents, which in turn exacerbate the child's behavior problems. This ongoing transactional process results in an antisocial lifestyle trajectory maintained by a combination of preexisting neuropsychological deficits, the cumulative negative consequences of ongoing antisocial behavior, and the absence of learned prosocial behavioral alternatives.

The biological component of [Moffitt's \(1993\)](#) theory has received substantial empirical support. Numerous studies have demonstrated the high correlation between persistent delinquency and neuropsychological deficits (e.g., [Berman & Siegal, 1976](#); [Brickman, McManus, Grapentine, & Alessi, 1984](#); [Fitzhugh, 1973](#); [Yeudall, Fromm-Auch, & Davies, 1982](#)), offering circumstantial support to Moffitt's contention that such deficits play a key role in the etiology of delinquency. Furthermore, the Verbal IQ deficit among juvenile delinquents is well established (e.g., [Haynes & Bensch, 1981](#); [Lynam, Moffitt, & Stouthamer-Loeber, 1993](#); [Prentice & Kelly, 1963](#); [Walsh, Petee, & Beyer, 1987](#)). Moreover, Moffitt and colleagues have garnered support for the life-course-persistent theory in a series of longitudinal studies involving a large New Zealand birth cohort ([Frost, Moffitt, & McGee, 1989](#); [Henry, Moffitt, & Silva, 1992](#); [Moffitt, 1990a, 1990b, 1993](#); [Moffitt et al., 1996](#); [Moffitt & Henry, 1989](#); [Moffitt & Silva, 1988a, 1988b, 1989](#); [White, Moffitt, Earls, Robins, & Silva, 1990](#); [White, Moffitt, & Silva, 1989, 1992](#)). Although there has been some empirical support for the predictive value of early temperament assessments ([Caspi, Henry, McGee, Moffitt, & Silva, 1995](#); [Henry, Caspi, Moffitt, & Silva, 1996](#)), these findings have been clouded by several methodological limitations (e.g., temperament assessments made at age 3 or later; criterion overlap between predictor and outcome variables). Nevertheless, Moffitt and colleagues have consistently found strong associations among early neuropsychological deficits, low Verbal IQ, and persistent behavior problems throughout the childhood and adolescent years.

Although both developmental theories have individually received strong support, relatively little empirical work has sought to incorporate the advantageous aspects of both in longitudinal designs. Several recent attempts have simultaneously evaluated the developmental consequences of both biological and familial risk for early-onset delinquency ([Aguilar, Sroufe, Egeland, & Carlson, 2000](#); [Fergusson, Horwood, & Nagin, 2000](#); [Patterson, DeGarmo, & Knutson, 2000](#)). Unfortunately, many of these studies have been plagued by a lack of crucial temperamental and neuropsychological assessments in the early years (0–5) of development ([Patterson et al., 2000](#)) as well as by a lack of focus on the most relevant family process variables—parental monitoring and discipline ([Fergusson et al., 2000](#)).

[Aguilar and colleagues \(2000\)](#) addressed these gaps in the literature by giving the children in their study an extensive battery of neuropsychological, temperamental, and psychosocial

assessments in the first 4 years of life and beyond. These investigators reported that children with early-onset persistent behavior problems were characterized by more severe psychosocial adversity in the first few years of life. Infant temperament variables did not predict the early-onset trajectory; neither did early neuropsychological variables. Individuals in the early-onset group differed from others only on later tests of neuropsychological functioning, which suggested that their cognitive deficiencies were progressive rather than present at or near birth. However, a relatively small sample size (62 boys and 58 girls) limited this study's statistical power and precluded the test of gender as a potential moderator. Recent research has suggested that girls may exhibit different developmental trajectories for delinquency ([Jang & Krohn, 1995](#); [Silverthorn & Frick, 1999](#)), and some etiological factors may have differential impacts on delinquent behavior in boys and girls ([Agnew & Brezina, 1997](#); [Cookston, 1999](#); [Kratzer & Hodgins, 1999](#); [Mears, Ploeger, & Warr, 1998](#)). Thus, the aggregation of male and female data in the [Aguilar et al. \(2000\)](#) sample may have obscured the effects of some etiological risk factors.

Thus far, this new line of research simultaneously examining social and biological processes in relation to the development of aggression has generally found a stronger relationship between aggression and social process variables than between aggression and biological risk indices. It is important to note, however, that not one of these studies reported a test of potential biosocial interactions. This is a serious omission in this area of research. The concept of biosocial interaction processes is a crucial element in [Moffitt's \(1993\)](#) theory, and the presence or absence of such interactions has important implications for intervention and prevention strategies focused on aggressive behavior.

The theoretical models of [Moffitt \(1993\)](#) and [Patterson \(1982\)](#) suggest specific biological and social risk factors that may play a potentially important role in the onset and maintenance of aggressive behavior problems. We propose that these two developmental models of antisocial behavior are complementary in nature. Specifically, the coercive response strategy described by Patterson may be more likely to develop and flourish in children with subtle deficits in verbal and executive functions. Furthermore, the coercion model represents a well-specified depiction of the maladaptive parent-child interactional process described by Moffitt. In this way, the etiological factors stressed by Moffitt's biosocial model (namely, neuropsychological deficits) may serve to elaborate on the predictions offered by Patterson's coercion model, and vice versa.

In the present study, we attempted to integrate the risk factors specified in these distinct theories. However, our goal was not to assess the relative importance of a particular biological factor (e.g., temperament) versus a particular family factor (e.g., parental monitoring) in predicting aggressive behavior problems. Our primary goal was to assess cumulative biological risk factors specified by [Moffitt \(1993\)](#), cumulative social risk factors specified by [Patterson \(1982\)](#), and their interaction in relation to aggressive behavior patterns in boys and girls. Although we make what we believe is an important distinction between biological and social risk factors, we used cumulative counts of these types of risk factors rather than examining individual risk factors separately. This cumulative risk approach originated in a study by [Rutter \(1979\)](#) that revealed that the number of risk factors, rather than any particular individual risk factor, best predicted psychiatric outcomes in children. Similar cumulative risk approaches have been shown to have utility in the prediction of a variety of child outcomes from intelligence to competence to behavior problems and also to have a greater predictive utility than specific patterns of individual risk factors ([Sameroff, 1998, 2001](#); [Sameroff, Seifer, Baldwin, & Baldwin, 1993](#); [Sameroff, Seifer, & Bartko, 1997](#); [Williams, Anderson, McGee, &](#)

[Silva, 1990](#)). In addition, the cumulative risk approach is consistent with the idea that many predictors likely have a nonlinear relationship to aggressive outcomes; and it is only when the level of risk is extreme that it affects aggressive behavior.

The present study addresses the shortcomings of previous efforts to integrate the prediction models of [Moffitt \(1993\)](#) and [Patterson \(1982\)](#) in two ways: First, the sample is large ($N = 370$), which contributes to statistical power and also permits the disaggregation of data for boys and girls. Second, the data set includes several early assessments of neuropsychological functioning and temperament as well as a number of social risk variables that are germane to the coercion process.

Thus, in the present study we incorporated the most relevant risk variables from two well-supported theories in order to determine if a hybrid of the two models would predict antisocial behavior outcomes more precisely than would either model alone. We focused on the specific aggressive behavior patterns that have been differentiated by the theoretical models of [Moffitt \(1993\)](#) and [Patterson \(1982\)](#): early-onset persistent, adolescent-onset, and nonaggressive. We hypothesized that the ability of the coercion model to predict which children will exhibit the early-onset trajectory would be improved by adding biological risk indices as predictor variables. We also hypothesized that the interaction between biological risks and social risks would predict early-onset persistent aggression but not adolescent-onset aggression. The biological and social risk factors that are the basis of this study are those most relevant to early-onset rather than adolescent-onset aggression. Therefore, we hypothesized that in terms of these biological and social risk factors, the children who evidenced adolescent-onset aggression would not differ significantly from the children who were classified as nonaggressive in our sample. Given both Patterson's and Moffitt's exclusive focus on boys' aggression, we explored whether these relationships would be similar or different for the boys and girls in our sample. And given the findings of [Aguilar and colleagues \(2000\)](#), we also explored whether biological and social risks measured early in childhood would have a different effect than biological and social risks measured in adolescence.

Method

Participants

Birth Cohort

Participants in this study were 370 fifteen-year-old adolescents and their mothers. Participants were drawn from a larger cohort of youths ($N = 7,775$) born between 1981 and 1984 at the Mater Misericordiae Mother's Hospital in Brisbane, Queensland, Australia. The goal of the original cohort study (the Mater University Study of Pregnancy [[MUSP](#)]; [Keeping et al., 1989](#)) was to examine social factors and children's health and development in the context of a community sample. Children in this birth cohort were representative of individuals born in public hospitals in Queensland, and they therefore represented a relatively lower

socioeconomic sector (working and lower-middle class) of the population of Australia. Mothers in the MUSP completed interviews and questionnaires about themselves and their children at five different times—during pregnancy, 3–4 days after the birth of the child, 6 months after the birth of the child, and when the child was 5 and then 14 years of age. In addition, hospital perinatal records were coded at birth, and the children were directly assessed for cognitive functioning at age 5.

High-Risk Subsample

The sample for the current study was drawn from a high-risk subsample of the larger birth cohort on the basis of maternal self-reports of depression from pregnancy through the age 5 follow-up. At each of the MUSP contacts, the mothers had completed a depression scale, the Delusions-Symptoms-States Inventory (DSSI) of [Bedford and Foulds \(1978\)](#). The DSSI had been chosen as the measure of maternal mental health for the Mater Hospital birth cohort study because it was a valid screening instrument for mental health (e.g., [Bedford & Foulds, 1977](#)) and did not include symptoms that might be confused with the effects of pregnancy or childbirth.

The high-risk subsample was to be used in an intensive follow-up study of a large sample of depressed and nondepressed women and their children. Therefore, women's DSSI scores at the initial four testings were reviewed, and on the basis of the mothers' patterns of elevated scores, families were targeted for inclusion when their children became 15 years old. The goal was to include as many women with putative depressive disorders as possible, with elevated scores (representing both chronicity and severity) varying in frequency, along with a sample of comparison women who had no or few depressive symptoms. Specifically, women were included if they indicated severe depression at two or more times, severe depression only once between pregnancy and when their children were 5 years old, moderate depression at two or more times but never severe depression, or low depression at all assessment phases.

A total of 991 families were targeted for inclusion in the high-risk subsample. Of the 991, 816 consented to participate and were included (82%), 68 could not be located, 103 declined to participate, 3 could not participate because of a child's hearing or visual impairment, and 1 had a child who died. The high-risk youths and their parents were administered interviews and questionnaires in their homes when the youths were 15 years old. The age 15 follow-up focused on family history of psychopathology (primarily maternal depression) and adolescent diagnostic, social, and behavioral outcomes. Diagnostic interviews administered to the mothers at the age 15 follow-up revealed that 358 mothers in this high-risk subsample (44%) had a lifetime history of major depression or dysthymia. Children in the high-risk subsample were not significantly different from the original birth cohort in terms of gender, $\chi^2(1, n = 7,775) = 0.53, p = .48$; income, $t(7,147) = 0.81, p = .42$; or mother's education, $t(7,612) = 1.70, p = .09$. However, the subsample had fewer ethnic minority members (8.6% vs. 11.3%), $\chi^2(3, n = 7,449) = 4.46, p < .05$, and fewer older mothers, $t(7,773) = 1.98, p < .05$, than the unselected birth cohort from which it was drawn.

Sample for Current Study

Six of the families in the high-risk subsample were excluded from the current study because of missing data that precluded classification of the adolescent's aggressive behavior patterns. Another 440 families were excluded because their aggressive behavior patterns could not be definitively classified into the three categories of aggressive behavior that served as the focus of this study (see below). The 370 remaining adolescents in this sample consisted of 198 boys and 172 girls, and their mean age was 15 years 2 months ($SD = 0.28$). Ninety-one percent of the study sample were Caucasians, the median family income was AU\$35,000–\$45,000, the mothers' median education was Grade 10 (approximately equivalent to graduation from a U.S. high school), and the mothers' mean age at the time of the child's birth was 25.8 years ($SD = 5.2$).

Children excluded because of missing data on aggression measures or inability to be classified into an aggression subgroup did not differ from the other children in the high-risk subsample in terms of youth age, $t(814) = 0.10, p = .92$; mother age, $t(814) = 1.96, p = .06$; mother education, $t(809) = 0.14, p = .89$; family income, $t(812) = 0.63, p = .53$; gender, $\chi^2(1, n = 816) = 2.09, p = .15$; ethnicity, $\chi^2(3, n = 816) = 3.51, p = .32$; self-reported nonviolent delinquency, $t(806) = 0.82, p < .41$; or self-reported violent delinquency, $t(806) = 0.45, p < .65$.

Measures

Cumulative Risk Counts

Each individual biological and social risk factor was dichotomized to allow for both a total count of each of these types of risks and comparable odds ratios across counts of social and biological risks. Unfortunately, sensitivity and specificity analyses are not available for the majority of the risk measures used in our study. Therefore, we had to dichotomize our items on the basis of an alternative method. Our preferred method was qualitative and conceptual in nature rather than strictly quantitative. For example, we did not arbitrarily make the cutoff at the most extreme 10% scores on all of our risk measures. Instead, we attended to the meaning of the scores for that particular risk variable and to which scores conceptually represented negative risk. For example, a score on a scale of maternal hostility might be calculated as the sum of responses (each scored from 1 to 5, from *strongly disagree* to *strongly agree*) on 10 items representing a mother's hostile behaviors toward the child. Scores on this scale could range from 10 to 50, with higher scores on this scale representing more hostile behaviors by the mother. Ten percent of the sample may have a mean score on this scale of 20 or higher. However, a score of 20 actually reflects a lack of hostility toward the child (it suggests a mean response of 2, which indicates disagreement with the scale item). Only total scores higher than 30 conceptually reflect a hostile behavior style on the part of the mother. In this instance, we would establish our cutoff for the presence of this risk factor at 30 rather than at 20. Some of our risk factor variables did not allow for this type of qualitative, conceptual analysis. In these instances, to establish dichotomization we used either (a) an established normative cutoff (e.g., Peabody Picture Vocabulary Test scores less than 85), (b) an operationalization of risk used in previous analyses with this cohort (e.g., income below the 25th percentile), or (c) a 10th percentile cutoff point (e.g., the highest 10th percentile of Stroop difference scores).

Many of our biological and social risk variables were measured concurrently with the outcome of aggression at the age 15 follow-up. We have included these measures for the following reasons: (a) because of their higher quality and standardization relative to the measures obtained in earlier follow-ups, (b) because many of them were obtained from sources other than maternal report, which reduces method variance between our independent and dependent variables, and (c) because these variables are central to [Patterson's \(1982\)](#) and [Moffitt's \(1993\)](#) theories. The method of counting risk factors across all developmental phases substantially impairs our ability to infer a temporal order between these biological and social risk factors and aggressive behavior problems. Therefore, we have included supplemental analyses that examined risk counts involving measures obtained at age 5 or below (i.e., mother's negative attitude toward the infant, mother's report of harsh discipline style, mother's permissiveness, poor maternal education, exposure to poverty, high number of family transitions, perinatal and birth complications, maternal illness during pregnancy, infant temperament, age 5 receptive vocabulary) versus risk counts involving measures obtained at the age 15 follow-up (inadequate parental monitoring, parental firm control, parental acceptance, maternal hostility, age 15 vocabulary scores, and age 15 executive functioning measures).

Social Risks

Social and familial risk factors were chosen to reflect [Patterson's \(1982\)](#) theory of early-starter aggression. These risk factors included the following: mother's report of a negative attitude toward the infant, mother's report of a harsh discipline style, maternal permissiveness, inadequate parental monitoring, youth perception of lack of paternal and maternal control, youth perception of lack of paternal and maternal acceptance, youth-perceived maternal hostility, poor educational background of the mother, exposure to consistent poverty, and a high number of family transitions.

Mother's report of negative attitude toward the infant

When the infants were 6 months old, their mothers were asked about the degree to which they agreed with the following statements: (a) "Caring for my baby is very satisfying," (b) "I feel so angry that sometimes I could smack my baby," (c) "My baby makes me too tired," (d) "My baby is so good I hardly know he/she is there," (e) "Sometimes I feel like hitting my baby," and (f) "I feel fed up looking after my baby all day." A scale of maternal negative attitude was constructed by summing the items after reversal of the first and fourth items; the scale produced an alpha of .77. Youths whose mothers agreed, on average, with the statements reflecting a negative attitude toward their infants were operationalized as having the social risk factor of early maternal negative attitude (9% of the sample).

Mother's report of harsh discipline style

During the age 5 follow-up, mothers were asked about their displays of affection and their discipline practices toward their children. Youths whose mothers reported both a low level of

affectionate behaviors (i.e., at times too busy to comfort the child and not wanting to cuddle the child) and a tendency to use physical punishment to discipline the child were operationalized as having the social risk factor of maternal harsh discipline style (10% of the sample).

Maternal permissiveness

When the children were 5 years old, mothers were asked at what age they thought it would be appropriate to allow children to independently do the following: travel on a bus alone, go to the movies with a friend, go on a holiday with a group of friends of the same age (unsupervised), smoke cigarettes, stay alone in the house while the parent was away, and drink alcohol. The mean age was calculated across these items. Youths whose mothers reported a mean age of less than 14 years (8% of the sample) were operationalized as having the social risk factor of maternal permissiveness.

Inadequate parental monitoring

At the age 15 follow-up, mothers were asked how often (*often, sometimes, almost never*) the following situations occurred: (a) "If you are not at home, does your child know how to get in touch with you?" (b) "How often do you know where your child is when he or she is not at home?" and (c) "How often do you know who your child is with when he or she is not at home?" These items were summed into a scale of parental monitoring ($\alpha = .73$). Youths whose mothers did not answer "often" for two or more of these items were operationalized as having the social risk factor of inadequate parental monitoring (8% of the sample).

Youth perception of paternal and maternal firm control

Youth perception of paternal and maternal control was assessed with the Firm Control subscale of the Children's Report of Parental Behavior Inventory (CRPBI; [Schludermann & Schludermann, 1988](#)). Youths completed the CRPBI separately for their fathers and their mothers at the age 15 follow-up. The Firm Control subscale included items such as "My mother [father] believes in having a lot of rules and sticking with them" and "My mother [father] is very strict with me" and reversed scored items such as "My mother [father] lets me off easy when I do something wrong" and "My mother [father] lets me go any place I please without asking." The alpha for the paternal Firm Control scale was .80, and the alpha for the maternal Firm Control scale was .76. The same cutoff score (15 or lower, indicating that, on average, the parent was "not like" the items in the subscale) was used to define youth perception of lack of control on the part of either parent. Six percent of the father scores fell into the social risk category of youth perception of lack of paternal control, and 8% of the mother scores fell into the social risk category of youth perception of lack of maternal control.

Youth perception of paternal and maternal acceptance

Youth perception of parental acceptance was assessed with the Acceptance subscale of the CRPBI. The Acceptance subscale includes items such as “My mother [father] believes in showing love for me” and “My mother [father] gives me a lot of care and attention.” In this sample, the alpha for the paternal Acceptance scale was .93, and the alpha for the maternal Acceptance scale was .89. The same CRPBI acceptance cutoff score (15 or lower, indicating that, on average, the parent was “not like” the items on the scale) was used to define youth perception of lack of acceptance on the part of either parent. Fifteen percent of the father scores fell into the social risk category of youth perception of lack of paternal acceptance, and 5% of the mother scores fell into the social risk category of youth perception of lack of maternal acceptance.

Youth perception of maternal hostility

At the age 15 follow-up, youths completed a 24-item scale about their mothers that contained items reflecting psychological hostility (e.g., “How often does your mother try to make you feel guilty?”), verbal hostility (e.g., “How often does your mother call you bad names?”), and physical hostility (e.g., “How often does your mother hit, push, grab, or shove you?”). Responses were made on a Likert-type scale from 1 (*always*) to 7 (*never*) (with 4 = *about half the time*). Coefficient alpha for this maternal hostility scale was .93. Youths who reported that their mothers' behaviors were, on average, hostile more than half of the time were defined as having the social risk factor of perceived maternal hostility (7% of the sample).

Poor educational background of the mother

Youths were considered to have this social risk factor if their mothers had not graduated from high school when the youths were born (16% of the sample).

Exposure to consistent poverty

Although the proportion of the Australian population living in poverty was estimated at approximately 15% of the general population at the time of the 5-year follow-up, our sample was skewed toward the low-income spectrum. In addition, there was a need to operationalize income cutoffs in a way that would be consistent across all follow-up phases of the study. On the basis of these considerations, the 25th percentile for each phase was selected as the cutoff below which gross family income was considered to reflect poverty. A similar cutoff has been used in previous studies that examined child outcomes in relation to poverty in the birth cohort from which the present sample was selected ([Bor et al., 1997](#)). Youths whose families fell into the lowest quartile of income at the prenatal, 6-month, and 5-year follow-ups were considered to have the social risk factor of exposure to consistent poverty (7% of the sample).

High number of family transitions

Youths whose mothers reported that they had two or more changes in marital status between the time the youths were born and the age 5 follow-up were considered to have the social risk factor of high family transitions (9% of the sample).

Biological Risks

Biological risk factors were chosen to reflect [Moffitt's \(1993\)](#) life-course-persistent offender theory. These risk factors included the following: high numbers of perinatal and birth complications, maternal illness during pregnancy, infant temperament problems, low receptive vocabulary scores at age 5, low Vocabulary IQ scores at age 15, and deficits on two separate neuropsychological measures of executive functioning at age 15. As with the social risk factors, each biological risk factor was dichotomized (0 vs. 1), and a total count of biological risks was calculated.

Perinatal and birth complications

Hospital records were used to establish gestational age, birth weight, Apgar scores, time to establish infant respiration, and neonatal intensive care placement. Maternal self-reports of cigarette and alcohol consumption during pregnancy were obtained from a prenatal interview with the mother. The presence of any one of the following perinatal risks or birth complications resulted in a score of 1 for this biological risk factor: low gestational age (less than 37 weeks at birth), low birth weight (below 2,500 g), low Apgar score (a score of 7 or less 5 min after birth), 3 or more minutes to establish infant respiration after birth, placement in neonatal intensive care, maternal prenatal smoking at the rate of 10 or more cigarettes per day, and maternal prenatal alcohol consumption at the rate of two times per week or more. Ten percent of the sample was considered to have this type of biological risk factor.

Maternal illness during pregnancy

At the immediate postpartum assessment, mothers reported whether they had no, minimal, moderate, or major problems with morning sickness, constipation, heartburn, backache, vaginal infection, leg cramps, and generally feeling unwell. Youths whose mothers reported moderate or major problems in these areas were considered to have the biological risk factor of maternal illness during pregnancy (7% of the sample).

Infant temperament

The mothers were asked at 6 months if their infants had experienced a range of behaviors that could be taken as a measure of infant temperament (i. e., sleeplessness, feeding problems, and overactivity). A scale was created that measured the number of behaviors reported by the mother ($\alpha = .62$). Infants whose mothers endorsed the majority (two or more) of the items on

this scale (24% of the sample) were considered to fall into the category of infant temperament risk.

Low receptive vocabulary scores at age 5

Youths who scored more than one standard deviation below average on the Peabody Picture Vocabulary Test-Revised ([Dunn & Dunn, 1981](#)) at the age 5 follow-up were considered to have the biological risk factor of a low receptive vocabulary score (8% of the sample).

Low Vocabulary IQ scores at age 15

Youths were administered the Vocabulary subtest of the Wechsler Intelligence Scale for Children (WISC-III) at the age 15 follow-up. Those who scored a scaled score of 5 or below on this subtest were considered to have the biological risk factor of low Vocabulary IQ scores (11% of the sample).

Deficits in executive functioning at age 15

Two separate neuropsychological measures of executive functioning were used in the scale of biological risks. Both of the tests that provided these measures were administered to the youths at the age 15 follow-up. The first measure was the difference score from the Dodrill format of the Stroop Color Word Test ([Dodrill, 1978](#)). In this task, youths were handed a sheet with a list of 176 color words printed in incongruous ink colors (e.g., the word *red* printed in green ink). They were asked to complete, as quickly as possible, two separate trials with this word list. In the first trial, they were asked to simply read off each of the words on the sheet. In the second trial, they were asked to name the printed colors of each of the words on the sheet. The difference score was calculated as the time in seconds to complete Trial 2 minus the time in seconds to complete Trial 1. This difference score is the most widely used Stroop measure of selective attention ([MacLeod, 1991](#)) and is theorized to reflect a failure in response inhibition ([Lezak, 1995](#)). Individuals whose Stroop difference scores were in the highest 10th percentile were considered to have this biological risk factor.

The second measure of executive functioning deficits used in this study was the perseverative error score on the Wisconsin Card Sort Test (WCST; [Berg, 1948](#)). In this task, the youths were presented with a series of cards with different shapes of different colors displayed on them. They were asked to sort the cards by category (e.g., by shape or color), and they were given feedback about whether they sorted each card correctly. Rules for correct categorization changed during the test (with no warning or explanation), and a failure to switch sets counted as a perseverative error. The Wisconsin perseverative error score has been found to be sensitive to frontal lobe damage in neuropsychological studies ([Lezak, 1995](#)). Individuals whose WCST perseverative errors were in the highest 10th percentile were considered to have this biological risk factor.

Aggressive Behavior

We defined aggressive behavior patterns in a manner consistent with the theories of [Moffitt \(1993\)](#) and [Patterson \(1982\)](#). Both theories suggest that there is a distinct group of boys who experience an early onset of aggressive behavior and continue to be aggressive into adolescence. This early-onset persistent aggression group has been contrasted with a distinct group of adolescent-onset aggressive boys and has also been compared directly with nonaggressive boys. In this study, we divided our sample (boys and girls) into these distinct aggressive behavior groups on the basis of maternal reports of aggression.

Specifically, our aggressive behavior measures were derived from maternal reports on the Child Behavior Checklist (CBCL; [Achenbach, 1991](#)) at ages 5, 14, and 15 years. Owing to resource constraints at the age 5 follow-up, a 33-item shortened version of the CBCL was used. This shortened form included 10 items from the Aggression subscale. These items were “argues a lot,” “demands a lot of attention,” “destroys own things,” “destroys things belonging to others,” “disobedient at home,” “gets into many fights,” “screams a lot,” “stubborn, sullen, or irritable,” “sudden changes in mood or feelings,” and “temper tantrums or hot temper.” For consistency across measures, the sum of these same 10 CBCL aggression items was used as our scale of aggressive behavior at the age 5 ($M = 6.79$, $SD = 3.75$), age 14 ($M = 5.19$, $SD = 3.74$), and age 15 ($M = 3.40$, $SD = 3.40$) follow-ups. At the age 14 follow-up, this 10-item subscale correlated .94 with the overall CBCL Aggression scale, and at the age 15 follow-up, this 10-item subscale correlated .96 with the overall CBCL Aggression scale. Alphas for our 10-item CBCL Aggression subscale were .84, .85, and .86 for the age 5, age 14, and age 15 reports, respectively.

In establishing our early-onset persistent, adolescent-onset, and nonaggressive groups, we used the same cutoff rule as [Moffitt and her colleagues \(1996\)](#) to define who was classified as aggressive (a cutoff of one standard deviation above the sample mean). We chose this measure of aggression because we could be more confident that individuals who obtained that cutoff were indeed aggressive. Similarly, to ensure that individuals classified as nonaggressive were indeed nonaggressive, we defined them as such only if their CBCL aggression scores were below the sample mean. Therefore, many individuals whose aggression scores were in the middle range (i.e., above the mean but below one standard deviation above the mean) were unclassifiable by our scheme and were therefore dropped from our sample. We believe that this conservative method of operationalization results in aggressive behavior categorizations that are more valid. Nevertheless, it should be noted that the distribution of youths in these specific aggression groups in our sample is not representative of their true distribution in the population.

Early-onset persistent aggression group

Membership in the early-onset persistent aggression group ($n = 62$; 39 boys and 23 girls) required the combined presence of (a) a raw score at least one standard deviation above the sample mean (calculated with both genders together) on the CBCL Aggression scale at age 5 and (b) a raw score at least one standard deviation above the sample mean on the CBCL Aggression scale at age 14 or at age 15.

Adolescent-onset aggression group

Youths who scored below the mean on the CBCL Aggression scale at age 5 but at least one standard deviation above the mean at either age 14 or age 15 (or both) were operationalized as belonging to the adolescent-onset aggression group ($n = 54$; 29 boys and 25 girls).

Nonaggressive group

Youths who scored lower than the mean on the CBCL Aggression scale at ages 5, 14, and 15 were classified as the nonaggressive comparison group in this study ($n = 254$; 130 boys and 124 girls).

Excluded from sample

As stated previously, 440 children in the overall high-risk sample of 816 families were excluded from this study because they did not fit into any of our aggressive behavior pattern comparison groups. Some of these children were desisters ($n = 25$). That is, they had high levels of aggression at age 5 but low levels at both ages 14 and 15. The small size of this group and the lack of theoretical predictions (by [Moffitt \[1993\]](#) or [Patterson \[1982\]](#)) about desistance precluded us from including this group in our analyses. Most of the children who were excluded as unclassifiable ($n = 415$) had moderate levels of aggression (above the mean but lower than one standard deviation above the mean), which precluded a clear classification as aggressive or nonaggressive at any point in time.

Relationship to youth self-reported delinquency and violence

Our aggressive behavior groups were based solely on mothers' reports because no other reporters provided data on youth behavior problems at all of the follow-ups. Youths did provide self-reports of delinquency at the age 15 follow-up, however, and this enabled us to validate our aggressive behavior groupings by comparing them to behavior reports from an alternative source.

Youth self-reports of both nonviolent and violent delinquent acts (see [Table 1](#)) were obtained at the age 15 follow-up. These self-report delinquency items were derived from the National Youth Survey and have established reliability and validity ([Elliott et al., 1985](#)). Several one-way analyses of variance found that aggression group status (based on maternal CBCL reports) was significantly related to youth self-reports of nonviolent delinquency, $F(2, 367) = 15.60, p < .001$, and violent delinquency, $F(2, 367) = 19.51, p < .001$, at age 15. Post hoc Duncan analyses revealed that mean scores for nonviolent delinquency were not significantly different for the early-onset persistent ($M = 5.57$) and the adolescent-onset ($M = 5.54$) aggression groups; however, both aggressive groups had significantly higher age 15 self-reported nonviolent delinquency scores than did the nonaggressive group ($M = 3.11$). In the case of self-reported violence, all three aggression groups were significantly different from one another, with the early-onset persistent group having the highest mean ($M = 0.97$), the

adolescent-onset group the next highest mean ($M = 0.67$), and the nonaggressive group the lowest mean ($M = 0.29$). This pattern of results provided convergent validity for our method of determining aggressive behavior groups.

Table 1
Self-Report Delinquency Items From the Age 15 Follow-Up

Nonviolent delinquency	
Have you run away from home?	
Have you skipped class without an excuse?	
Have you lied about your age to get into some place or to buy something?	
Have you hitchhiked a ride with a stranger?	
Have you been rowdy in a public place where somebody complained and got you in trouble?	
Have you made obscene telephone calls, such as calling someone to say dirty things?	
Have you been drunk in a public place?	
Have you damaged, destroyed, or marked up someone else's property on purpose?	
Have you avoided paying for things like a movie, taking bus rides, or anything else?	
Have you gone into or tried to go into a building to steal or damage something?	
Have you tried to steal or actually stolen money or things worth \$10 or less?	
Have you tried to steal or actually stolen money or things worth more than \$10?	
Have you tried to buy or sell something that you knew was stolen?	
Have you taken a car or a motorcycle for a ride without the owner's permission?	
Have you stolen or tried to steal a car or other motor vehicle?	
Have you forged a check or used fake money to pay for something?	
Have you used or tried to use a credit card or bank card without permission?	
Have you drunk beer or wine without your parent's permission?	
Have you drunk hard liquor without your parent's permission?	
Have you used marijuana?	
Have you used hard drugs such as heroin, cocaine, or LSD?	
Violent delinquency	
Have you carried a hidden weapon?	
Have you set fire on purpose or tried to set fire to a house, building, or car knowing someone was inside?	
Have you attacked someone with a weapon or with the idea of seriously hurting them?	
Have you thrown objects such as bottles or rocks at people?	
Have you used a weapon or force to make someone give you money or things?	
Have you physically hurt or threatened to hurt someone to get them to have sex with you?	

[Enlarge this image.](#)

Self-Report Delinquency Items From the Age 15 Follow-Up

Parental Psychiatric History

Given the high-risk nature of the sample, it is not surprising that there were a large number of individuals with positive family psychiatric histories (see [Table 2](#)). The presence of these family histories allowed us to examine the relationship among parental substance abuse, paternal antisocial behavior, maternal depression, and aggressive behavior outcomes. We examined these relationships in the context of our analyses of biological and social risks for aggressive outcomes. We did not include these measures of parent psychopathology in either the biological or the social risk category because our study design did not allow us to tease apart which of these types of risks they reflected (i.e., it was a family design, not a twin or adoptive design).

Table 2

Descriptive Statistics for the Risk Factors Assessed in Relation to Aggressive Behavior Outcomes

Group	Biological risks						Social risks						Mother depression		Parental substance abuse (%)
	Total		Early		Later		Total		Early		Later		M	SD	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD			
Overall sample	0.79	1.04	0.50	0.74	0.31	0.63	1.12	1.19	0.55	0.72	0.59	0.93	5.36	4.73	24.9
Early-onset persistent	1.37	0.17	0.84	0.11	0.53	0.11	1.85	0.17	0.94	0.11	0.98	0.16	10.82	0.81	43.5
Adolescent-onset	0.81	0.13	0.65	0.11	0.19	0.07	1.30	0.17	0.54	0.09	0.80	0.13	8.54	0.70	31.2
Nonaggressive	0.65	0.06	0.38	0.04	0.28	0.04	0.90	0.07	0.46	0.04	0.45	0.05	5.01	0.31	18.9

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Descriptive Statistics for the Risk Factors Assessed in Relation to Aggressive Behavior Outcomes

Maternal depression

Maternal depressive symptoms were indicated by self-report on the seven depression items of the DSSI during pregnancy, at birth, and at the 6-month, 5-year, and 15-year follow-ups. The DSSI is comparable to other validated self-report instruments that screen for major depressive episodes. For example, five of its seven items overlap with the Beck Depression Inventory ([Beck, Ward, Mendelson, Mock, & Erbaugh, 1961](#)), and four overlap with the Center for Epidemiological Studies Depression Scale ([Radloff, 1977](#)). In addition, the DSSI includes one explicit suicidal item and two hopelessness items that typically predict suicidal thoughts. These types of items identify significant depression and have been found to be the self-report items that distinguish major depression from dysthymic disorder ([Steer, Beck, Brown, & Berchick, 1987](#)).

A response of “some of the time,” “most of the time,” or “all of the time” to a depression item from the DSSI indicated that a symptom of depression had been endorsed by the mother. Internal consistencies (alphas) for the depression symptom items of the DSSI ranged from .71 to .81 across the administrations of this measure. For the purposes of the analyses in this article, a total count of depressive symptoms endorsed at all assessment phases was used as a measure of maternal depression.

Parental substance abuse and paternal antisocial personality disorder

Each mother's lifetime diagnosis of substance abuse was obtained with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; [First, Spitzer, Gibbon, & Williams, 1995](#)), which was administered at the age 15 follow-up. Father substance abuse and antisocial personality disorder diagnoses and symptoms were obtained from the father's self-report on the SCID (64% of the cases) or, in cases where the father was not interviewed directly, from the mother's report on the Family History Research Diagnostic Criteria ([Andreasen, Endicott, Spitzer, & Winokur, 1977](#)) at the age 15 follow-up. Interrater reliabilities for diagnoses ranged from .82 to 1.0 for mothers and from .72 to .99 for fathers.

Results

Descriptive statistics for our predictor variables are provided in [Table 2](#). We present them first for the overall sample and then separately for each of our three aggressive behavior outcome groups.

Spearman rho correlations for the relationships among gender, biological risk factors, social risk factors, and parent psychopathology are presented in [Table 3](#). Several of the risk variables were significantly related to one another, but the significant correlations were quite low in magnitude. The only predictor significantly related to gender was the number of biological risks. Boys scored higher on this measure than girls.

Table 3
Spearman's rho Correlation Coefficients and Two-Tailed Significance Values for Gender, Biological Risk Factors, Social Risk Factors, and Parent Psychopathology Predictors

Variable	No. of social risk factors	Maternal depression	Paternal APD	Parental substance abuse	Gender
No. of biological risk factors					
ρ	.15	.28	.12	.06	-.11
p	.003	.001	.025	.28	.044
No. of social risk factors					
ρ		.30	.12	.20	-.03
p		.001	.019	.001	.560
Maternal depression					
ρ			.09	.13	.06
p			.089	.010	.290
Paternal APD					
ρ				.23	-.03
p				.001	.597
Parental substance abuse					
ρ					-.01
p					.854

Note. Significant correlations and p values appear in boldface type. APD = antisocial personality disorder.

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Spearman's rho Correlation Coefficients and Two-Tailed Significance Values for Gender, Biological Risk Factors, Social Risk Factors, and Parent Psychopathology Predictors

Three types of bivariate logistic regression analyses were performed to examine the relationship between biological and social risk factors and aggressive behavior outcomes. In the first type of analysis, the early-onset persistent aggression group was compared with the nonaggressive group. In the second type of analysis, the adolescent-onset aggression group was compared with the nonaggressive group. And in the third type of analysis, the early-onset persistent aggression group was compared with the adolescent-onset aggression group. In all of the logistic regression analyses, the parental psychopathology variables and the cumulative biological and social risk variables were entered in the first step, and the interaction term of biological and social risks was entered in the second step.

Gender interaction terms were tested to see whether gender moderated the effect of the biological and social risk factors, separately or in combination, on the bivariate aggression outcomes. In other words, for each set of analyses on a particular bivariate outcome, we examined whether the Gender \times Biological Risk, the Gender \times Social Risk, and the Gender \times Biological Risk \times Social Risk interaction terms were significant. In cases where any of the gender interaction terms were significant, the analyses for that bivariate aggression outcome

were completed separately for boys and girls. In cases where none of the gender interaction terms was significant, analyses were undertaken with both genders combined, and gender was included as a statistical control (entered in the first block of the analysis). Alpha levels were set at .05.

Early-Onset Persistent Aggression Versus Nonaggression

No gender interaction terms were significant in the prediction of early-onset persistent aggression versus nonaggression. [Table 4](#) presents the significance levels and odds ratios of the predictor variables (and gender) included in the first step of these logistic regression models. In the bivariate comparison of early-onset persistent aggression and nonaggression, cumulative social risk factors as well as parent psychopathology were found to significantly predict aggressive outcome, and cumulative biological risk factors showed a trend toward significance. Odds ratios for the biological and social risk variables reflect the multiplicative increase in risk at each increasing level of biological or social risk, respectively. For example, children with four social risks were ($1.61 \times 1.61 \times 1.61 \times 1.61$ or) 6.7 times as likely to evidence early-onset persistent aggression than were children with zero social risks.

Table 4
Logistic Regression Models of the Relationship Between Biological and Social Risks and the Outcomes of Early-Onset Persistent Aggression Versus Nonaggression

Variable	Exp(β)	95% CI
Gender	0.50*	0.25–1.0
Maternal depression	1.17**	1.10–1.24
Paternal antisocial personality	3.11*	1.16–8.36
Parental substance abuse	2.07*	1.02–4.23
Biological risks	1.31†	0.99–1.73
Social risks	1.61**	1.22–2.12
Age 5 biological risks	1.34	0.90–1.98
Age 5 social risks	1.65*	1.10–2.48
Age 15 biological risks	1.47†	0.95–2.29
Age 15 social risks	1.60**	1.15–2.21

Note. Exp(β) is the factor by which the odds for aggressive behavior change when the independent variable increases by one unit. CI = confidence interval.

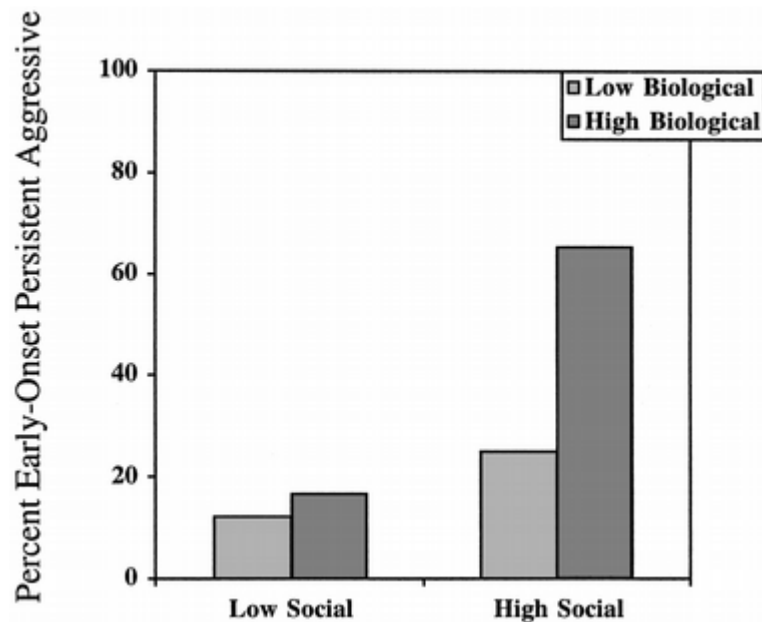
† $p < .10$. * $p < .05$. ** $p < .01$.

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Logistic Regression Models of the Relationship Between Biological and Social Risks and the Outcomes of Early-Onset Persistent Aggression Versus Nonaggression

Results from the second step of our logistic regression analyses revealed that, consistent with our hypothesis, the biosocial interaction term was significantly related to early-onset persistent aggression versus nonaggression, $\chi^2(1, n = 316) = 5.10, p = .02$. In order to interpret and graphically represent this biosocial interaction, we divided our subjects into the four distinct categories of (a) low social and low biological risk, (b) low social and high biological risk, (c) high social and low biological risk, and (d) high social and high biological risk. High-risk status reflected the presence of two or more risk factors, and low-risk status reflected the presence of one or fewer risk factors. We then graphed the percentage of children in each of these biosocial risk comparison groups who evidenced the behavior pattern of early-onset persistent aggression. [Figure 1](#) shows the percentage of children in each of these risk groups

who were early-onset persistent aggressive versus nonaggressive. As can be seen, the high-biological/high-social risk group is the most likely to evidence early-onset persistent aggression. Chi-square analyses revealed that in cases of low social risk, high biological risk was not associated with the outcome of early-onset persistent aggression, $\chi^2(1, n = 218) = 0.45, p = .50$, but that in cases of high social risk, it was, $\chi^2(1, n = 98) = 12.34, p < .001$.



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Biosocial risk status and early-onset aggression versus nonaggression.

Next we examined biological and social risk counts from earlier versus later developmental phases of measurement in terms of their relation to early-onset persistent aggression versus nonaggression. [Table 4](#) presents the significance levels and odds ratios of these biological and social risk count variables. As can be seen in the table, age 5 and age 15 social risks were significantly related to early-onset persistent aggression versus nonaggression, and age 15 biological risks showed a trend toward significance. We next explored whether any of these age 5 and age 15 risk counts interacted with one another such that a biosocial interaction predicted early-onset persistent aggression. None of these biosocial interaction terms was significant: Age 5 Biological \times Age 5 Social, $\chi^2(1, n = 316) = 0.98, p = .32$; Age 15 Biological \times Age 15 Social, $\chi^2(1, n = 316) = 0.06, p = .81$; Age 5 Biological \times Age 15 Social, $\chi^2(1, n = 316) = 2.07, p = .15$; and Age 5 Social \times Age 15 Biological, $\chi^2(1, n = 316) = 2.02, p = .16$.

Adolescent-Onset Aggression Versus Nonaggression

No gender interaction terms were significant in the prediction of adolescent-onset aggression versus nonaggression. Therefore, analyses were undertaken with boys and girls combined. [Table 5](#) presents the results of logistic regression analyses examining our parent psychopathology and biosocial risk factors in association with adolescent-onset aggression. These results are largely consistent with our hypotheses predicting that the selected risk factors in this study would not differentiate these aggression groups. Contrary to our hypothesis, however, age 15 cumulative social risk factors significantly predicted the

difference between the outcomes of adolescent-onset aggression and nonaggression in our sample.

Table 5
Logistic Regression Models of the Relationship Between Biological and Social Risks and the Outcomes of Adolescent-Onset Aggression Versus Nonaggression

Variable	Exp(β)	95% CI
Gender	.81	0.43–1.52
Maternal depression	1.12**	1.06–1.19
Paternal antisocial personality	1.23	0.38–3.90
Parental substance abuse	1.65	0.82–3.31
Biological risks	1.08	0.78–1.47
Social risks	1.21	0.92–1.59
Age 5 biological risks	1.31	0.87–1.97
Age 5 social risks	0.91	0.58–1.42
Age 15 biological risks	0.73	0.40–1.35
Age 15 social risks	1.45*	1.03–2.04

Note. Exp(β) is the factor by which the odds for aggressive behavior change when the independent variable increases by one unit. CI = confidence interval.

* $p < .05$. ** $p < .01$.

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Logistic Regression Models of the Relationship Between Biological and Social Risks and the Outcomes of Adolescent-Onset Aggression Versus Nonaggression

No Biological \times Social risk count interaction terms were significant in differentiating adolescent-onset aggression and nonaggression: Total Biological \times Total Social, $\chi^2(1, n = 308) = 1.03, p = .31$; Age 5 Biological \times Age 5 Social, $\chi^2(1, n = 308) = 0.69, p = .41$; Age 15 Biological \times Age 15 Social, $\chi^2(1, n = 308) = 0.02, p = .90$; Age 5 Biological \times Age 15 Social, $\chi^2(1, n = 308) = 0.34, p = .56$; and Age 5 Social \times Age 15 Biological, $\chi^2(1, n = 308) = 1.29, p = .26$. These results were consistent with our predictions.

Early-Onset Persistent Aggression Versus Adolescent-Onset Aggression

Both the Gender \times Social Risk and the Gender \times Biological Risk \times Social Risk interaction terms were significant in predicting early-onset persistent aggression versus adolescent-onset aggression. Therefore, as per our analysis plan, we undertook separate analyses for the boys and girls in the sample to examine the effect of biological risks, social risks, and their combination on the outcomes of early-onset persistent aggression and adolescent-onset aggression. [Table 6](#) presents the results of the analyses for individual biological and social risk predictor variables separately for each gender. As can be seen in the table, the only significant association was for girls, and it was between social risk factors and early-onset versus adolescent-onset outcomes. A higher number of social risks was associated with a higher likelihood of early-onset persistent aggression.

Table 6
Logistic Regression Models of the Relationship Between Biological and Social Risks and the Outcomes of Early-Onset Persistent Aggression Versus Adolescent-Onset Aggression

Variable	Exp(β)	95% CI
Boys ($n = 68$)		
Maternal depression	1.07	0.97–1.17
Paternal antisocial personality	0.76	0.17–3.44
Parental substance abuse	3.12 [†]	0.90–10.88
Biological risks	1.54 [†]	0.95–2.47
Social risks	0.94	0.60–1.47
Girls ($n = 48$)		
Maternal depression	1.06	0.93–1.21
Paternal antisocial personality	7.96	0.65–96.84
Parental substance abuse	0.75	0.19–3.04
Biological risks	1.22	0.65–2.30
Social risks	1.85*	1.08–3.18

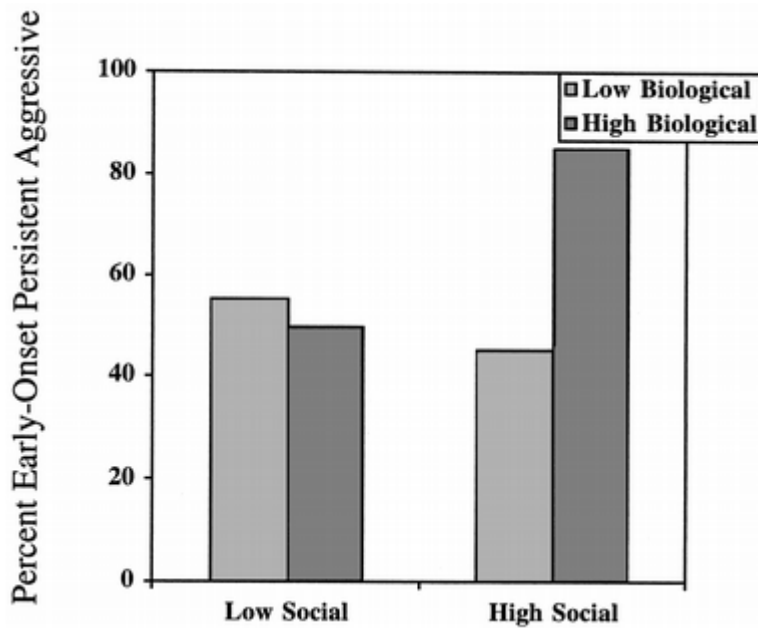
Note. Exp(β) is the factor by which the odds for aggressive behavior change when the independent variable increases by one unit. CI = confidence interval.

[†] $p < .10$. * $p < .05$.

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Logistic Regression Models of the Relationship Between Biological and Social Risks and the Outcomes of Early-Onset Persistent Aggression Versus Adolescent-Onset Aggression

Logistic regression analyses testing the Biological Risk \times Social Risk interaction term separately by gender revealed that this interaction significantly predicted early-onset persistent aggression versus adolescent-onset aggression for boys, $\chi^2(1, n = 68) = 5.45, p = .02$, but not for girls, $\chi^2(1, n = 48) = 2.50, p = .11$. [Figure 2](#) presents a graphic representation (using the same methods described for [Figure 1](#)) of this interaction effect for boys. As can be seen in [Figure 2](#), the group of boys exposed to *both* biological and social risks had an increased risk for early-onset persistent aggression. Chi-square analyses revealed that in cases of low social risk, high biological risk was not associated with early-onset persistent aggressive outcomes, $\chi^2(1, n = 35) = 0.54, p = .82$, but in cases of high social risk, it was, $\chi^2(1, n = 33) = 5.18, p = .02$.



[Enlarge this image.](#)

Biosocial risk status and early-onset aggression versus adolescent-onset aggression in boys.

Gender did not interact with the developmentally specific biological and social risk factors in their prediction of early-onset versus adolescent-onset aggression. Therefore, we examined these risk factors in analyses with both genders combined. [Table 7](#) presents these results. As can be seen in the table, both age 5 social risks and age 15 biological risks are significantly related to early-onset persistent aggression versus adolescent-onset aggression.

Table 7
Logistic Regression Models of the Relationship Between Developmentally Specific Biological and Social Risks and the Outcomes of Early-Onset Persistent Aggression Versus Adolescent-Onset Aggression

Variable	Exp(β)	95% CI
Age 5 biological risks	1.12	0.69–1.80
Age 5 social risks	1.79*	1.04–3.08
Age 15 biological risks	2.05*	1.08–3.90
Age 15 social risks	1.15	0.79–1.65

Note. Exp(β) is the factor by which the odds for aggressive behavior change when the independent variable increases by one unit. CI = confidence interval.

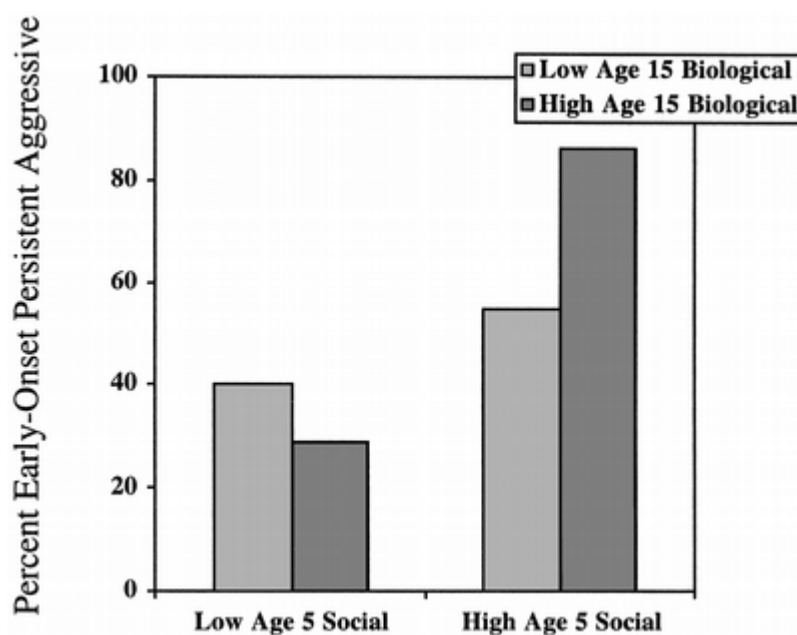
* $p < .05$.

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Logistic Regression Models of the Relationship Between Developmentally Specific Biological and Social Risks and the Outcomes of Early-Onset Persistent Aggression Versus Adolescent-Onset Aggression

All of the developmentally specified Biological \times Social risk count interaction terms were then tested in relation to early-onset aggression versus adolescent-onset aggression. Age 5 Biological \times Age 5 Social, $\chi^2(1, n = 116) = 2.23, p = .14$, Age 15 Biological \times Age 15 Social, $\chi^2(1, n = 116) = 0.43, p = .51$, and Age 5 Biological \times Age 15 Social, $\chi^2(1, n = 116) = 0.73, p = .39$, interaction terms were all nonsignificant. The Age 5 Social \times Age 15 Biological interaction term, however, was significant, $\chi^2(1, n = 116) = 5.87, p = .02$. In order to interpret

and graphically represent this particular biosocial interaction, we divided our subjects into the four distinct categories of (a) low age 5 social risk and low age 15 biological risk, (b) low age 5 social risk and high age 15 biological risk, (c) high age 5 social risk and low age 15 biological risk, and (d) high age 5 social risk and high age 15 biological risk. High-risk status reflected the presence of one or more risk factors, and low-risk status reflected the presence of zero risk factors. We then graphed (in [Figure 3](#)) the percentage of subjects in each of these biosocial risk comparison groups who evidenced the behavior pattern of early-onset persistent aggression. As can be seen in the figure, it is the combination of high levels of age 5 social risk and high levels of age 15 biological risk that is potent in increasing the likelihood of early-onset persistent aggression. Chi-square analyses revealed that in cases of low age 5 social risk, high age 15 biological risk was not associated with early-onset persistent aggressive outcomes, $\chi^2(1, n = 50) = 0.31, p = .58$, but in cases of high age 5 social risk, it was, $\chi^2(1, n = 66) = 6.54, p = .01$.



[Enlarge this image.](#)

Early-onset aggression versus adolescent-onset aggression: early social risks and later biological risks combined.

Discussion

Researchers have frequently observed that persistent antisocial behavior in adulthood is nearly always presaged by serious childhood behavior problems (e.g., [Robins, 1978](#)). The relatively bleak outcomes predicted by early-onset aggression highlight the importance of developing effective interventions for this type of behavior problem. Research guided by a developmental framework is perhaps the most useful approach to the design of interventions for aggressive youths and their families. The developmental approach allows a more complete understanding of individual differences in aggression and of the processes that may serve to initiate, escalate, maintain, and extinguish aggressive behavior over time. The goal of the present study was to evaluate the relationship of biological and social processes to the early-

onset aggression pathway in the context of a theoretical framework informed by two distinct developmental models of persistent antisocial behavior.

The results of this study suggest that a combined focus on biological and social risk factors is informative in the examination of developmental patterns of aggressive behavior. Biological risk factors specified by [Moffitt \(1993\)](#) and social risk factors specified by [Patterson \(1982\)](#) were found to significantly interact in the prediction of early-onset persistent aggression versus nonaggression in both boys and girls, as well as in the prediction of early-onset persistent aggression versus adolescent-onset aggression in boys. In both of these instances, children who were characterized as high in biological risk and high in social risk were more likely than other children in the sample to evidence early-onset persistent aggression.

An examination of the main effects of biological and social risk factors on aggressive behavior patterns yielded results similar to those noted by [Aguilar and colleagues \(2000\)](#). The main effect of social risk factors appears to be stronger than the main effect of biological risk factors in predicting aggressive behavior patterns. We would argue, however, that such an analysis is incomplete and that further attention to and testing of interaction effects is warranted, especially given the results of the current study.

Related to this conclusion is the fact that our analyses also replicated Aguilar et al.'s finding that early social risks and later biological risks were associated with persistent aggressive behavior. Our study has extended those results, however, by showing that early social risks *interact* with later biological risks to predict persistent aggression and that cumulative (lifetime) biological and social risks (in interaction with one another) are stronger predictors of persistent aggression in boys than are risk counts restricted either to early childhood or to adolescent phases of development. This pattern of results suggests that it may not simply be that cognitive deficits during adolescence result from prolonged childhood aggression. Rather, our findings are consistent with a cumulative, transactional process whereby biological risks, aggressive behavior, and social risks interact over time to keep children on a deviant behavioral trajectory.

We hypothesized that adolescent-onset aggressive boys and nonaggressive boys would not differ in terms of associated biological and social risks. We made this prediction because the biological and social risk factors that we chose to study were those most relevant to early-onset rather than adolescent-onset aggression ([Moffitt, 1993](#); [Patterson & Yoerger, 1993](#)). Nevertheless, our results suggest that adolescent-onset aggression is associated with a higher level of age 15 social-familial risk factors than is nonaggression. One possible explanation for this finding is that our social-familial risk factors might be correlated with deviant peer characteristics that were not measured in our study but that have been theorized to be centrally related to adolescent-onset aggression ([Moffitt, 1993](#); [Patterson & Yoerger, 1993](#); [Pettit, 1997](#)). Another possible explanation is that our age 15 social risk factors are reflecting the types of coercive processes that can develop once aggression has begun. This explanation would be consistent with [Patterson's \(1982\)](#) theory as well. Although he emphasized the importance of coercive processes in the prediction of early-starter aggression, he also acknowledged that they played a role for late starters as well.

Our results suggest that the processes that are related to persistent aggressive behavior patterns in boys and girls may be somewhat different. It was cumulative social risk factors alone that differentiated early-onset persistent aggression from adolescent-onset aggression in girls. Biological risk factors or their interaction did not significantly differentiate these

aggressive behavior patterns in girls. In this and other studies, it is gradually becoming clear that models of aggression that are primarily focused on boys are not easily generalized to girls ([Agnew & Brezina, 1997](#); [Cookston, 1999](#); [Kratzer & Hodgins, 1999](#); [Mears et al., 1998](#)). Therefore, we encourage the growth of the evolving literature on aggression and delinquency in girls.

It has been suggested that early-onset persistent aggression may be solely a male phenomenon ([Silverthorn & Frick, 1999](#)). It should be noted that our data suggest, however, that a group of girls does exist that displays the same pattern of early-onset persistent aggression as boys. Our aggressive behavior groupings were made for boys and girls combined, using cutoffs based on CBCL raw scores. Nine percent of boys and 7.4% of girls in our overall high-risk sample ($n = 816$) were classified as displaying early-onset persistent aggressive behavior according to this method. Given the fact that ours was a high-risk sample, and that social risk factors were correlated with parent psychopathology for girls, it is possible that the prevalence of this type of aggressive behavior pattern was higher than it would have been in other samples of girls. It will be an important next step to test for replication of our findings in unselected samples of youths.

In our study, we labeled some factors as “biological” and others as “social.” We acknowledge that this classification of variables is somewhat arbitrary. Our “biological” risk factors may have a social basis, and vice versa. It is perhaps more accurate to say that our study examined a combination of [Moffitt's \(1993\)](#) and [Patterson's \(1982\)](#) interaction processes rather than biosocial interaction processes. Nonetheless, we use the terminology of “biological” and “social” risks because it is consistent with what has been discussed in the aggressive behavior and criminological literature to date. The results of our study suggest, however, that arguments over the relative importance of internal, or “biological,” factors and environmental, or “social,” factors may inhibit progress in this field of study. It is when these two types of factors—biological and social—interact with one another that we find the strongest relationships with early-onset persistent aggressive behavior.

Our findings also suggest that parental psychopathology may play a role in predicting early-onset persistent aggression as well as adolescent-onset aggression. In fact, maternal depression was the only predictor that differentiated the outcomes of adolescent-onset aggression and nonaggression. Parent psychopathology variables were not the primary focus of this study, and most were retrospective rather than prospective measures, but our results suggest that these risk factors deserve further attention in this area of prediction.

Our study highlights the complementary relationship between the [Moffitt \(1993\)](#) and [Patterson \(1982\)](#) theories. For instance, Moffitt's theory can elaborate on Patterson's coercion model by predicting which children are at greater risk for initiating or exacerbating the coercion process: The child with subtle cognitive deficits and/or an undercontrolled temperament may be more likely to respond to aversive parental authority by using coercive behaviors (as opposed to some other deflection strategy, such as humor, acceptance, etc.). Likewise, Patterson's model can contribute meaningfully to Moffitt's theory by describing in greater detail the process of “failed parent-child interactions” that is so theoretically crucial to the etiology of early-onset antisocial behavior.

Our study suggests that both family environment and biological processes are related to early-onset persistent aggression in boys. One weakness of our integrated theoretical model, however, is the lack of attention given to peer influences on the development and

maintenance of aggressive behavior. Research that has focused on peer influences has found them to be important in predicting the continuity between early behavior problems and adolescent offending ([Fergusson & Horwood, 1996](#)). The examination of biological, familial, and peer processes together would be necessary to develop a complete understanding of developmentally based aggressive behavior patterns.

Further methodological weaknesses of our study were our lack of observational data on parenting and our reliance on a single source (maternal reports) to classify the aggressive behavior problems of the youths in our sample. Despite these weaknesses, our operationalized social risk factors were found to be strongly related to aggressive behavior outcomes in both boys and girls. And our maternal-report-based measures of aggression were found to be related to youth self-reports of nonviolent and violent delinquency in a manner consistent with what has been noted in previous studies (e.g., [Moffitt et al., 1996](#)). Nonetheless, we acknowledge that multiple informants would have provided a more complete picture of child aggressive behavior patterns, and a replication of these results based on multiple informant data would be an important goal for future research.

We defined children as aggressive on the basis of CBCL scores falling in the 84th percentile or higher (i.e., one standard deviation above the mean). This criterion was more liberal than we preferred, but it was necessary to create subgroup sizes large enough for reliable data analyses. It should be noted that the children defined as aggressive in our sample may not have displayed clinically significant levels of aggressive behavior. Therefore, despite the high-risk nature of our sample, our results may be more likely to generalize to community samples than to clinical samples of youth.

Our study was not an experiment; we did not assess causal relations between biological and social risk factors and aggressive behavior patterns in children. In addition, many of our biological and social risk factors were measured after the onset of aggressive behavior problems. Therefore, we cannot conclude that associated biological and social processes played a causal role in aggression. Future studies are needed that assess biological and social risk factors early in development and their relationship to aggressive behavior. More fine-grained analyses of such data would allow for a more comprehensive understanding of the interplay between the individual and the environment in the development of early-onset and adolescent-onset aggressive behavior problems.

Both [Patterson's \(1982\)](#) early-starter model and [Moffitt's \(1993\)](#) life-course-persistent offender theories have individually demonstrated excellent heuristic value, and both have generated much insightful research on aggressive outcomes in boys. Our study suggests that prediction models based on an integration of the two theories, rather than on one or the other alone, may be more useful in guiding research efforts in the future.

The results of our study also have implications for prevention and intervention. The pattern of risk factors associated with an early age of onset of aggression is different from the pattern of risk factors associated with a later age of onset. Children who have an early onset of aggression appear to be at risk for also having a greater number of biological and social risk factors with which to contend. Practitioners may want to obtain a complete physiological as well as social history on children who present with aggressive behavior at an early age so that a greater number of these risk processes can be attended to and ameliorated.

Our study did not include an examination of desisters because of issues of low statistical power. Further research on children who desist from early-onset aggression will be extremely useful for prevention and intervention. Specifically, an examination of protective factors that lead to desistance might help in the design of prevention strategies that enhance such factors. In addition, further differentiation of persistent aggressive behavior patterns and patterns of desistance may help to better identify those aggressive children who need intervention programs the most.

Our results suggest that the combination of high levels of biological risk and high levels of social risk is associated with persistent forms of aggressive behavior. This finding also has implications for intervention and prevention. In cases where children are identified as being at high risk in either of these areas (biological or social), extra therapeutic attention can be given to enhancing their skills and decreasing their risks in the other area. Of course, the linear relationship between number of risk factors and aggression suggests that interventions would be most effective if they work to ameliorate multiple types of social risk factors or multiple types of biological risk factors within this context. Simply put, our results suggest that intervention strategies that assess a broad range of both biological and social risk factors are likely to have the greatest success in reducing persistent aggressive behavior in children.

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